Common Surgical Diseases
Second Edition
This book is dedicated to my wife, Beth, for her infinite patience, love, and support, and to our son, Jack, for his never-ending curiosity and boundless energy.

Jonathan A. Myers

To my parents, John and Joan, for making it all possible.
To my wife, Janet, for her never-ending understanding and support of my career and, finally, to my children, Keith, Michael, Kyle, Cameron, Samantha, and John, for inspiring my optimism for the future.

Keith W. Millikan

I dedicate this to the physicians and healers whose work and lives have been a constant source of inspiration for me and countless others. May their memory be eternal.

Theodore, Neofytos, John, Cosmas, Damian, Panteleimon, Nektarios, Savas, Herman, Rose.

Theodore J. Saclarides
Acknowledgments

We are grateful to Steven G. Economou and Alexander Doolas for their encouragement and tutelage over the last several decades. Our deepest gratitude to Eileen Pehanich for her untiring efforts in the completion of this work.

Jonathan A. Myers
Keith W. Millikan
Theodore J. Saclarides
The printing of the 2nd edition of *Common Surgical Diseases: An Algorithmic Approach to Problem Solving* attests to its usefulness and popularity. Dr. Steven Economou, who wrote the first foreword, died on April 7, 2007, and despite his prolonged and debilitating illness, he retained his cheerful and optimistic nature. Those of us who have been mentored by this great human being hope to emulate, but will probably never achieve, his Olympian deeds as a surgeon, author, teacher, and leader. I am very honored to have been asked by the authors to write the foreword for the 2nd edition.

Years ago, the resident or medical student was commended for making a long list of obscure differential diagnoses regardless of the remoteness from the patient’s problem. Of course, these diagnoses have to be excluded or confirmed by numerous investigations, which could be painful, time consuming, dangerous, and costly. One might say that having many diagnoses and performing many tests protects the physician from litigation. In fact, it will do the opposite if they are not appropriate and complications arise. Medicine has turned from performing too many investigations to expeditious and concise “evidence-based medicine.” The authors have predated this trend by a few years and the printing of the 2nd edition is timely. *Common Surgical Diseases: An Algorithmic Approach to Problem Solving* is not only for residents and students or the young surgeon but also for *all physicians who deal with surgical patients*. After all, a nonspecialist may not know the intricacies of decision making for a Whipple operation and may order a percutaneous biopsy or an ERCP when, regardless of the result, the patient may or may not be a surgical candidate.

Common surgical diseases are not the only topics. There are at least 20 general topics on preoperative and postoperative care. Drs. Myers, Millikan, and Saclarides have taken advantage of the many talented attending physicians and residents at Rush University to write most of the chapters. The authors themselves wrote numerous chapters on their specialty. All the chapters were reviewed, corrected, and edited by the authors and they have left their indelible character in the text. This book belongs in the pocket of residents and students and on the desk of practicing physicians who deal with a wide range of patients.

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Part I
General Preoperative Considerations
Assessing Preoperative Cardiac Risk in Patients Undergoing Noncardiac Surgery

David M. Rothenberg

A. Emergency surgery. For the patient who presents with an emergency such as a perforated viscus or intraabdominal hemorrhage, surgery must be undertaken without delay. Clearly, in these instances, there isn’t sufficient time to proceed with a cardiac workup; however, invasive monitoring may help perioperative fluid management.

B. Elective surgery. Minimizing perioperative morbidity and mortality is best achieved by performing a thorough preoperative evaluation. This is particularly imperative in patients with suspected coronary artery disease where an accurate history is 80–90% sensitive and specific in making a diagnosis. To determine the likelihood of intra- or postoperative cardiac complications, one must (1) identify preexisting or coexisting cardiac disorders, (2) assess the severity or stability of the cardiac disease, (3) maximize medical therapy, and (4) determine the likelihood surgery will place the patient at risk.

C. Major predictors. Major clinical predictors of cardiac complications are recent myocardial infarction (MI), unstable angina, decompensated congestive heart failure (CHF), severe arrhythmias (atrioventricular heart block, supraventricular tachycardia, and ventricular tachycardia), and valvular dysfunction (aortic stenosis). Patients with recent MI are at great risk for reinfarction and death, the risk is greatest when patients undergo elective noncardiac surgery within 6 months of the myocardial infarction. Patients with major predictors of cardiac complications should have their elective surgery rescheduled, while medical therapy is intensified and invasive testing such as angiography is considered.

D. Intermediate predictors. Intermediate predictors of cardiac complications include recent myocardial infarction (MI), unstable angina, decompensated CHF, severe arrhythmias (atrioventricular heart block, supraventricular tachycardia, and ventricular tachycardia), and valvular dysfunction (aortic stenosis). Patients with recent MI are at great risk for reinfarction and death, the risk is greatest when patients undergo elective noncardiac surgery within 6 months of the myocardial infarction. Patients with intermediate predictors should have their elective surgery rescheduled, while medical therapy is intensified and invasive testing such as angiography is considered.

E. Minor predictors. Minor predictors of cardiac complications include advanced age, abnormal ECG, nonsinus heart rhythms, history of stroke, or uncontrolled hypertension. Again, cardiac evaluation is undertaken in consideration of the patient’s functional capacity and the type of surgery planned. Patients with less than 4 METs who are scheduled for high-risk procedures should have noninvasive testing (see Algorithm).

F. Operate. On the day of surgery, patients should receive their cardiac medications and antihypertensives with a sip of water, although diuretics are often withheld to minimize perioperative hypovolemia. (If diuretics are being administered for the treatment of CHF, they may be given.) Antiangular medications (beta-antagonists), drugs used to treat CHF (digoxin- and angiotensin-converting enzyme inhibitors), are also given. Anticoagulants, however, must be withheld so as to prevent intraoperative hemorrhage. Coumadin, an anticoagulant used to prevent systemic embolization in patients with atrial fibrillation, must be discontinued 3–4 days prior to surgery, while intravenous heparin may be stopped 6–8 h prior to the operation. Patients with prosthetic heart valves should receive antibiotic prophylaxis to prevent subacute bacterial endocarditis (SBE). Patients with other valvular heart disease or congenital cardiac anomalies may also require SBE prophylaxis depending on the nature of the surgery. In general, preoperative antibiotics are given to patients with valvular complications and should undergo a noninvasive evaluation with stress testing, nuclear imaging, and echocardiography to determine if angiography is needed. If these tests produce favorable results, surgery may be undertaken; otherwise angiography should precede surgery.

Certain surgeries carry a greater risk than others, particularly abdominal, thoracic, and major vascular surgery. This most likely relates to the more intense humoral stimulation and hemodynamic instability associated with these types of procedures. For example, patients with intermediate predictors who have greater than 4 METs but are scheduled for high-risk procedures should probably undergo noninvasive testing. If the procedure is considered low risk, surgery can be done without delay.

G. Operate. On the day of surgery, patients should receive their cardiac medications and antihypertensives with a sip of water, although diuretics are often withheld to minimize perioperative hypovolemia. (If diuretics are being administered for the treatment of CHF, they may be given.) Antiangular medications (beta-antagonists), drugs used to treat CHF (digoxin- and angiotensin-converting enzyme inhibitors), are also given. Anticoagulants, however, must be withheld so as to prevent intraoperative hemorrhage. Coumadin, an anticoagulant used to prevent systemic embolization in patients with atrial fibrillation, must be discontinued 3–4 days prior to surgery, while intravenous heparin may be stopped 6–8 h prior to the operation. Patients with prosthetic heart valves should receive antibiotic prophylaxis to prevent subacute bacterial endocarditis (SBE). Patients with other valvular heart disease or congenital cardiac anomalies may also require SBE prophylaxis depending on the nature of the surgery. In general, preoperative antibiotics are given to patients with valvular complications and should undergo a noninvasive evaluation with stress testing, nuclear imaging, and echocardiography to determine if angiography is needed. If these tests produce favorable results, surgery may be undertaken; otherwise angiography should precede surgery.
heart disease, prior valve replacement surgery, and congenital anomalies who undergo upper respiratory, genitourinary, or gastrointestinal surgery.

While there are no definite outcome studies regarding preoperative coronary revascularization (either with coronary artery bypass grafting or with coronary angioplasty), a select group of patients may benefit from one of these procedures prior to their elective noncardiac surgery. Patients undergoing major vascular surgery such as elective abdominal aortic aneurysm repair or carotid endarterectomy are those who often have improved outcome when myocardial revascularization is performed first.

<table>
<thead>
<tr>
<th>Estimated energy requirements for various activities.</th>
</tr>
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<tbody>
<tr>
<td><strong>1 MET</strong></td>
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<tr>
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<tr>
<td></td>
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<tr>
<td><strong>4 METs</strong></td>
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</table>

**Table 1.1.**
Assessing Preoperative Cardiac Risk in Patients Undergoing Noncardiac Surgery

Non-cardiac surgery → A. Emergency → Operate

B. Elective → Favorable status

Changing symptoms, unfavorable status

C. Major clinical predictors
   - unstable coronary disease
   - decompensated CHF
   - severe arrhythmias
   - severe valvular disease

   delay surgery

   angiogram

D. Intermediate predictors
   - mild angina
   - prior MI
   - compensated CHF
   - diabetes mellitus

   < 4 MET
   non-invasive testing
   high-risk disease
   Operate

   > 4 MET
   high-risk procedure
   low-risk procedure
   Operate

E. Minor predictors
   - advanced age
   - abnormal EKG
   - non-sinus rhythm
   - history stroke
   - uncontrolled HTN

   > 4 MET
   high-risk procedure
   low-risk procedure
   Operate

   < 4 MET
   non-invasive testing
Management of Preoperative Bleeding Disorders—Intraoperative Coagulopathy

Jeffrey Shuhaiber and Jonathan Silverstein

The main contributors to hemostasis are the vessel wall (endothelium and subendothelium), platelets and circulating proteins with procoagulant and fibrinolytic activities. The surgeon may be the first to witness abnormal hemostasis and should know how to reach an immediate solution in a timely fashion.

Localized bleeding must be distinguished from diffuse bleeding that occurs spontaneously. The latter may be characterized by poor clot formation in the surgical field to extreme bleeding from previously hemostatic areas such as venipuncture sites. In many instances, the mainstay of treatment is functional and quantitative repletion of the deficient components. Even in the presence of coagulopathy, however, most bleeding complications are due to failure of local control in the operative field. Hypothermia, acidosis and shock should be identified and corrected as soon as possible. In the absence of a life threatening problem, the surgeon should terminate the operation to study, manage, and resuscitate the patient. The history can define hereditary or acquired factors. For the purpose of this discussion, preoperative work up and patient screening will be mentioned first followed by diagnosis and treatment of specific and common bleeding disorders.

A. Preoperative workup. It is important to identify hematic defects preoperatively so that surgery can be planned with appropriate hemostatic support. The assessment should inquire about history of bleeding problems, including spontaneous bleeding and following a hemostatic challenge (e.g., dental extraction or previous surgery). Liver disease and renal impairment should alert the clinician to a possibility of coagulopathy. Drugs such as aspirin, NSAIDs and warfarin may affect bleeding. Food like fish oil, red wine or garlic have been associated with a thrombocytopathy. A family history should be obtained remembering that hereditary conditions may miss generations and mild coagulation defects may become apparent only at times of extreme hemostatic stress. Clinical signs of bruising, skin purpura, petechiae or buccal mucosal hemorrhages should be recognized. More recently in females, menorrhagia has been associated with vWF deficiency.

It is important to recognize that certain religions prohibit blood transfusion even when death is probable. The use of erythropoietin, iron and autologous transfusion by normovolemic hemodilution or RBC saver machines should be considered in advance. The use of RBC substitutes have demonstrated promise in clinical trials but are not yet FDA approved.

Assessing the patient’s drug history is important. NSAIDs and newer agents like clopidogrel and ticlodipine interfere with platelet aggregation either by inhibiting platelet thromboxane A2 or blocking the ADP receptor and thus decreasing platelet GIIb/IIIa expression. The effects can last up to 7 days. Patients taking heparin sq, low molecular weight heparin (LMWH) sq, or warfarin p.o. should be identified. Correction of coagulopathy can be performed slowly by halting these medications or acutely by fresh frozen plasma, Vitamin K and/or cryoprecipitate (factor VIII and vWF and fibrinogen rich). Protamine sulfate reverses heparin activity by liberating antithrombin III. (Table 2.1 represents coagulation cascade)

B. Diagnosis. When elective and sometimes emergent operations are required, the management of therapeutic anticoagulation prior to operation requires an assessment of the risks and benefits of discontinuing anticoagulation. The operation can be safely performed when the INR is less than 1.5. However, such a decrease will be associated with some increased level of thrombogenic activity. Acutely lowering the INR with fresh frozen plasma and vitamin K is appropriate when emergency operation is required. Under typical circumstances, the INR returns to normal within 3–5 days after medication is stopped. Subcutaneous heparin can be used to maintain the anticoagulation status and to protect against thromboembolic phenomena. However, the intravenous route of heparin has more predictable pharmacokinetics.

Diagnosis of bleeding disorders may be aided by thromboelastography (TEG), which measures overall coagulopathy and fibrinolytic function. TEG can measure blood clotting time, platelet and factor function, fibrinolysis and hypercoagulability. Another test, euglobulin clot lysis time (ECLT),
measures the time for a clot to lyse and can be used if fibrinolysis is suspected.

### Bleeding Disorders

#### C. Disseminated intravascular coagulation (DIC). It is a complex pathological process resulting from inappropriate activation of both the coagulation cascade and the fibrinolytic system. Precipitating causes include sepsis, fat embolism, massive hemorrhage and placental abruption. There is microvascular thrombosis in small arterioles resulting in tissue ischemia and end organ damage (e.g., renal failure) and subsequent consumption of platelets and blood coagulation factors causing generalized hemorrhage. In response to the tissue ischemia and in an attempt to maintain microvascular patency, excess plasmin is generated causing systemic fibrinogenolysis and local fibrinolysis. Classically, thrombocytopenia occurs, and INR, FDP, and D-Dimer are elevated. The ECLT has little value in DIC where clot formation and lysis are both abnormal. The decision to start replacing blood components does not depend not on absolute values, but on whether the patient is bleeding or is about to have a procedure. Treatment with platelets, fresh frozen plasma and/or cryoprecipitate should be given if there is clinically significant bleeding. The response (measuring PT, PTT, platelet count, fibrinogen level) should be monitored regularly. Heparin may be useful if the clinical problems are dominated by end organ damage but should be used in consultation with a hematologist.

#### D. Platelet abnormalities. Quantitative decline in platelets (thrombocytopenia) may be seen in sepsis, myeloma, large-volume blood transfusion, DIC, heparin-induced thrombocytopenia (either LMWH or standard heparin) and hypersplenism associated with idiopathic thrombocytopenic purpura. The underlying disorder should be treated and platelets should be replaced to a goal of between 20,000 and 50,000. Quality of platelet function is altered by chronic uremia in end stage renal failure, Von Willebrand’s disease (factor VIII R:WF deficiency), Glanzmann (GPIIb/IIIa defect), Bernard-Soulier (GPIb/IX/V defect), and Scott (defect in membrane thrombin formation) syndromes. Rare abnormalities in platelet secretion in dense granules or defective secondary mechanisms can also occur. Von Willebrand’s disease can be inherited or acquired as in hematologic malignancies usually from development of antibodies against vWF or factor VIII. Treatment includes platelet transfusion or DDAVP that increases factor VIII and vWF levels especially in uremic patients. Note that type II vWD is dysfunctional factor while type III is absence of vWF altogether. Antifibrinolytics can also indirectly help by stabilizing any clot that has formed.

#### E. Coagulation factor defects. The most common hereditary defect of this type is hemophilia, which is an X-linked inherited deficiency of factor VIII (hemophilia A) or factor IX (hemophilia B). Mild hemophilia (factor VIII or IX levels 10–25% of normal) may be undiagnosed until unexpected postoperative bleeding occurs, but severe hemophilia (<1% normal) usually presents in infancy with spontaneous bleeding problems. Surgery in any hemophilic patient must be planned and managed in cooperation with a hematologist experienced in such cases.

Before surgery, every hemophilic patient should be screened for the presence of an inhibitor to factor VIII. Patients with hemophilia who do not have an inhibitor should receive factor VIII infusions just before surgery and will require daily monitoring so that the factor VIII level is maintained >50% for 10 to 14 days after surgery. When patients undergo joint replacement or other major orthopedic surgery, therapy should be continued for 3 weeks to permit wound healing and the institution of physical therapy. Following multiple transfusions, 10–20% of patients with severe hemophilia develop inhibitors to factor VIII. Inhibitors are usually IgG antibodies that rapidly neutralize factor VIII activity. Two types of inhibitors are found with different biologic characteristics and different clinical presentations.

For patients with hemophilia B, accurate laboratory diagnosis is critical, as it is clinically indistinguishable from factor VIII deficiency (hemophilia A), but requires different treatment. Either fresh-frozen plasma or a plasma fraction enriched in the prothrombin complex proteins is used. Monoclonally purified
or recombinant factor IX preparations are now available. In addition to the expected complications of hepatitis, chronic liver disease, and AIDS, the therapy of factor IX deficiency has a special hazard of causing thrombosis and embolism. As a result, some centers have returned to fresh-frozen plasma for factor IX-deficient surgical patients, while others have recommended the addition of small doses of heparin to the concentrate to activate antithrombin III during the infusion and reduce hypercoagulability. The recombinant or monoclonally purified products are less likely to be thrombogenic.
A. Preoperative Workup
1. History of Bleeding
2. Liver or Renal Disease
3. Drugs
4. Family History
5. Easy Bruising
6. Menorrhagia

B. Diagnosis
1. IWR > 1.5
2. ThromboElastography (TEG)
3. Euglobulin Clot Lysis (ECLT)

C. Disseminated Intravascular Coagulation
1. Sepsis, fat embolism, massive hemorrhage, placental abruption
2. Thrombocytopenia with elevated, INR, FDP and D-Dimer
3. Infuse platelets, FFP on cryoprecipitate. Possibly heparin

D. Platelet Abnormalities
1. Quantitative: sepsis, myeloma, transfusion, DIC, heparin induced, ITP

E. Coagulation Factor Defects
1. Hemophilia (A or B) [Factor VIII or IX]
2. Mild Hemophilia (Factor VIII or IX)
A. Assessment. To prevent the morbidity and mortality associated with malnutrition, an evaluation of a patient’s nutritional status early in their clinical course and again throughout the patient’s treatment is essential. The most common nutritional deficiency is protein–calorie malnutrition, the least common is vitamin or mineral deficiency; multiple factors can contribute to either. The history and clinical examination may reveal obvious problems which lead to or result from nutritional deficits (i.e., poor dentition inhibiting oral intake; alopecia as a result of zinc deficiency). A review of current medical data and past medical history will identify conditions that influence nutritional status, such as chronic or acute disease states, surgery, chemotherapy, radiation therapy, and/or medications with possible drug–nutrient interactions. Diet history allows for the review of current oral intake with emphasis on eating habits and preferences, physical activity, and recent alterations in intake.

Stressed or catabolic patients may develop total or partial starvation quickly. Assessment of anthropometrics and somatic protein stores can determine severity of malnutrition. Severe weight loss over time can be categorized as follows: >2% in 1 week, >5% in 1 month, >7.5% in 3 months, and >10% in 6 months. More generally, significant weight loss is considered to be loss of 10–20% of usual body weight.

Deficits in nutrition-related lab values help identify the presence of malnutrition. Visceral protein levels are commonly used to evaluate nutritional status and become highly useful in determining protein stores as a direct indicator of nutritional intake; examples include serum albumin, thyroxine-binding prealbumin, and transferrin. Visceral protein stores can be affected by other factors, such as hydration status, which need to be considered during evaluation. Serum transferrin < 150 mg dl⁻¹, serum albumin < 2.5–3.0 ug dl⁻¹, and the prealbumin levels < 10 mg dl⁻¹ are considered significant deficits in terms of malnutrition. Evaluation of immunocompetence can be useful in nutritional assessment since total lymphocyte count less than 1,500 is an indicator of cellular immunity which is negatively affected by protein–calorie malnutrition.

B. Requirements. Accurately determining nutritional requirements, including energy, protein, electrolyte, and vitamin and mineral needs, is essential. Standardized equations or indirect calorimetry via metabolic cart assessment of oxygen consumption versus carbon dioxide release is typically used to determine calorie needs. Typically, 20–35 kcal kg⁻¹ satisfy patients’ energy requirements. Protein requirements are based on the amount protein necessary to maintain nitrogen balance. The recommended amount of protein for the healthy adult is 0.8 g kg⁻¹. Variations in requirements are due to stress- and disease-related limitations. Typical requirements for protein for the surgical patient are 0.8–2.5 g kg⁻¹. Nitrogen balance via urine collection or ongoing monitoring of visceral protein stores can be useful in evaluating adequacy of protein intake.

Various equations can be used to determine fluid needs, including 35 ml kg⁻¹ if patient is < 55 years of age, 30 ml kg⁻¹ if patient is 56–75 years of age, and 25 ml kg⁻¹ if patient is 75+ years of age. Careful monitoring for signs of overhydration and dehydration with use of supplemental fluids is necessary. Electrolyte requirements need to be evaluated on an individual basis and supplementation should be provided when necessary. Vitamin and mineral needs are typically based on Recommended Dietary Allowances.

C. GI tract functional? Preoperative determination of significant malnutrition necessitates early intervention via oral supplementation, tube feedings, or total parenteral nutrition (TPN). Parenteral feedings should be used when the gastrointestinal tract is deemed nonfunctional. General guidelines to determine significant malnutrition are based on visceral and somatic protein store depletion. A significant risk in TPN use includes catheter-related infections. Tube feeding use is dependent on tube availability and tolerance to formula introduction into the gastrointestinal tract. If immediate surgery is required and the patient will have an extended period without use of the gastrointestinal tract postoperatively, a central venous access device should be placed at the time of surgery. Alternatively, one may be able to postpone surgery to optimize nutritional status. Seven to ten days of full nutritional support prior to surgery...
in those individuals with significant malnutrition is thought to improve outcomes.

D. Specific formulations. Determination of patient-specific enteral and parenteral formulations is necessary to meet the patient’s nutritional requirements. Types of ready-made enteral formulas are preselected in most institutions and a volume appropriate to meet the patient’s nutritional needs and restrictions should be ordered. Formulation of a patient-specific parenteral formula requires careful consideration and review of all components; nutrition support teams or other trained personnel can assist in TPN management. Dextrose is the principal source of carbohydrates and provides 3.4 kcal g\(^{-1}\). Dextrose is available in 5, 10, 20, 30, 50, and 70% solutions. Maximal glucose infusion rate is 5 mg kg\(^{-1}\) min\(^{-1}\) and this rate should not be exceeded to prevent hyperglycemia. Additional calorie needs can be met with the addition of fat (9 kcal g\(^{-1}\)). Dextrose is available in 5, 10, 20, 30, 50, and 70% solutions. Maximal glucose infusion rate is 5 mg kg\(^{-1}\) min\(^{-1}\) and this rate should not be exceeded to prevent hyperglycemia. Additional calorie needs can be met with the addition of fat (9 kcal g\(^{-1}\)). Dextrose is available in 5, 10, 20, 30, 50, and 70% solutions. Maximal glucose infusion rate is 5 mg kg\(^{-1}\) min\(^{-1}\) and this rate should not be exceeded to prevent hyperglycemia. Additional calorie needs can be met with the addition of fat (9 kcal g\(^{-1}\)). Dextrose is available in 5, 10, 20, 30, 50, and 70% solutions. Maximal glucose infusion rate is 5 mg kg\(^{-1}\) min\(^{-1}\) and this rate should not be exceeded to prevent hyperglycemia. Additional calorie needs can be met with the addition of fat (9 kcal g\(^{-1}\)). Dextrose is available in 5, 10, 20, 30, 50, and 70% solutions. Maximal glucose infusion rate is 5 mg kg\(^{-1}\) min\(^{-1}\) and this rate should not be exceeded to prevent hyperglycemia. Additional calorie needs can be met with the addition of fat (9 kcal g\(^{-1}\)). Dextrose is available in 5, 10, 20, 30, 50, and 70% solutions. Maximal glucose infusion rate is 5 mg kg\(^{-1}\) min\(^{-1}\) and this rate should not be exceeded to prevent hyperglycemia. Additional calorie needs can be met with the addition of fat (9 kcal g\(^{-1}\)). Dextrose is available in 5, 10, 20, 30, 50, and 70% solutions. Maximal glucose infusion rate is 5 mg kg\(^{-1}\) min\(^{-1}\) and this rate should not be exceeded to prevent hyperglycemia. Additional calorie needs can be met with the addition of fat (9 kcal g\(^{-1}\)).

E. Monitoring. Monitoring protocols when using TPN are institution specific and include assessment of minerals, electrolytes, triglyceride levels, liver function tests, serum glucose, CBC with differential, PT and PTT, fluid inputs and outputs, weight, and visceral protein changes.

F. Initiation of TPN and gradual attainment of initial solution infusion goals over a 24-h period is typical. Once tolerance is demonstrated, TPN can be advanced to meet nutritional goals and cycled.

G. Transition. Discontinuation of TPN prior to surgery is imperative given the possible fluid and electrolyte shifts and stress-related factors associated with surgery. If the gastrointestinal tract can be used, tube feeding tube access should be considered and placed during surgery. Reevaluation of patients’ nutritional needs and TPN formula postsurgery is prudent and should include adjustments for the metabolic changes associated with the surgery.
Preoperative Nutritional Assessment/Intervention

A. Nutritional Assessment
- Clinical exam
- Medical history
- Anthropometric
- Somatic protein stores
- Laboratory values
- Immunocompetence

B. Nutritional Requirements
- Energy
- Protein
- Micronutrients
- Fluid

C. G.I. Tract Functional?
- Yes
  - Evaluate and place Venous access device
  - Proceed with tube feeding and tube placement
- No
  - Evaluate and place Venous access device

D. Specific Formulations
- Determine individual nutritional requirements and restrictions to formulate goal TPN

E. Monitor
- Obtain initial lab values including electrolytes, liver function tests, serum glucose, PT and PTT, CBC with diff., minerals and triglycerides

F. Initiate and advance TPN to goal

G. Transition
- Transition to tube feedings when GI tract functional and appropriate access placed
Transfusion Reactions

Jennifer B. Manders

Despite advances that have been made in collection, distribution, and safety of blood and blood products, serious complications can arise from their transfusion. After a massive transfusion (a single transfusion of 2,500–5,000 ml over 24 h), a variety of problems can occur. These include circulatory overload, DIC, dilutional thrombocytopenia, impaired platelet function, hypothermia-induced coagulopathy due to cold products, deficiency of factors V, VII, XI, and hyperkalemia.

The use of blood from several donors increases the likelihood of a hemolytic reaction due to incompatibility. The incidence of nonfatal hemolytic reactions is approximately 1 in 6,000 units of blood administered. Fatal hemolytic reactions occur in 1 out of 100,000 units administered.

The risk of infectious disease transmission increases progressively with each succeeding unit. Epstein-Barr, cytomegalovirus, hepatitis, HIV, and HTLV-1 and -2 are among the most commonly transmitted viruses. Bacteria include syphilis, malaria, Yersinia enterocolitica, Babesia microti, and Trypanosoma cruzi (see Table 4.1).

A. Of the several reactions that can occur, the most severe and life threatening are hemolytic, allergic, and septic shock. Each of these entities has a recognizable clinical manifestation and treatment protocol.

B. Hemolytic reactions are thought to be mediated by ABO and Rh group incompatibility, possibly as a result of clerical errors in the lab or at the time of transfusion. Intravascular destruction of RBCs occurs with consequent hemoglobinuria and hemoglobinemia. Circulating haptoglobin binds up to 100 mg of hemoglobin per dl of plasma, and the complex is cleared by the reticuloendothelial system. If the binding capacity is exceeded, free hemoglobin binds albumin to form methemalbumin; if free plasma levels reach 150 mg dl⁻¹, heme is excreted in urine. Damage to the kidneys occurs as tubular necrosis, as a result of hemoglobin precipitation within the tubules. DIC may be initiated by circulating antibody–antigen complexes activating factor XII end compliment, leading to the activation of the coagulation cascade. The kallikrein–bradykinin system may also be activated, affecting the circulatory system. If the patient’s antibody titer is low at the time of transfusion, a hemolytic reaction may be delayed for several days. Hemolytic reactions are manifested by heat or pain in the affected veins, a flushed face, pain in the lumbar area or chest, respiratory distress, hypotension, or tachycardia. Treatment includes stopping the transfusion and sending both the patient and donor blood to the blood bank. Alkalization of the urine, IV fluid hydration, and diuresis are essential in addition to Foley catheter placement and measurement of serum bilirubin content.

C. Febrile and allergic reactions are frequent, occurring in about 1% of all transfusions. Reactions are usually mild and are characterized by fever and urticaria. Rarely, they may lead to anaphylaxis if the unit includes antibodies from a hypersensitive donor or if antigens are transfused into a hypersensitive recipient. Treatment may require epinephrine in addition to steroids and antihistamines.

D. Bacterial sepsis may occur as a result of contamination from the container or the donor’s skin. Gram negatives, especially coliforms or Pseudomonas, which can exist at 4°C, are most common. The clinical manifestations include fever, chills, nausea, vomiting, diarrhea, and abdominal cramping. Patients undergoing surgery may demonstrate hypotension and bleeding. Bacterial toxins can produce profound shock. Treatment is supportive: O₂, antibiotics, adrenergic support, and IV fluid. Blood cultures should be sent.

E. Other risks of transfusion include air embolism, thrombophlebitis, acute pulmonary edema, and, rarely, foreign body embolism. Healthy adults can tolerate up to 200 ml of embo- lized air, though smaller amounts may have serious effects. Manifestations may include increased venous pressure, cyanosis, a murmur over the precordium, hypotension, syncope, and respiratory failure. Treatment involves placing the patient with the left side down, head down, and feet up. Thrombophlebitis, usually superficial, is associated with prolonged infusion into a peripheral IV. Treatment includes discontinuing the transfusion and local compression. Superficial vein thromboembolism is rare. Fluid overload is an avoidable complication. Monitoring for an increase in central venous pressure,
dyspnea, cough, and rales is indicated, especially in patients with known heart disease. Rarely, plastic tubes and catheters may embolize if broken off in the vein. If passed into the right atrium or the pulmonary artery, complications may be fatal.

F. The prevention of transfusion-related complications may be improved by the following guidelines. One unit should be transfused at a time, with reassessment of further need upon its completion. Perioperative blood loss should be minimized, oxygen delivery should be maximized, iron supplementation and other methods of increasing red blood cell mass should be utilized, and the use of leukocyte-reduced cells should be considered with each transfusion.

### Table 4.1. Risks of transfusion.

<table>
<thead>
<tr>
<th>Agent/complication</th>
<th>Risk of transmission per unit</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>CMV</td>
<td>Infection endemic: 20% of donors by age 20, 70% by age 60</td>
<td>Carried by WBC, problematic in immunocompromised host</td>
</tr>
<tr>
<td>HIV</td>
<td>1:200,000 to 1:2,000,000</td>
<td>Infectious window 45 days</td>
</tr>
<tr>
<td>Hep B</td>
<td>1:30,000 to 1:250,000</td>
<td></td>
</tr>
<tr>
<td>Hep C</td>
<td>1:50,000 to 1:150,000</td>
<td>Half of these pts develop chronic form, many develop liver dysfunction/cirrhosis</td>
</tr>
<tr>
<td>HTLV-1</td>
<td>0.025% of all donors tested in USA</td>
<td>Transmission to immunocompromised recipient can cause T cell leukemia, spastic paralysis, myelopathy</td>
</tr>
<tr>
<td>Minor allergic reaction</td>
<td>1:100</td>
<td></td>
</tr>
<tr>
<td>Delayed hemolytic reaction</td>
<td>1:1,000</td>
<td></td>
</tr>
<tr>
<td>Acute hemolytic reaction</td>
<td>1:600,000</td>
<td></td>
</tr>
<tr>
<td>Transfusion-related lung injury</td>
<td>1:500,000</td>
<td></td>
</tr>
<tr>
<td>Bacterial contamination</td>
<td>1:500,000</td>
<td>May occur from container or donor’s skin. Toxins can produce profound shock</td>
</tr>
</tbody>
</table>
A. Transfusion Reaction Suspected

Stop transfusion, give epinephrine, steroids, antihistamines

F. Preventative Measures
Assess each transfusion
Minimize periop blood loss
Maximize O₂ delivery
Supplement iron
Consider use of leukocyte-reduced RBC

E. Other Risks
Air embolism
Thrombophlebitis
Pulmonary edema
Foreign body embolism

B. Hemolytic Reaction
Heat/pain in vein, flushed face, pain in lumbar area and chest, respiratory distress, hypotension, tachycardia
Stop transfusion, place foley, send patient’s blood and donor unit to blood bank, measure patient’s serum bili, alkalinate urine, diurese

C. Allergic Reaction
Febrile, urticaria within 60-90 minutes, rarely anaphylaxis
Stop transfusion, give epinephrine, steroids, antihistamines

D. Septic Shock Reaction
Fever/chills, abdominal cramps, vomiting, diarrhea, hypotension/shock
Stop transfusion, culture blood, give O₂, antibiotics, adrenergic support, fluid
5
Asymptomatic Carotid Bruits
Joseph D. Vijungco

A. Carotid Bruits. Stroke is the third leading cause of death in the United States and is caused by carotid occlusive disease in 20–30% of cases. The majority of these will be due to disease at the carotid bifurcation. Only a minority of patients will have had warning symptoms of transient cerebral ischemia; most stroke patients are asymptomatic until the stroke occurs.

A bruit over the midportion of the neck may be a possible indicator of carotid artery stenosis. Its presence is not tantamount to carotid stenosis, but it may indicate the presence of a lesion involving the bulb of the carotid artery. Approximately 20% of patients with carotid bruits will have a hemodynamically significant stenosis of the carotid bulb. However, many patients with documented hemodynamically significant stenosis may not have a bruit. The absence of a bruit does not rule out underlying carotid disease. Patients with risk factors such as coronary artery disease, peripheral vascular disease, hypertension, diabetes, hyperlipidemia, and smoking are at risk for stroke or transient ischemic attacks (TIAs).

There is much controversy surrounding the presence of a cervical bruit and the risk of subsequent stroke or TIAs. Auscultation for carotid bruits is noninvasive and inexpensive, but its ability to detect asymptomatic older adults at risk for stroke has not been completely determined. Early studies found a stronger association of carotid bruits among persons suffering stroke compared with control subjects, but these studies were nonblinded and subject to observer bias. Population-based studies found that persons with carotid bruits were at higher risk of subsequent stroke, but age-specific risks were not reported. A recent study evaluated the natural history of asymptomatic carotid bruits, and showed that the risk of progression of internal carotid artery stenosis steadily increased with time, with an annualized risk of progression of 9.3%. A baseline internal carotid stenosis ≥50% and systolic hypertension are strong predictors of progression.

B. Evaluation. The standard for evaluating patients with suspected carotid bifurcation disease has become the carotid duplex scan. Having established the presence of a high-grade stenosis by duplex scan, an increasing number of surgeons and medical centers feel that further testing before surgery is not necessary. By avoiding an angiogram, the risk related to this invasive procedure is eliminated. In the Asymptomatic Carotid Artery Stenosis (ACAS) trial, the complication rate for carotid endarterectomy (CEA) was 2.7%, but 1.2% of the complications were related to the preoperative angiogram. For those clinicians who want more information, consideration may be given to magnetic resonance angiography, computed tomographic angiography, or rarely, contrast arteriography.

C. Medical Management. Prevention of progressive carotid occlusive disease and stroke must include aggressive medical management. Hypertension should be controlled with medications to keep the systolic blood pressure less than 160 mm Hg. Patients should stop smoking. Other measures include enforcing glucose control in diabetic patients, lowering serum cholesterol, and pursuing regular exercise. Antiplatelet medications have shown a benefit in preventing stroke, TIA, and death from symptomatic carotid artery disease. Available antiplatelet therapies include aspirin, ticlopidine, and clopidogrel. Aspirin reduces the risk of stroke and stroke-related deaths as well as myocardial infarction. Low-dose (80 mg/day) therapy is as effective as therapy with high doses (1,200 mg/day).

Several trials have evaluated surgical therapy versus medical therapy for asymptomatic carotid occlusive disease. The Veterans Affairs Cooperative Study (1987) demonstrated an 8% incidence of subsequent neurologic events (TIA and stroke) in the surgical group versus 20.6% incidence in the medical group. Although this study did not include a sufficiently large sample to evaluate stroke alone as an end point, the incidence of ipsilateral stroke in the surgical group was 4.7% compared with 9.4% in the medical group. The ACAS trial (1995) demonstrated that the risk over 5 years for ipsilateral stroke was 5.1% in the surgical group and 11% in the medical group. This finding provided an absolute risk reduction of 5.9% and a relative risk reduction of 53% in favor of CEA.

D. Carotid Endarterectomy. Prophylactic CEA is a therapeutic option, provided the risk of the procedure does not outweigh its potential benefits. The Stroke Council of the American Heart Association has stipulated that CEA in
asymptomatic patients with severe stenosis should carry a perioperative 30-day mortality rate of less than 3%. On the basis of the ACAS trial, patients with a stenosis of 60% or more and whose general health makes them a good candidate for elective surgery will have a reduced 5-year risk of ipsilateral stroke if CEA is added to aggressive medical management of risk factors.

The indications for CEA include (a) asymptomatic patients with greater than 60% stenosis; (b) symptomatic patients with greater than 70% stenosis; and (c) symptomatic patients with greater than 50% stenosis, an ulcerated lesion, or persistent symptoms while on aspirin. CEA is generally contraindicated in patients with an evolving stroke. Surgery should be delayed until 4–6 weeks have elapsed and should be offered only to those patients who do not have a persistent, severe neurologic deficit.

CEA may be performed under general endotracheal anesthesia, regional cervical block, or local anesthesia. Neurologic function should be monitored if the patient is under general anesthesia; this can be performed with intraoperative electroencephalogram. Shunting devices to maintain perfusion to the internal carotid artery are usually placed selectively on the basis of patency of the contralateral internal carotid artery and intraoperative neurologic assessment.

Postoperative care includes maintaining the systolic blood pressure between 140 and 160 mm Hg, starting aspirin therapy promptly, monitoring the wound for hematoma formation, and frequent neurologic assessment. Complications include stroke, myocardial infarction, cranial nerve injury (hypoglossal most common), and recurrent stenosis.

**E. High risk.** Carotid angioplasty and stenting is a potential new therapeutic option for patients who are at high risk for surgery. Those patients who are considered high risk have the following: (1) multiple comorbidities, (2) a previous history of neck surgery or neck radiation, (3) complete contralateral occlusion of the CIA, and (4) very high carotid lesion or very low carotid lesion near the aortic arch. This new therapeutic modality should be reserved for patients who are enrolled in randomized clinical trials or are in the defined group of high-risk patients.
Asymptomatic Carotid Bruits

A. Carotid Bruits
   stroke-3rd cause of death
   carotid source in 20-30%
   most are asymptomatic
   stenoses progress at 9%/yr

B. Evaluation
   carotid duplex scan

1. control hypertension
2. control diabetes
3. stop smoking
4. regular exercise
5. lower cholesterol
6. antiplatelet therapy
   (controversial)

C. Medical Management
   arrest progression of occlusive
disease, prevent stroke

D. Carotid Endarterectomy
   to be justified for asymptomatic patients,
mortality should not exceed 3%

E. Endovascular Stenting

Indications:
1. asymptomatic pts, > 60% stenosis
2. symptomatic pts, > 70% stenosis
3. symptomatic pts with an ulcerated
   lesion or persistent symptoms while
   on aspirin, > 50% stenosis

Intra-operative Management:
1. monitor neurologic function
2. selective shunting

Post-operative Management:
1. control systolic pressure
2. start aspirin therapy promptly
3. monitor wound for hematoma
General Operating Room Precautions

Jonathan A. Myers

A. Introduction: Health-care personnel (HCP) are exposed to a variety of body fluids containing infectious diseases in the operating room. Among these, hepatitis B virus (HBV), hepatitis C virus (HCV), and human immunodeficiency virus (HIV) are the most prevalent. The Centers for Disease Control and Prevention (CDC) has recorded 56 documented cases of occupationally acquired HIV infection among HCP through June 2000. Actual numbers may be significantly higher since many exposures go unreported. Of the documented cases, 86% acquired the infection percutaneously while 10% had mucocutaneous exposure, and 45% developed AIDS. The most common vehicle for transmission is infected blood (88%), with exposure to other body fluids or concentrated virus in the laboratory comprising the remainder. The estimated risk of HIV infection after percutaneous exposure to HIV-infected blood is 0.3%. Risk of infection from contact with mucous membranes is significantly lower. In contrast, the risk of hepatitis B infection from percutaneous exposure is 30%. Infection from hepatitis C exposure is reported at 1.8%.

The risk of infection increases if the exposure involves a larger quantity of blood. Examples of this include devices that are visibly contaminated with blood, procedures involving a needle placed directly into an artery or vein, and deep injuries. Hollow bore needles carry a higher risk than do suture needles.

C. Handling Equipment: The overwhelming majority of occupational infections occur from percutaneous punctures by needles or other sharp instruments. Many of these injuries can be prevented by adhering to the following guidelines. Disposable needles are intended for single use only and should be discarded, along with scalpel blades and other sharp items, in a puncture-resistant container. Disposable needles should not be detached from the syringe and recapping the needle after use should be avoided. Needles should not be bent, broken, or otherwise manipulated by hand. Hand-to-hand transfer of sharp instruments should also be avoided.
**Hepatitis B virus:** HBV postexposure management involves administration of the hepatitis B vaccine series to any unvaccinated HCP who sustains an exposure. After evaluating the hepatitis B surface antigen status of the source, PEP with hepatitis B immune globulin (HBIG) and/or hepatitis B vaccine series should be considered.

**Hepatitis C virus:** Studies have not demonstrated any protective antibody response with administration of immune globulin following HCV exposure. Clinical trials with antiviral agents (interferon with or without ribavirin) have not been conducted for prevention of HCV infection. Therefore, the recommendations for postexposure management of HCV include early identification of disease with follow-up testing. The source should be tested for anti-HCV. The exposed HCP should have baseline testing for anti-HCV and liver enzymes, and follow-up testing at 4–6 months. If the HCV status of the HCP becomes positive, prompt referral for evaluation and treatment is initiated.

**Human immunodeficiency virus:** Studies with the use of zidovudine (ZDV) alone for PEP demonstrate a reduction in the risk of HIV infection by 81%. Combination drug regimens in HIV-infected patients have proven superior to monotherapy in reducing viral load; thus, combining agents could theoretically provide additional preventive benefit to exposed HCP undergoing PEP. There are currently five classes of antiretroviral agents available to treat HIV infection. These include the nucleoside reverse transcriptase inhibitors (NRTIs), nucleotide reverse transcriptase inhibitors (NtRTIs), protease inhibitors (PfIs), and a simple fusion inhibitor. PEP for HIV exposure is based on the exposure type (less severe vs. more severe percutaneous injuries or small volume vs. large volume mucous membrane and nonintact skin exposure) as well as the infection status of the source (asymptomatic individuals with low viral load, symptomatic individuals, AIDS, high viral load, source with unknown HIV status or unknown source). Once this information is obtained, a decision is made to consider basic 2-drug PEP, recommend 2-drug PEP, or recommend expanded ≥3-drug PEP. This should be instituted as soon as possible after exposure. If tolerated, PEP should be administered for 4 weeks. It is stressed that the treatment regimen must be discussed with the infectious disease physician prior to initiation to assess the most effective option. Side effects of these medications include gastrointestinal symptoms, headache, and fatigue, among others. More serious side effects, including nephrolithiasis, hepatitis, and pancytopenia, have been reported from using a combination of medications for PEP. In addition to the source being tested for HIV, the exposed HCP should receive follow-up counseling, medical evaluation, and postexposure testing. This includes a baseline HIV blood test, as well as further testing at 6 weeks, 12 weeks, and 6 months unless PEP has been initiated in which case blood will be analyzed more frequently for drug-toxicity monitoring. Seroconversion beyond 6 months is very rare.

Following exposure to any of these infectious agents, fever, rash, or lymphadenopathy in the HCP should be investigated; causes include infection, drug reaction, or other medical condition. In addition, exposed HCP should avoid unsafe sexual behaviors and other practices that might promote transmission to others.
General Operating Room Precautions

A. Introduction

B. Universal Precautions
- use gloves, mask, gown, eyewear.

C. Handling Equipment
- avoid recap personally交易中心 needle and hand to hand transfer.

D. Postexposure Guidelines
1. Clean Wound
2. Contact EMS or ER
3. Consult ID
4. Draw Pt’s blood for HIV, HBV, HCV

HBV PEP Guidelines
1. Assess HCP HBV series
2. Give HBIG and/or hep-B vaccine

HCV PEP Guidelines
1. Test HCP for HCV and liver enzymes immediately and @ 4-6 mos
2. If becomes (+) refer for evaluation and tx

HIV PEP Guidelines
1. Baseline HCP HIV test
2. Determine exposure type + & infection status of source
3. Base 2 vs > 3 drug PEP regimen or type + source information with ID
4. Follow counseling, medical evaluation, and postexposure testing with blood test @ 6 weeks, 12 weeks, 6 months

Monitor for signs of dz
Avoid practices promoting transmission to others

HBV PEP Guidelines
1. Assess HCP HBV series
2. Give HBIG and/or hep-B vaccine

HCV PEP Guidelines
1. Test HCP for HCV and liver enzymes immediately and @ 4-6 mos
2. If becomes (+) refer for evaluation and tx

HIV PEP Guidelines
1. Baseline HCP HIV test
2. Determine exposure type + & infection status of source
3. Base 2 vs > 3 drug PEP regimen or type + source information with ID
4. Follow counseling, medical evaluation, and postexposure testing with blood test @ 6 weeks, 12 weeks, 6 months

Monitor for signs of dz
Avoid practices promoting transmission to others
Part II
Trauma
Initial Assessment and Resuscitation of the Trauma Patient

Frederic Starr

In the United States, accidental injury is the leading cause of death for people between the ages of 1 and 44. Traumatic injury of all causes is responsible for over 160,000 deaths annually. Over 40 million people will seek medical care because of intentional or accidental trauma this year, accounting for nearly 40% of all emergency department visits in the USA.

The injured patient must be assessed quickly and treatment of life-threatening injuries begun immediately. A systematic approach to this evaluation ensures that the most critical injuries are identified early, and that potentially lethal injuries are not missed. The “ABCDE” primary survey is the most widely accepted initial approach. This strategy also provides a framework for reevaluation if the patient’s condition deteriorates, redirecting the physician back to the start of the algorithm in search of a missed or worsening injury.

Once the primary survey is complete and the patient’s vital signs are normalizing, the secondary survey is begun. Preparations: Before the patient’s arrival, the hospital trauma team should be mobilized, including notification of radiology, blood bank, respiratory therapy, and the operating room. IV fluids should be warmed, and the trauma bay should also be warmed if possible. The team should follow standard precautions (cap, gown, gloves, mask, shoe covers, face shield).

A. Airway: Evaluation of the patient’s airway is the first priority. Spontaneous speech in an awake patient indicates a patent airway. Severe facial trauma or depressed level of consciousness may cause airway obstruction which can be improved with a jaw thrust or chin lift maneuver. Indications for intubation include inability to protect the airway, profound shock, or a Glasgow Coma Scale (GCS) score less than or equal to 8. Nasotracheal intubation requires a spontaneously breathing patient and is contraindicated in patients with severe facial trauma or suspected basilar skull fractures. The orotracheal route is generally the preferred method for airway control. Inability to intubate mandates a surgical airway, with cricothyroidotomy the preferred procedure in adults. Needle cricothyroidotomy is favored in children younger than 12 years because of their narrow cricoid ring. All patients with blunt head trauma or altered mental status should be presumed to have a cervical spine injury until proven otherwise. Inline cervical spine stabilization should be maintained at all times when securing a definitive airway.

B. Breathing: Once the airway is assessed and secured, all trauma patients should be started on supplemental oxygen. Breathing first should be evaluated by inspection, looking for external signs of injury, asymmetry of chest rise, paradoxical motion, and the use of accessory respiratory muscles. Tracheal deviation or the presence of distended neck veins should be noted. The chest should also be palpated to identify areas of tenderness or subcutaneous emphysema. Percussion may illicit hyperresonance or dullness, indicating pneumothorax or hemothorax, respectively, though in a noisy trauma bay this may be impractical. Finally, auscultation may demonstrate absent or diminished breath sounds from a hemo/pneumothorax or malpositioned endotracheal tube. Injuries that must be identified and addressed during the primary survey include tension pneumothorax, massive hemothorax, flail chest, and open pneumothorax. A tension pneumothorax should be suspected in a hypotensive patient with absent breath sounds, hyperresonance, distended neck veins, and deviated trachea. Treatment is immediate needle decompression (before the chest x-ray) with a large gauge angiocatheter through the second intercostal space in the midclavicular line. Massive hemothorax is defined as greater than 1500 ml of blood within the pleural space, and initial treatment requires prompt chest tube placement (36–40 Fr). Flail chest occurs with segmental fractures in three or more adjacent ribs. The danger lies in the underlying pulmonary injury as well as resultant hypventilation secondary to pain. Treatment is supportive, occasionally requiring mechanical ventilation. Lastly, an open pneumothorax (“sucking chest wound”) occurs with chest wall defects that are greater than two-third the diameter of the trachea, resulting in air flowing predominantly through this new path of least resistance. A flutter valve should be created using an occlusive dressing taped on three sides, allowing air to flow...
out but not in with each breath. A chest tube must be inserted, and ultimately the defect closed.

C. Circulation: Shock, defined as inadequate organ perfusion and tissue oxygenation, can be categorized as hemorrhagic, neurogenic, cardiogenic, or septic. Hypotension in a trauma patient is due to blood loss until proven otherwise, and the degree of hemorrhage can be estimated quickly by physical exam. Level of consciousness, skin color, pulse rate, and pulse pressure rapidly reflect volume status. Systolic blood pressure, however, usually does not fall until 30% of the blood volume is lost, and therefore may deceive one into a false sense of security. The most important concept is locate and stop the bleeding. External bleeding must be identified and controlled, usually with direct pressure. Other possible locations of hemorrhage include the thorax, abdomen, retroperitoneum, and extremities. Intravenous access must be established, generally with two 16-gauge (or larger) antecubital IVs, and a 2-L bolus of lactated Ringer’s solution given. Triple lumen catheters should be avoided because of their high resistance to flow (long tube and small diameter). Instead, a 7.5 French cordis is preferable, generally placed in the femoral vein. If the patient improves hemodynamically, the fluids are decreased to a maintenance rate. If there is no response to the initial bolus, one must consider transfusing blood (O negative or type-specific) and then other potential causes of shock must be entertained. Tension pneumothorax has been discussed. Neurogenic shock is characterized by hypotension in the face of a normal heart rate (or even bradycardia due to unopposed vagal cardiac stimulation). It occurs with spinal cord injury and NOT brain injury, and treatment consists of fluids initially, followed by vasopressors if necessary. Pericardial tamponade is generally associated with penetrating injuries and can be recognized by hypotension, tachycardia, jugular venous distention (JVD), and muffled heart sounds. Echocardiography is the mainstay of diagnosis. Treatment is immediate operation, though pericardiocentesis can stabilize the patient until the operating room is available or transfer to an appropriate facility can be achieved. Cardiogenic shock can also be due to blunt cardiac injury, which is diagnosed by electrocardiogram (EKG) and echocardiogram. Treatment is supportive. Septic shock is extremely rare in the acute trauma setting.

D. Disability: Brain or spinal cord injury can be detected by a brief neurologic exam. Before any paralytic agents are given for intubation, movement of all four extremities should be assessed and lateralizing signs noted. Abnormal pupillary exam, including size and reactivity, can indicate intracranial injury. The GCS is the most widely used assessment of level of consciousness and incorporates the best exam score in three categories—eye opening, verbal response, and motor response. Scores range from 3 (worst) to 15 (best). One advantage of using the GCS is its reproducibility and simplicity, allowing frequent reevaluations by different physicians. All patients with suspected head injury require emergent noncontrast head CT for rapid diagnosis and differentiation of operative and nonoperative pathology. Severe head injury with elevated intracranial pressure is treated initially with mannitol and mild hyperventilation. Furthermore, cervical spine injury must be assumed with all blunt trauma, and cervical immobilization with a C-collar employed until bony and ligamentous injury can be ruled out.

E. Exposure: The patient should be completely exposed (including rolling), and the entire body examined for signs of injury. A rectal exam should be performed. To avoid hypothermia, the patient should then be covered with warm blankets once the inspection is complete. Again, IV fluids and blood should be warmed.

Adjuncts: During the primary survey, blood pressure, EKG, and pulse oximeter monitors should be placed. Supplemental oxygen should be administered. If clinically indicated, a nasogastric tube and Foley catheter are inserted. Basic imaging, including chest, pelvis, and lateral cervical spine radiographs, as well as FAST scan can also be obtained at this point.

F. Secondary Survey: Once the primary survey is completed and the patient’s vital signs are normalizing, a thorough head-to-toe exam is performed. A complete neurologic exam is performed. A brief “AMPLE” history (A = allergy, M = medications, P = past medical/surgical history, L = last meal, E = events of injury) should be taken. Any change in the patient’s condition mandates reassessment of the primary survey. If the patient’s injuries require management not immediately available at the current hospital, transfer to a higher level of care must be considered and arrangements initiated. Do not let diagnostic studies delay transfer to definitive care.
A. Airway
- Speaking?
  - Yes →
  - Protecting airway?
    - Shock?
      - GCS>8?
        - Chin lift/jaw thrust
          - Definitive airway
            - orotracheal intubation
            - circothyroidotomy

  - NO →
  - Chin lift/jaw thrust

B. Breathing
- Inspection
- Palpatation
- Auscultation
- Supplemental O₂
- Tension PTX?
  - Needle decompression/chest tube
- Massive HTX?
  - Chest tube
- Flail chest?
  - Chest tube
- Open PTX?
  - Flutter valve/chest tube

C. Circulation
- BP, HR, Pulse pressure
- Physical exam
- External bleeding - direct pressure
- IV access
  - Bolus
  - No response
    - Ongoing bleeding?
      - Tension PTX?
      - Neurogenic?
      - Cardiogenic?
      - Septic?

D. Disability
- Neurrologic exam
- GCS
  - Immobilize C-Spine
  - CT head
  - Neurosurgical consult
  - Mannitol/hyperventilate?

E. Exposure
- Complete exposure
- Rectal exam
- Avoid hypothermia
  - Vital signs normalizing
    - Yes →
      - F. Secondary Survey
        - AMPLE history
        - Head to toe physical exam
        - Complete neurologic exam
        - Consider transfer to higher level of care
    - NO
A. Initial Resuscitation: A systematic approach to the trauma patient with a suspected head injury is essential in order to provide an expedient diagnosis and treatment. The head is one of the most commonly injured structures of the body; trauma can cause a constellation of symptoms including a decrease in mental status or loss of consciousness. Other etiologies of loss of consciousness must also be considered including shock, seizures, metabolic disturbances, and intoxication.

As with all trauma patients, ATLS (Advanced Trauma Life Support) guidelines recommend management of the “A, B, C’s” (Airway, Breathing, and Circulation) first. Patients who are spontaneously breathing may be observed. However, patients who present with altered mental status and inability to protect their airway need to be intubated, either orally or nasally. Head-injured patients have a high likelihood of cervical spine injury; therefore, cervical spine stabilization should be maintained at all times, even when intubating. Breathing should be assessed next with auscultation of the chest cavity for breath sounds. Definitive treatment is needed for pneumothorax or other injuries affecting ventilation. Circulation is assessed by taking the patient’s pulse and blood pressure. Two large-bore peripheral IVs should be placed and resuscitation started with crystalloid fluid and blood, if needed for severe hypotension. Hypotension and hypoxemia can be devastating for the patient with a head injury and may actually cause secondary insult to the brain. All efforts should be made to maintain blood pressure and oxygenation for adequate cerebral perfusion.

B. Assessing Neurological Status: Primary survey of the head-injured patient also entails a neurological examination. The Glasgow Coma Scale (GCS) is a rapid reproducible way of evaluating the severity of the brain injury and is used to follow the patient’s progress over time (Table 8.1). Patients are evaluated in three categories using the best response in eye opening, motor, and verbal. Scores range between 3 and 15, with 3 being no response to any stimulus and is the lowest score. Any patient suspected of having increased intracranial pressure (ICP) or a GCS score between 3 and 8 should be treated with intubation and mild hyperventilation to maintain $pCO_2$ between 30 and 35 mm Hg. Hyperventilation should only be used for short periods of time, as this has been shown to increase areas of ischemia in the injured areas of the brain.

A more complete head examination should be included in the secondary survey of the injured patient. Signs of head injury include seizure activity, hemiparesis, and decerebrate or decorticate posturing. Pupillary light response, gag reflex, cough reflex, corneal reflex, and Babinski’s reflex are important to evaluate brain stem function. Raccoon eyes or periorbital ecchymosis, postauricular ecchymosis (bruising behind the ears), hemotympanum, signs of CSF leak (otorrhea or rhinorrhea), or injury to cranial nerves may be signs of a basilar skull fracture. The cranium should be checked for skull fractures or depressions. Large lacerations should be gently probed to check for open fractures.

C. Differential Diagnosis: Other causes of depressed mental status should be quickly considered. Glucose levels should be checked and treated. The presence of alcohol and drugs should be checked with blood and urine alcohol and drug screens. Administration of naloxone or flumazenil may be considered for emergent reversal. Also, for patients who are suspected of having consumed alcohol, 100 mg of intravenously thiamine is administered to treat possible Wernicke’s encephalopathy.

D. Radiographic Assessment: Once the initial stabilization and evaluation are complete, the patient is taken as quickly as possible for a noncontrast CT scan of the brain. Abnormalities sought for include mass lesions (e.g., subdural or epidural hematomas), midline shift, cerebral edema, subarachnoid hemorrhage, cerebral contusions, or hematomas.

E. Surgical Emergencies: Care should be rendered at a facility where neurosurgical care is available around the clock. If this is not available at the initial facility, transfer of the patient is essential.

1. *Acute subdural hematomas* are life-threatening extra-axial blood collections caused by tearing of the bridging veins found between the cerebral cortex and the overlying dura. Subdural hematomas are often associated with more severe
generalized brain injuries and cerebral contusions. They appear as a concave rim of localized blood around the brain on CT scans. Subdural hematomas greater than 1 cm are often associated with decreased mental status and midline shift. Treatment is with craniotomy and decompression.

2. **Epidural hematomas** are usually caused by blunt trauma to the head, producing tears in the meningeal vessels, typically the middle meningeal artery in the temporal area. They may also be associated with skull fractures, which traverse the superior sagittal or transverse sinuses. The history is that of a patient who has a transient loss of consciousness after a head injury, followed by a lucid phase and then neurological deterioration. Epidural hematomas have a convex shape on CT. Most epidural hematomas require emergent evacuation in the operating room.

3. **Depressed skull fractures** require surgical elevation when the depth of the depression meets or exceeds the thickness of the adjacent skull or is greater than 8–10 mm. Also, they require elevation when a cerebrospinal fluid leak is suspected or when a neurological deficit is related to pressure or injury of the underlying brain by the fracture. Open depressed fractures should be treated promptly to minimize the risk of infection and antibiotics should be administered.

4. **Penetrating injuries** and gunshot wounds to the cranium usually have a very poor prognosis and are often accompanied by intra- or extra-axial cerebral hematomas, contusions, and bone fragments or debris in the tracks. Treatment is very controversial. Some recommend debridement of the track and removal of devitalized brain with closure of the dura and broad-spectrum antibiotics, while others recommend local debridement of the entry/exit sites and closure followed by broad-spectrum antibiotics.

F. **Nonsurgical:** Emergencies are best treated by controlling the ICP. Patients with a GCS score of less than 8 should be intubated and treated with mild hyperventilation. Hyperventilation for long periods has fallen out of favor and is recommended only for brief periods (<24h) or when the ICP is refractory to treatment by other means. These patients should also have ICP monitors placed. Ventriculostomies may be considered. Therapy is indicated when the ICP is above 20. Mannitol causes an osmotic diuresis and is indicated when there is progressive neurological deterioration, ICP above 20, or evidence of herniation (unilateral or bilateral pupillary dilatation, asymmetric pupil reactivity, or motor posturing). A dose of 1 g/kg is given over 15 min. Patients should be watched for hypovolemia and hypotension and may need fluid replacement. Serum osmolality should be kept below 320 mOsm. An alternative to mannitol is hypertonic saline, 7.5% in 30 ml bolus or a 3% saline drip; the goal is to keep the serum sodium between 155 and 160. ICP may also be controlled with sedatives (diprovan, benzodiazepines), paralytics, or narcotic pain medications. If these treatments fail, the last resort may be to induce a barbiturate coma.

1. **Subarachnoid hemorrhage** is bleeding into the subarachnoid space, which is located between the surface of the brain (pia mater) and the arachnoid. *This usually does not cause a mass effect* because it is spread diffusely. Vaso-spasms may occur several days after the injury and is treated with calcium channel blockers and hydration. The goal of treatment is supportive care to keep the ICP within an acceptable range.

2. **Intracerebral hematomas and contusions** are scattered areas of hemorrhage that form over the surface of the brain, most commonly along the undersurface and poles of the frontal and temporal lobes. They arise when the brain strikes a ridge on the skull or a dural fold. Cerebral edema develops around contusions within 48–72h after injury. If there is considerable pressure effect (causing raised ICP) or if the hemorrhage progresses to form a sizeable blood clot in the brain (an intracerebral hematoma), surgical removal through a craniotomy may be required.

3. **Diffuse Axonal Injury (DAI)** may occur in patients with blunt head trauma. CT scan of the brain may be unimpressive, but the patient has profound neurological deficits. Injury to the axon causes structural failure at the mictotubular level. MRI may be helpful in defining the injury 1–2 weeks after. There is no treatment for DAI, except supportive care to control the ICP.

### Table 8.1. Glasgow Coma Scale.

<table>
<thead>
<tr>
<th>Points</th>
<th>Eye opening</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Spontaneous</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>To voice</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>To stimulation</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Points</th>
<th>Motor response</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>To command</td>
</tr>
<tr>
<td>5</td>
<td>Localizes</td>
</tr>
<tr>
<td>4</td>
<td>Withdraws</td>
</tr>
<tr>
<td>3</td>
<td>Abnormal flexion</td>
</tr>
<tr>
<td>2</td>
<td>Extension</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Points</th>
<th>Verbal response</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Oriented</td>
</tr>
<tr>
<td>4</td>
<td>Confused but comprehensible</td>
</tr>
<tr>
<td>3</td>
<td>Inappropriate or incoherent</td>
</tr>
<tr>
<td>2</td>
<td>Incomprehensible (no words)</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
</tr>
</tbody>
</table>
Head Trauma

A. Initial Assessment
   “ABC’s”

B. Neurologic Exam
   Glasgow Coma Scale (table 1)
   intubate if score 3-8

C. Differential Diagnosis
   hypoglycemia, alcohol intoxication, drugs

D. Radiographic Assessment
   CT head

E. Surgical Emergencies
   1. acute subdural hematoma-decompression
   2. epidural hematoma-decompression
   3. depressed skull fracture-possible surgery
   4. penetrating injuries

F. Non-surgical Emergencies
   1. subarachnoid hemorrhage-support
   2. intracerebral bleeding-possible surgery
   3. diffuse axonal injury-support
A. Need for an Airway: It is important to assess the airway of every trauma patient encountered. This may be as simple as asking the patient a question and listening to voice quality as he/she responds. Indications for further airway intervention include apnea, respiratory distress, airway obstruction, and inability to protect the airway. A patient with a depressed level of consciousness due to head injury or shock will be unable to protect the airway and may have airway obstruction due to the tongue falling posteriorly into the oropharynx.

B. Initial Maneuvers: When it is decided that a patient needs an airway intervention, the oropharynx should be cleared of secretions and foreign bodies. This may be done by (carefully) sweeping a gloved finger in the back of the patient’s mouth. A Yankauer suction is used to remove secretions such as blood, saliva, and vomitus. Two quick methods for opening the airway are the chin lift and the jaw thrust. The chin lift is performed by pulling anteriorly on the patient’s mentum. The jaw thrust is performed by pushing both angles of the mandible anteriorly. Both can be done without any flexion or extension of the cervical spine. Any patient who has sustained trauma above the clavicles should be considered at risk for a cervical spine injury and care should be taken not to manipulate the neck. If there is no concern for cervical spine injury, the patient may be placed in the “sniffing position” with his occiput elevated and his neck extended. This will further open the airway.

The airway may be maintained in an open position by the use of an oral airway or a nasal airway. The nasal airway is preferred in awake patients because the oral airway may induce a gagging sensation. The nasal airway is contraindicated in the presence of a coagulopathy, midface trauma, or a suspected basal skull fracture.

C. Definitive Airway: Most patients require a more definitive airway than that provided by the oral or nasal airway. These patients should have an endotracheal intubation performed. It is important that all equipment be checked prior to embarking upon this. The physician should don gloves, mask, and eye shield and ensure that the laryngoscope light, suction, and endotracheal cuff are in working order. The ambu bag should be connected to an appropriate size face mask and an oxygen source. Assistants are useful for cervical spine immobilization or cricoid pressure. If rapid sequence intubation is being performed, intravenous access should be present. An appropriately sized laryngoscope blade should be used. The MacIntosh (curved) blade is useful for patients with large tongues but requires more skill to place into the vallecula. The Miller (straight) blade is used to lift the epiglottis directly. After ensuring that the patient’s oxygen saturation is 100%, the mask is removed and the mouth is opened with the right hand. The laryngoscope is held in the left hand and advanced into the oropharynx. Once in place, the laryngoscope is pulled anteroinferiorly (not levered onto the upper teeth) and the vocal cords are visualized. The endotracheal tube is advanced through the cords under direct visualization. Both lung fields and the epigastrium are auscultated to confirm proper tracheal placement, the tube is secured to the patient’s face, and a chest radiograph is obtained to confirm proper position.

Nasotracheal intubation may be used in the awake patient; however, it is discouraged because of the relatively high incidence of sinusitis and otitis resulting from prolonged intubation.

Rapid sequence induction is a valuable adjunct to oral endotracheal intubation. This requires intravenous access. There are several different drugs which may be given. A common “cocktail” includes an induction agent such as thiopental or etomidate and a short-acting muscle relaxant such as succinylcholine or vecuronium. If there is a concern of intracranial hypertension (as in a head-injured patient), lidocaine and a defasciculating agent such as “mini-dose” succinylcholine or pavulon should precede the induction agent. It is important to maintain cricoid pressure continuously beginning with the administration of drugs until the tube position has been confirmed by auscultation.

Occasionally, it will be difficult to obtain an airway using this standard technique. If the patient remains 100% saturated with ambu-bagging, retrograde intubation may be attempted. This is done by inserting an 18-gauge needle through the
cricothyroid membrane into the airway. A guide-wire (from a central line insertion kit) is then advanced through the needle retrograde into the oropharynx. Using a laryngoscope to visualize the posterior pharynx, the wire is grasped with a Magill forceps and retrieved through the mouth. The endotracheal tube is then threaded onto the wire and advanced into the trachea. Once the tube is through the vocal cords, the wire is removed and tube position is confirmed by auscultation.

*Fiberoptic intubation* may be useful in certain circumstances. This requires a spontaneously breathing patient and a physician who is skilled in using the flexible bronchoscope. This technique is not indicated for emergent airways because time is of the essence. Adequate local anesthesia of the nasopharynx and oropharynx are essential. The endotracheal tube is placed over the bronchoscope before insertion, when the vocal cords are visualized, the tube is then advanced directly into the airway. Many trauma patients are not candidates for this technique because excess blood and secretions may hinder visualization. In addition, patients with penetrating neck trauma may gag on the bronchoscope, leading to a Valsalva, which may result in bleeding from a carotid or jugular injury. These patients are better candidates for rapid sequence induction technique with a surgeon prepared to perform a cricothyroidotomy if intubation is unsuccessful.

D. *Surgical Airway*: If the patient is desaturating and an immediate airway is necessary, a surgical cricothyroidotomy should be performed if attempts at oral intubation have been unsuccessful. A 2–3-cm transverse incision should be made directly over the cricothyroid membrane. This incision should be carried through the skin, subcutaneous tissue, and membrane. Once the airway is entered, a tracheal hook is placed to stabilize the thyroid cartilage. The membrane may be gently dilated with a tracheal dilator or hemostat. The tracheostomy tube is then placed through the cricothyroid membrane into the trachea. *This tube should be no larger than a #6 Shiley or 7.0 endotracheal tube to minimize trauma to the thyroid and cricoid cartilages.* Once inserted, placement is confirmed by auscultation and the tube is secured to the patient.

A transverse incision is best as it facilitates rapid access through the cricothyroid membrane. If laryngeal pathology such as a fracture is suspected, the surgeon may choose a longitudinal midline incision instead. This incision may more easily be extended inferiorly if an emergent tracheostomy is necessary due to distorted laryngeal anatomy.

E. *Pediatric Intubations*: Pediatric intubations require special consideration. Very young children should be pretreated with atropine prior to manipulation of their airway. The ambu bag, mask, and endotracheal tube must be the appropriate size for the child; size may be estimated by comparing the tube to the child’s fifth finger or the internal diameter of the tube may be calculated as \((\text{age in years}/4) + 4\). *Children younger than 8 years should have an uncuffed tube placed.*

*Surgical cricothyroidotomies are contraindicated in children younger than 8 years.* If endotracheal intubation cannot be performed, a needle cricothyroidotomy may be necessary. This is done by inserting a large-bore (14/16-gauge) angiocatheter through the cricothyroid membrane. Once air is aspirated, the metal needle is removed and the hub of the catheter is connected to an ambu bag or ventilator tubing. This is performed by using the adapter from a 3.0 endotracheal tube to connect the catheter to the standard ambu bag or ventilator connection. Needle cricothyroidotomy will provide adequate oxygenation and ventilation for a few hours until a formal tracheostomy can be performed. This catheter is too small to adequately ventilate an adult patient.
Management of the Difficult Airway

A. Airway Needed?
- Apnea
- Respiratory Distress
- Airway Obstruction
- Can’t protect airway

B. Initial Maneuvers
- Chin lift
- Jaw thrust
- Oral nasal airway

C. Definitive Airway
Endotracheal Intubation

D. Surgical Airway
Contraindicated for children younger than 8 years

E. Pediatric Intubation
Uncuffed tube for Children < 8 years old
The neck has many vital structures packed into a small amount of space vulnerable to penetrating trauma. Injuries to the airway and arteries that course this region are the primary cause of immediate morbidity and mortality. But just as morbid are unidentified/unassuming-isolated injuries to the esophagus. Penetrating injury may be caused by projectile or blade with each assuming different subtleties in management. Penetrating injury caused by projectiles is associated with a higher incidence of significant injury, yet a stab wound tract commonly is more difficult to delineate, often underestimating the potential for and significance of injury. Although, the majority of isolated Zone II injuries (~50%) are caused by knife wounds, surgical management is more frequently required in those sustaining gunshot wounds (GSW); 60 and 80% respectively. Absolute indications, regardless of mechanism, such as hemorrhage, expanding hematoma, neurologic deficit, airway compromise, or impaled object necessitate emergent surgical management. Other symptoms such as subcutaneous emphysema, hemoptysis, stridor, odynophagia, or dysphagia also often necessitate urgent surgical exploration.

The neck is divided into three zones by horizontal planes. Two different classifications are described based on different borders. Monson et al. in 1969 described the first classification. Here, Zone I is described as below the medial head of the clavicles, Zone II between the medial head of the clavicles and the angle of the mandible, and Zone III above the angle of the mandible. This classification was proposed to better identify areas of the neck that warrant further diagnostic studies (namely angiography) prior to operative management in preparation of appropriate and possibly complicated exposure. Later, a second definition was introduced changing the borders of Zone I and II to the cricoid cartilage. It is important when reviewing this literature to make this distinction for comparison of outcomes. Zone I injuries often necessitate median sternotomy or clavicle resection while Zone III injuries may require mandibular disarticulation; hence the need for further diagnostic modalities in preparation for operative management.

A. The first priority in trauma management is the institution of the American College of Surgeons Advanced Trauma Life Support guidelines. The airway is insured of patency and protection, breath sounds are confirmed bilaterally and circulation is identified with peripheral pulses. If the patient requires an artificial airway, a surgical tray should be available because tracheal injury or neck hematoma may often render an endotracheal intubation unsuccessful. Many advocate bronchoscopic aid in intubation so as not to create a false passage with direct laryngoscopy. Regardless of intubation, a chest radiograph (CXR) is promptly obtained to identify potential thoracic pathology (i.e., pneumothorax, pneumomediastinum, hemothorax, or subcutaneous emphysema). Once the primary survey is complete, a better evaluation of the neck and all other possible injuries can ensue.

B. Physical examination of the neck first takes into account the previously listed absolute indications for immediate operative intervention regardless of zone of injury. If immediate operative intervention is required, the patient should be prepped and draped for a possible median sternotomy or mandible disarticulation and neck exploration performed. An incision is made along the anterior border of the sternocleidomastoid muscle and the course of the wound explored.

C. If absolute indications for exploration are not present, a more thorough examination of the neck is performed after all immediate other life-threatening injuries are addressed.

D. Local wound exploration is next performed to identify depth of penetration. If the platysma is deemed intact or not penetrated, no further work up is necessary.

E. When the platysma is deemed violated, the next priority is to identify which zones are involved. GSWs are managed only slightly different here. An AP neck film is obtained for GSWs with a presumed retained projectile to identify potential multiple zones of involvement. A CXR will identify any projectile that exited the neck into the thorax.
F. Involvement of Zone I, III, or multiple zones require arteriography to evaluate for a potential vascular injury and esophagography for a potential esophageal injury. If esophagography is equivocal, then rigid esophagoscopy is indicated. Again, this is done for these zones of injury for (1) preparedness of an extensive surgical approach outside of a standard neck exploration and (2) to possibly obviate surgical management. Also, many Zone III injuries are inaccessible via surgery and transcatheter embolization may be required for definitive management. Bronchoscopy and laryngoscopy is performed in stable patients with upper airway trauma evidenced by dyspnea, hemoptysis, subcutaneous emphysema, or air bubbling through the wound.

Multiple zones of injury secondary to stab wounds (SW) are more difficult to appreciate because depth and angle of penetration are not easily determined. Therefore, Zones I and III injuries are managed as previously described above without the need for plain films of the neck. A CXR may also aid in identifying thoracic involvement. A distinction between a stab and slash wound will also dictate different management tracts. A true slash wound essentially exposes the base of the injury obviating the need for further diagnostic studies and dictating surgical management as needed (including a complex closure).

G. The greatest controversy in penetrating neck trauma is the management of isolated Zone II injuries. Selective management including four-vessel angiography, esophagography, and panendoscopy has been advocated because of the high negative neck exploration rate (up to 76%). Proponents of mandatory neck exploration however believe that any delay in operative intervention leads to higher morbidity and mortality. It is also associated with a low morbidity and most patients can be discharged within 24 h after operative management. Most recent reviews do not show a greater benefit with either procedure. The decision to perform selective management or mandatory exploration should be made with each individual institution based on afforded resources. One caveat should be considered. Many proponents of selective management cite a significant strain on resources to perform mandatory exploration. This strain can be no greater than that performed coordinating and completing the multiple diagnostic studies with selective management.

H. The definitive management of carotid artery injuries is somewhat controversial. Multiple factors have been considered to direct surgical repair including ischemic time, neurological impairment, presence of carotid flow, etc. Many recent studies have shown improved success of these injuries with surgical repair despite neurologic exam. Earlier contraindications to repair and reestablishment of flow were based on the possibility of converting an ischemic infarct to a hemorrhagic one. Kuehne from the University of Southern California prospectively studied revascularization in internal carotid artery injuries and showed improved results despite neurologic impairment. It is thought that the “reversibly ischemic penumbra” benefits from revascularization even in the “hemiplegic comatose patient”. However, a dense neurologic deficit with carotid artery occlusion and a hemispheric infarct still proposes a poor prognosis despite surgical management. Venous injuries are ligated with impunity. Esophageal injuries should be primarily repaired and drained with a soft flexible drain. Tracheal injuries are also primarily repaired. Tracheostomy placement is based on level of injury but is not commonly required.

Recently, publications have pointed to physical examination as means of identifying diagnostic and therapeutic management of vascular injuries. Although the evidence is broad and convincing, many more studies are needed to confirm these preliminary results.
Penetrating Neck Trauma

A. ABCs of ATLS
- airway control
- thoracostomy tube

B. Neck Hemorrhage

C. Evaluate for other life threatening injuries

D. Local wound exploration

E. Platsyma Violated GSW (AP neck film)

F. Zone I, III or multiple

G. Zone II

H. OR Neck Exploration
- Carotid Injury-primary repair
- Venous Injury-ligate
- Esophageal Injury-primary repair and drain
- Tracheal Injury-primary repair (tracheostomy depends on level)

F. Esophagography (if equivocal)
Bronchoscopy (if clinical signs/symptoms exist)
Penetrating Chest Trauma

Marie Crandall

The management of patients with penetrating thoracic injuries is initiated with the ABCs of resuscitation. A key principle in the management of these injuries is that outcomes improve with shorter transport time. Certain interventions may be performed in the field or at nontrauma centers to prevent imminent death, but rapid evacuation to a trauma center should not be delayed.

A. Airway assessment: If there is any question regarding ability to maintain an airway, prompt intubation and positive pressure ventilation should be performed.

B. Assess breathing: Chest wall injuries which leave a tract two-thirds the diameter of the trachea or larger may lead to a “sucking chest wound,” so named because of the sucking sound made when air preferentially enters the pleural space through this defect. This can rapidly lead to a pneumothorax. The unstable patient with a penetrating chest injury should undergo ipsilateral or bilateral needle thoracostomies in the field to alleviate tension pneumothorax. Definitive management of this injury is tube thoracostomy; one does not need to perform a chest x-ray before placing a chest tube for the unstable patient. Tube thoracostomy is also indicated for hemothorax. In 85% of patients, this therapy alone will suffice; once the lung reexpands with evacuation of the pleural space, most bleeding will tamponade and most parenchymal air leaks will seal. However, if drainage from the thoracostomy tube exceeds 1500 cc immediately after insertion or is persistently over 200 cc/h, operative thoracotomy is indicated. Thoracotomy is also necessary for persistent large air leaks from the thoracostomy tube. If, at any time, airway or breathing becomes compromised, prompt intubation and positive pressure ventilation should be performed.

C. Assess circulation: Large-bore peripheral IVs should be inserted for every patient and resuscitative fluids started. Causes of hemodynamic compromise include tension- and hemothorax, external blood loss, and pericardial tamponade. A pericardioscintesis during transport or at a non-trauma center may alleviate pericardial tamponade but should not delay transport to a trauma center. The patient in extremis may benefit from an emergency thoracotomy. Survivors are taken to the operating room for definitive surgical repair of injuries. It should be noted that there is considerable surgical controversy surrounding the amount and type of resuscitative fluids used, as well as the indications for pericardioscintesis and emergency thoracotomy.

D. Secondary survey: The stable patient can be more carefully assessed during a secondary survey. Wound location(s) is noted. All patients suspected of having hemo- or pneumothoraces are evaluated with an arterial blood gas and baseline chest x-ray. If positive for injury, a tube thoracostomy is placed. Because of the small but real possibility of slow-onset pneumothorax, patients with a negative baseline chest x-ray should have a repeat x-ray at 6h. If positive for injury at that time, a tube thoracostomy is placed.

Patients must be carefully monitored for changes in the ABCs during the observation period.

Injuries to specific areas are as follows:

E. Anterior box injuries: The anterior “box” is a space that overlies the heart and extends from the sternal notch to the xiphoid between the nipples. Eighty percent of penetrating chest wounds which injure the heart occur in this space. If a stable patient has an injury in this box, an echocardiogram is done to assess for the presence of pericardial fluid. The echocardiography may be done on its own or as part of the focused abdominal sonography for trauma (FAST exam). If there is fluid, the patient is taken to the operating room for a subxiphoid pericardial window. Blood found in the pericardium indicates a possible cardiac injury and the need for sternotomy and exploration.

F. Posterior box: The posterior “box” is a space that overlies the descending aorta and esophagus. This box is bounded by the scapulae. Crepitus, dysphagia, or change in phonation may indicate an airway or esophageal injury, but symptoms are absent in 10–50% of patients. Stable patients undergo tests to rule out injury to these structures. Angiography is the gold standard to rule out aortic injury, but transesophageal echo has been used successfully at several centers to view the descending aorta. Occasionally, mediastinal air...
indicating esophageal injury is seen on plain chest x-ray; however, this is not very sensitive. Esophageal endoscopy combined with barium swallow has a sensitivity rate of at least 95% for diagnosing esophageal injuries. Aortic and esophageal injuries must be promptly repaired surgically. Rarely, a stable patient with a persistent chest tube air leak, crepitus, or changes in phonation will be found to have an airway injury on bronchoscopy. Management of these injuries is individualized.

G. Thoracoabdominal injuries: Thoracoabdominal injuries are to be suspected with wounds that occur from the nipples to the costal margins anteriorly and the scapular tips to the costal margins posteriorly. The concern with these injuries is that the diaphragm may have been traversed and the abdominal cavity entered. Many trauma surgeons believe that all diaphragm injuries should be repaired because of the risk of herniation of abdominal contents into the chest. To assess this, a diagnostic peritoneal lavage is performed. A red blood cell count over 10,000 indicates abdominal penetration, hence diaphragmatic penetration and injury. These patients are taken to the operating room for exploratory laparotomy and prophylactic ipsilateral thoracostomy tube.
Penetrating Chest Trauma

A. Airway assessment
   - if patient is unstable, intubate and provide positive pressure ventilation

B. Breathing assessment
   - if unstable, intubate and ventilate, needle thoracostomy

C. Circulation
   - IV access

   - patient stabilizes
   - patient is unstable
   - insert chest tube
   - quantitate bleeding
   - emergency thoracotomy

D. Secondary survey
   - arterial blood gas
   - chest X-ray
   - monitor ABC’s

E. Anterior "box"
   - sternal notch to xyphoid
   - nipple to nipple

   - echocardiogram
   - (+) --- (-) observe
   - pericardial window, if positive then sternotomy and repair of injury

F. Posterior "box"
   - scapula to scapula
   - overlies aorta, esophagus

   - endoscopy +/- swallow angiogram
   - (+) --- (-) surgery
   - (-) --- observe

G. Thoracoabdominal
   - nipples to costal margin
   - scapular tips to costal margins

   - DPL
   - (+) --- (-) observe
   - laparotomy
   - chest tube
   - observe
Penetrating Abdominal Trauma

James M. Waltenberger

A. Primary Surgery. As with any trauma, the management of penetrating abdominal trauma begins with the primary survey and the ABCs (Airway, Breathing, and Circulation). After the patient’s airway is controlled, two large-bore peripheral intravenous lines should be placed (18 gauge or larger) and fluid resuscitation begun immediately. If the patient remains unstable after 2 L of crystalloid, blood should be administered. If cross-matched blood is not readily available, type O blood (Rh negative for female patients of childbearing age) or type-specific blood may be safely transfused. A quick assessment of any immediately life-threatening injuries should be undertaken and the patient should be disrobed.

B. The secondary survey involves a thorough assessment of all the patient’s injuries and the patient is given supplemental oxygen. Eviscerated abdominal contents should be covered with sterile, saline moistened gauze, but not manipulated further. Retained implements (bladed weapons) should be left in position, as premature removal may result in loss of vascular tamponade, massive, uncontrollable hemorrhage, and death. Baseline lab studies include a complete blood count, electrolytes, coagulation studies, pregnancy test, urinalysis, toxicology screen, and a blood sample for type and cross-matching. A nasogastric tube (NGT) and Foley catheter should be inserted. A thorough exam is performed of all orifices, identifying all the injuries and classifying them by mechanism (bullet, stab wound, shotgun, etc.) and location. Immediate indications for surgery include shock, transperitoneal path of bullet, evisceration, peritonitis, free air on x-ray, retained stabbing implement, or gross blood per NGT or rectum.

The focused assessment for the sonographic examination of the trauma patient (FAST) is a rapid diagnostic test used during the secondary survey. It systematically surveys the pericardial sac and the dependent regions of the abdomen for blood (Morrison’s pouch in the right upper quadrant, the splenorenal recess in the left upper quadrant, and the pelvis). FAST scan’s role in the assessment of penetrating trauma is evolving; its advantages are that it is a rapid, noninvasive, portable test that is sensitive for hemoperitoneum and hemopericardium. Its limitations are that it has a long learning curve and misses hollow viscus injuries.

All injuries from penetrating trauma should be evaluated with plain films using radiodense markers on the wound sites. This allows the physician to determine potential missile trajectories and possible transperitoneal path. Once a bullet is seen, a lateral film is used to define its location. All bullet wounds should be accounted for, meaning each wound should have a corresponding retained missile or entrance/exit wound associated with it.

In addition to mechanism, wounds are characterized by location. The locations of importance are thoracoabdominal, anterior abdomen, back and flank, and pelvis. The thoracoabdominal region is below the nipples/scapula and above the costal margin. The diaphragm at the end of full expiration may rise as high as the fourth intercostal space anteriorly and the tips of the scapula posteriorly. The anterior abdomen is from the costal margin to the inguinal ligament, anterior to the mid-axillary line. The back and flank region is bordered by the scapulae superiorly and the iliac crest inferiorly, posterior to the mid-axillary line.

C. Stab wounds most commonly occur in the upper anterior quadrants. The probability of an intra-abdominal injury is 40–60% for anterior abdominal wounds, 20–40% for flank and thoracoabdominal wounds, and 10–20% for back wounds. Some institutions advocate local wound exploration (LWE) for anterior abdominal stab wounds, while others immediately proceed to diagnostic peritoneal lavage (DPL). While blunt probing of wounds is unreliable and potentially dangerous, LWE can be carried out safely under aseptic conditions and local anesthesia. This procedure entails surgically extending the wound in order to better visualize the injury tract. An intact posterior fascia constitutes a negative LWE, and management consists of gentle wound irrigation, closure of the extended portions of the wound, and discharge. In cases where clear peritoneal violation or posterior fascial disruption is observed (positive LWE), and in cases where the entire wound tract cannot be adequately visualized (equivocal LWE), the exploration
is considered positive, and further testing to identify possible visceral injury is indicated.

DPL can be used to quickly determine whether a patient with a penetrating abdominal injury has either intra-peritoneal hemorrhage or peritonitis. Before performing DPL, the bladder should be drained and an NGT inserted. Aspiration of gross blood, bile, or bowel contents is an indication for laparotomy. In an adult 1L of 0.9 NS is instilled and drained. Peritonitis is confirmed by WBC in DPL fluid > 500 cells/cc. The DPL is positive for intra-abdominal injury in an anterior abdominal stab wound if the total RBC count is >100,000 cells/cc. For thoracoabdominal wounds, DPL is positive if the RBC count is >10,000 cells/cc, to increase the sensitivity for possible diaphragm injuries. DPL’s advantage over CT is that it is faster, no contrast is needed, has a lower cost, the patient does not have to be moved, and it has a low false-negative rate. CT scans can easily miss small bowel and diaphragm injuries. The disadvantage of DPL is that it is more invasive (1–3% chance of iatrogenic injury) and its high sensitivity may lead to negative laparotomies (5–12% false positive). Contraindications to DPL include pregnancy, obesity, and prior abdominal surgeries.

The role of laparoscopy in trauma is still not clear; however, it may be a useful tool for abdominal stab wounds. Visualization of the adjacent peritoneum while probing the wound may confirm or refute peritoneal penetration. It is sensitive for identifying small liver, splenic, or diaphragmatic injuries. Small splenic and liver injuries can often be managed without laparotomy and an uncomplicated diaphragm injury may be repaired laparoscopically.

D. Gunshot wounds. The treatment of gunshot wounds (GSWs) is different than that of stab wounds. If penetration of the peritoneal cavity can be demonstrated (based on radiographs, physical exam, or bullet trajectory), then operative intervention is indicated, even if the patient is stable. GSW injuries have reported an 80–85% incidence of peritoneal penetration, 95% of which had some degree of visceral injury. The liver is the most commonly injured organ (37%), followed by small bowel (26%), stomach (19%), and colon (17%).

Patients with thoracoabdominal GSWs who do not have immediate operative indications should undergo a DPL. They should also be worked up for pneumothorax. Triple contrast CT is the first test of choice in patients with back and flank wounds. This allows one to evaluate the retroperitoneum and hopefully identify the tract of the bullet. If the tract cannot be clearly identified and there is no indication for laparotomy, a DPL should be performed. If a GSW is felt to be tangential, intra-abdominal injury should be ruled out using DPL. Penetrating pelvic wounds may result in injury requiring laparotomy. The outlet tracts must all be evaluated depending on possible trajectory. If the bladder is at risk, a cystogram should be performed. Proctoscopy is performed to rule out rectal injury. Females should have a vaginal speculum exam. DPL is performed for transpelvic GSWs with the threshold for laparotomy being >10,000 RBCs.

Once the decision is made to take the patient to the operating room, IV antibiotics and tetanus toxoid should be given. Control of hemorrhage is the first priority. The abdomen should be rapidly inspected in a systematic manner, taking care to survey and pack all quadrants; the mesentery, omentum, diaphragm, and retroperitoneum should be inspected. Second priority is controlling contamination. Gross contamination from hollow viscus injuries can be temporarily controlled with bowel clamps or a quick suture. Intestinal repair should be undertaken after hemorrhage has been controlled. Injury to the retroperitoneum is suspected when there is bile staining, a retroperitoneal hematoma, or crepitation in the tissues. In these situations, structures which may sustain retroperitoneal injury (right colon, left colon, splenic and hepatic flexures, and the duodenal sweep) should be mobilized for better identification and repair of injury. Once the necessary repairs have been completed, the entire abdomen should be copiously irrigated with warm saline.
Penetrating Abdominal Trauma

A. Primary Survey

B. Secondary Survey, Resuscitation labs

C. Stab Wounds
   - Absolute OR Indication *
     - (+) Back + Flank
     - (-) Local Wound Exploration
   - Laparotomy

D. Gunshot wound
   - Anterior Abdominal
     - (-) No Penetration of Fascia
     - (+) Penetration of Fascia
       - (+) DPL or Laparoscopy
       - (-) >100K RBC
   - Local Wound Exploration
   - Serial Exams

* Gross oral or rectal blood
  Shock
  Transperitoneal path of bullet
  Evisceration
  Peritonitis
  Free air
  Retained Implement
13
Blunt Abdominal Trauma

Marie Crandall

The abdomen is the third most commonly injured body region after the head and extremities, and blunt trauma is the most common mechanism of injury. Injuries can occur with direct blows, by shear forces, with rupture of a hollow viscus from increased intra-abdominal pressure, or from crushing between the abdominal wall and the vertebral column.

A. ABCs: The approach to all trauma patients begins with the ABCs of resuscitation. “A” is for airway assessment, “B” is for ensuring that the patient is breathing and there is bilateral exchange of air, and “C” is for circulation assessment and taking of vital signs. Once this is underway, the evaluation of a patient with blunt abdominal trauma begins.

B. Secondary survey: A secondary survey is performed, examining the patient from head to toe. Plain radiographs and basic blood work are obtained as indicated by the patient’s injuries and may include cervical spine films and x-rays of the pelvis. The focused abdominal sonogram for trauma (FAST exam) is gaining popularity around the country. Many studies have validated its use as a screening tool for hemoperitoneum and fluid within the pericardial sac. Trained trauma staff can rapidly perform this specific ultrasound exam. When the FAST exam is routinely used, it can reduce the use of CT scanning and diagnostic peritoneal lavage (DPL), reduce cost, and reduce morbidity associated with DPL. FAST sequentially checks for blood in the pericardial sac, right upper quadrant, left upper quadrant, and the pelvis. A full urinary bladder is required. For cardiac imaging, the transducer is positioned in the subxiphoid region. To inspect for blood between the liver and right kidney, the probe is placed in the right midaxillary line between the 11th and 12th ribs. On the left side, the probe is placed between the 10th and 11th ribs in the left posterior axillary line. For imaging the pelvis, the probe is positioned transversely 4 cm above the bladder.

C. Stable patients with blunt trauma who are conscious, not intoxicated, and have no distracting injuries (head, extremity) may be expected to provide a reliable abdominal exam. If these patients do not have abdominal pain or tenderness to palpation, observation is sufficient. Patients with blunt abdominal trauma who require surgery for other injuries during the period of observation, and patients whose exam changes (i.e., develop pain, lose consciousness) will need either a CT scan or DPL to rule out visceral injury. CT is generally preferable for patients who are stable and can be transported away from the resuscitation area. The CT or DPL findings suggestive of visceral injury are listed in Table 13.1.

D. The hemodynamically stable patient with an unreliable exam, due to intoxication or head injury, should undergo CT scan. DPL can be used as well.

E. Unstable patients and patients being hurried to the operating room for other injuries (e.g., head, orthopedic, and vascular injuries) need rapid assessment for potentially life-threatening abdominal trauma. Transport to the radiology suite may not be safe, hence the need to use an alternative to CT scanning to assess for intra-abdominal injuries. These patients should undergo DPL, as it can be performed quickly and does not require transport of the patient away from the trauma resuscitation suite. If the FAST exam is available, it can be used to determine whether or not hemoperitoneum, cardiac tamponade, etc. are present.

F. Very rarely, a patient will present after blunt abdominal trauma with signs of an obviously injured abdomen (e.g., abdominal wall avulsion, peritonitis, and expanding abdomen). These patients should not undergo diagnostic testing, as they require immediate laparotomy. All other patients with a work up indicating high likelihood of life-threatening visceral injury should undergo exploratory laparotomy.

G. Spleen: The spleen is the most commonly injured organ in blunt abdominal trauma. Seventy percent of injuries to the spleen may be managed nonoperatively with bed rest and 48 h of careful monitoring and serial hemoglobin checks. Patients who become unstable during the period of observation or have evidence of ongoing hemorrhage should undergo abdominal exploration. Splenic salvage (splenorrhaphy with hemostatic agents, splenic wrapping, or hemostatic sutures) and partial splenectomy may be successful in a subset of stable patients.
Other patients should undergo splenectomy. Antipneumococcal and antihemococcal vaccines are given postoperatively to prevent postsplenectomy sepsis. A scoring system for splenic injuries is shown in Table 13.2.

**H. Liver:** The liver is the second most commonly injured organ in blunt abdominal trauma. Stable patients may be managed nonoperatively. This mode of therapy is being used more frequently; over 50% of patients with blunt hepatic injuries can be managed without surgery, with success rates of up to 96%. When surgery is indicated, hemostasis is the key; this may be achieved with hemostatic agents, ligation of bleeding vessels, or cautery. A Pringle maneuver, compression of the hepatic artery within the hepatoduodenal ligament, is often helpful to decrease blood loss while injuries are identified and repaired. This can be performed for 20–30 min without causing long-term hepatic dysfunction. Packing and planned reoperation may be the best where hypothermia and coagulopathy promote continued hemorrhage. Reoperation is then undertaken after satisfactory patient warming and correction of coagulation deficits has occurred. A scoring system for liver injuries is shown in Table 13.3. Nonoperative management can be theoretically used for any degree of injury; however, failure rates increase with the more severely injured patients.

**I. Pancreas:** Hemorrhage from the pancreas is treated with hemostasis and drainage. Ductal injuries are drained externally in the unstable patient to control the pancreatic fistula. Stable patients with pancreatic transactions lateral to the superior mesenteric vessels require distal pancreatectomy. More proximal ductal injuries require pancreaticojejunostomy for definitive management; however, these patients are almost always unstable and external drainage is often the best choice, with a delayed definitive procedure to repair the pancreatic fistula. Pancreatic injuries without major duct disruption are treated with external drainage alone. An intraoperative pancreatogram may help define ductal anatomy and guide management in the stable patient.

**J. Intestinal injuries:** Small and large intestinal injuries are managed by debridement of nonviable tissue and by either primary repair or fecal diversion (stoma). Treatment choice is determine by extent of injury, presence of associated injuries, degree of intra-abdominal contamination, and patient condition. For the stable patient with minimal contamination and who does not have other serious intra-abdominal injuries, primary repair is safe; this is true even for left colon injuries. Resection and anastomosis is indicated if over 70% of the bowel wall circumference is disrupted.

Since the duodenum cannot be brought to the skin to create a stoma, diversion is achieved by exclusion or diverticulization. In duodenal exclusion, the pylorus is stapled closed and a gastrojejunostomy is performed. Diverticulization includes antrectomy, gastrojejunostomy, and tube duodenostomy. Occasionally, internal and external tube drainage alone will be used to support a duodenal repair.
Blunt Abdominal Trauma

A. ABC's

B. Secondary survey
   - X-rays
   - FAST exam

C. Stable patient
   - no distracting injuries
   - reliable exam
   -> CT for pain

D. Stable patient
   - unreliable exam
   -> CT or DPL

E. Unstable patient
   or
   - Needs other procedure
   -> DPL
   - FAST exam

F. Obvious intra-abdominal injury
   -> Go to surgery

G. Spleen
   - observe
   - splenorrhaphy
   - splenectomy

H. Liver
   - observe
   - repair, pringle maneuver
   - packing, warming, re-operation

I. Pancreas
   - if bleeding-hemostasis and drainage
   - duct injury, unstable patient-drain
   - distal duct injury-resection
   - proximal duct injury-drain then reoperate for internal drainage

J. Intestine
   - type of repair depends on location, degree of soilage, associated injuries, patient condition
Pelvic Fractures

Benjamin P. Crane

A. Introduction. Pelvic fractures usually result from high-energy trauma and have a mortality rate of ∼10%. The high mortality rate is a result of hemorrhage from large bleeding bony surfaces or disruption of the pelvic venous plexus. Arterial involvement, however, is seen in only 10% of all pelvic fractures. In addition, because of the high-energy trauma, pelvic fractures are frequently associated with intrapelvic and abdominal visceral trauma. Thus the workup and management of all pelvic fractures must be carried out in unison with the efforts of the entire trauma team.

The pelvic ring is made up of the sacrum and two innominate bones consisting of the ilium, ischium, and pubis. The innominate bones are joined anteriorly at the pubic symphysis and posteriorly to the sacrum at the sacroiliac joint. Transverse ligaments stabilize the symphysis pubis while the sacroiliac joint is stabilized by anterior and posterior ligaments. Disruption of a single element of the ring does not render the pelvis unstable. However, if several elements are injured the pelvis may become unstable.

The immediate goals of pelvic fracture management are hemodynamic stability, prevention of septic sequelae, and stabilization of the fracture to allow early patient mobility. Pelvic fractures can be classified into three main groups, stable, partially stable, and unstable, based on fracture pattern and associated soft tissue injury.

B. Stable pelvic fractures, while painful, by definition the pelvic ring is not disrupted. They are usually avulsion fractures of the ischium or iliac spines, iliac wing fractures, and non-displaced or minimally displaced fractures of the pubic rami. Surgery in not indicated and the patient may bear weight on the lower extremities.

C. Partially stable pelvic fractures by definition are rotationally unstable but vertically stable. These can be divided into two main groups based on the mechanism of injury: anterior-posterior compression injuries and lateral compression injuries.

D. Anterior-posterior compressive forces cause external rotation of one or both hemipelvcs. This force causes diastasis injury of the symphysis pubis and a sacroiliac ligament disruption or fracture. If the pubic symphysis separation is < 2.5 cm, then the sacrospinous ligament is intact and pelvic instability is minor. These “open book” injuries are treated nonoperatively. When the pubic symphysis diastasis is > 2.5 cm, the sacrospinous ligament is disrupted causing the hemipelvis to be rotationally unstable. The separation of the symphysis pubis can cause a marked increase in volume of the pelvis. The increased pelvic volume contributes to the patient’s hemodynamic instability. Every effort must be made to tamponade the bleeding while at the same time provide adequate resuscitation. In the emergency department closed reduction can be performed manually until a pelvic clamp can be applied, or the patient can be taken to the operating room for placement of an external fixator. If the patient remains hemodynamically stable, then the injury can be treated with a pelvic sling, an external fixator, or symphyseal plating.

E. Lateral compression injuries are caused by falls from heights onto the pelvis or side impact motor vehicle crashes. A posteriorly-directed lateral compression force can cause a cancellous impaction fracture of the sacrum. An anteriorly-directed lateral compression force will cause a pubic rami fracture or an overlap and fracture of the anterior portion of the sacral ala. The latter compression fracture rarely causes the marked increase in pelvic volume typically seen in anterior-posterior pelvic fractures. Thus, the lateral compression fracture patients are less likely to be hemodynamically unstable. These patients do, however, suffer from leg length discrepancies, and internal rotation of the affected side. If the leg length discrepancy is < 2 cm and there is < 30° of internal rotation of the affected side, the injury in managed conservatively. The patient can be ambulatory with protected weight bearing. If the leg length discrepancy is > 2 cm or there is > 30° of internal rotation of the affected side, the patient can be managed in two ways. Either the patient can undergo closed reduction followed by external fixation or open reduction and symphyseal plating. Women must be counseled that malunion of lateral compression fractures may cause dyspareunia and/or difficulty with vaginal childbirth.
F. Unstable pelvic fractures by definition are rotationally and vertically unstable. These fractures are usually caused by vertical shear forces resulting from a fall from a height. The hemipelvis may appear to be shifted vertically. The shearing force transmitted through the pelvis results in an anterior and a posterior break of the ring. Anteriorly there is commonly a fracture of the pubic rami or disruption of the pubic symphysis. Posteriorly there is a vertical fracture of the sacrum or complete disruption of the anterior and posterior sacroiliac ligaments. Anterior and posterior fixation is required to achieve pelvic stability.

G. In the hemodynamically unstable patient definitive treatment is deferred until the patient is stable. If the patient does not need a laparotomy, temporary stability can be maintained with an external fixator. If, however, the patient requires a laparotomy and the abdomen is not contaminated, provisional stability is achieved by anterior symphyseal plating.

In the hemodynamically stable patient a distal femoral traction pin may be placed for skeletal traction. If the residual vertical displacement is >1 cm after skeletal traction, anterior and posterior fixation is recommended.
Pelvic Fractures

A. Introduction
   History
   Physical Exam
   X-Ray

B. Stable Fracture
   • Avulsion Fracture
   • Iliac wing fracture
   • Non-or minimally displaced fracture of pelvic ring

C. Partially Stable Fracture

D. AP Compression Injury
   Symphysis <2.5 cm
   Symphysis >2.5 cm

E. Lateral Compression Injury
   <2 cm LLD & <30° Internal rotation
   >2 cm LLD & >30° Internal rotation

F. Unstable Fracture
   Hemodynamically Stable
   Skeletal Traction
   Late anterior and posterior fixation
   Hemodynamically Unstable

G. Hemodynamically Unstable
   Pelvis Unstable
   Emergency Pelvis External Fixator
   (-) Peritoneal Lavage
   (+) Laparotomy
   Pelvis Stable
   Peritoneal Lavage

H. Hemodynamically Stable
   Angiography and Embolization
   Continued Assessment
   Hemodynamically Unstable
   Non-Operative Management

X-Ray Pelvis

• Avulsion Fracture
• Iliac wing fracture
• Non-or minimally displaced fracture of pelvic ring

• Pelvic Sling
• External Fixation
• Symphyseal plating

Non-operative Management

• Closed Reduction
• External fixation or open reduction
• Symphyseal plating

Late anterior and posterior fixation
A. Introduction. Patients sustaining blunt or penetrating trauma may have associated genitourinary injuries that can present as gross, microscopic, or no hematuria. Gross hematuria is defined as readily visible and microscopic hematuria as >5 RBC/hpf. Rapid and accurate evaluation and diagnosis may prevent serious complications. The differential diagnosis includes renal, ureteral, bladder, and urethral injury. In general, genitourinary injuries are the least life threatening, but are most likely to affect long-term quality of life with respect to their complications.

B. Clinical Assessment, Physical Examination, and Urinalysis. The degree of hematuria does not necessarily correlate with the severity of the genitourinary injury. Accordingly, traumatic hematuria must be fully evaluated and careful clinical assessment may provide clues to the extent of injury. Hemodynamic instability may indicate a significant injury. The mechanism of injury (blunt or penetrating) is important for localizing and predicting potential injuries.

Physical examination is focused on the abdomen and external genitalia. Blood at the meatus, a high-riding prostate on digital rectal examination, and hematoma of the penis, scrotum, or perineum are signs of a urethral injury. In this case, catheterization should not be performed until urethral integrity is ascertained. Fractured ribs, flank tenderness, and flank ecchymosis (Grey-Turner sign) are associated with renal injuries. Inability to void, lower abdominal pain, and pelvic fractures are associated with bladder injuries. In stable, conscious patients, urine is collected by clean-catch technique. In more seriously injured patients, urethral catheterization should be performed if there is no blood at the urethral meatus in order to monitor the patient’s urine output.

The collected urine sample can be quickly tested for blood using a dipstick urinalysis. Positive dipstick samples should undergo further evaluation with microscopic examination for accurate assessment of the hematuria. Significant hematuria is defined as >5 RBC/hpf. Grossly red urine samples should also undergo microscopic examination because certain drugs and food colorings can cause red-colored urine, for example, phenazopyridine, rifampin, metronidazole, and beets.

Visible blood at the urethral meatus or with voiding may indicate a significant genitourinary injury. It is important to emphasize that the severity of injury does not necessarily correlate with the degree of hematuria. Occasionally, patients with a renal pedicle injury will have a normal urinalysis, but in general, the presence of gross hematuria indicates a greater likelihood of a major urological injury. Stable patients with blood visible at the urethral meatus should undergo a retrograde urethrogram (see Sect. E) to assess for urethral injury. If a urethral injury is confirmed, a catheter should not be placed and urological consultation should be obtained. Bladder integrity should be assessed with a cystogram (see Sect. E). All patients with gross hematuria (with or without urethral injury) require urgent renal imaging, with computed tomography (CT) being the preferred modality. Unstable patients who require immediate operative intervention should undergo renal imaging with an intraoperative one-shot intravenous pyelogram (IVP) (see Sect. E). In addition, if the unstable patient has blood at the urethral meatus, a suprapubic catheter should be placed intraoperatively.

Microscopic hematuria in patients without shock is highly unlikely to be associated with significant genitourinary injury. The majority of these cases occur with blunt trauma and usually reflect a minor urological injury, most commonly a renal contusion. Accordingly, these patients can be observed closely without further initial evaluation. An important exception to this rule is patients who sustain rapid deceleration injuries. This mechanism of injury is associated with renal pedicle avulsion in adults and ureteropelvic junction disruption in children. These serious injuries can present with microscopic hematuria or normal urinalysis. Therefore, these patients require radiological assessment with CT. In addition, microscopic hematuria in patients with shock should undergo radiological evaluation with CT. Unstable patients who require immediate operative intervention should undergo an intraoperative IVP.

As outlined above, the clinical assessment, physical examination, and urinalysis results will direct the patient’s subsequent urological assessment. This work-up may include
imaging of the kidneys, ureters, bladder, and urethra. As previously discussed, patients with blood at the urethral meatus may have a urethral injury, and urethral integrity is assessed with a retrograde urethrogram. This is performed by first placing a 12–14 French Foley catheter 1–2 cm into the urethra. The catheter’s balloon is filled with 2–3 ml sterile water and 10–12 ml contrast material is injected into the urethra while taking plain radiographs or using portable fluoroscopy to assess urethral integrity. In addition, patients with blood at the urethral meatus, and especially patients with pelvic fractures, may have associated bladder injuries. Bladder integrity is assessed with a cystogram, which can be performed immediately following the retrograde urethrogram if urethral injury is excluded and the Foley catheter can be safely passed into the bladder. A cystogram should be performed under gravity drainage of ~300 ml contrast material into the bladder. Obtaining post-drainage bladder films is most important. Alternatively, a CT cystogram should be performed at the time of abdominal/pelvic CT.

Suspected renal injuries may be assessed with CT, IVP, ultrasonography, and angiography. Of these, CT is the study of choice in stable patients. In unstable patients who require immediate operative intervention, a one-shot IVP should be performed intraoperatively. This is accomplished with a bolus intravenous injection of 2 ml/kg of contrast agent followed by a plain radiograph taken after 10–15 min. This examination allows for an assessment of potential renal injury and for a confirmation of the existence of two kidneys should renal exploration, repair, and/or nephrectomy be contemplated.

C. Urethral Injury. Urethral injuries are classified as anterior or posterior depending on the affected urethral segment. Most urethral injuries are caused by blunt trauma. Both types typically present with gross blood at the urethral meatus. Anterior urethral injuries (bulbar urethra) classically occur in patients who sustain a straddle injury to the perineum. These patients usually present with a large perineal, scrotal, and penile hematoma. Treatment of this type of urethral injury usually involves urinary diversion with a suprapubic catheter with a delayed surgical repair of the urethra if necessary. Posterior urethral injuries (membranous or prostatic urethra) are classically associated with pelvic fractures. Specifically, 5–10% of pelvic fractures are accompanied by a posterior urethral injury, and 90% of posterior urethral injuries occur with a pelvic fracture. In addition, 10–20% of posterior urethral injuries have an associated bladder injury. Treatment options for these injuries include immediate suprapubic catheter drainage followed by delayed repair if a stricture develops, immediate primary endoscopic alignment, or immediate open operative repair. Of these, immediate suprapubic catheter drainage with delayed urethroplasty (if necessary) is most common. Immediate open operative repair may be hazardous and complicated, especially with large pelvic fractures and hematomas.

D. Renal Injuries. Blunt trauma accounts for 80% of all traumatic renal injuries, the majority of which are simple renal contusions. Renal lacerations and renal vascular injuries comprise 10–15% of all blunt renal injuries. The majority of these blunt injuries are managed nonoperatively, including major lacerations and those involving the collecting system. In addition, urinary extravasation is not an absolute indication for operative intervention. Absolute indications for surgical management include uncontrollable renal bleeding with hemodynamic instability, main renal vessel avulsion, bilateral main renal artery injury, an injured solitary kidney, and an expanding or pulsatile retroperitoneal hematoma. Recent reports have even advocated expectant management for a shattered kidney if the patient is hemodynamically stable. Penetrating renal injuries usually require immediate operative management, and most of these injuries can be successfully reconstructed. Recent reports, however, have advocated conservative management of penetrating renal injuries.

E. Bladder Injury. Bladder injuries are divided into two categories, extraperitoneal and intraperitoneal bladder ruptures. Eighty-five percent of extraperitoneal bladder injuries are associated with pelvic fractures and occur almost twice as often as intraperitoneal ruptures. Additionally, 10% of pelvic fractures are accompanied by bladder rupture. Extraperitoneal ruptures are diagnosed with a cystogram that reveals extravasation of contrast material limited to the pelvis. They are managed with long-term (10–14 days) Foley catheter drainage. Intraperitoneal ruptures occur at the dome of the bladder and usually occur in patients with a full bladder. The dome perforation allows intraperitoneal extravasation of urine, which appears as layering of contrast material between loops of bowel on cystogram. These injuries require immediate operative repair.

F. Ureteral Injuries. The vast majority of ureteral injuries are associated with other abdominal injuries and are usually caused by penetrating trauma. Isolated ureteral injuries are extremely uncommon. Intraoperative assessment of ureteral integrity is performed by occlusion of the ureter with simultaneous intravenous injection of indigo carmine or methylene blue. If extravasation is noted or ureteral injury is otherwise discovered, immediate repair is of critical importance. unrecognized ureteral injuries can present with serious complications, that is, urinoma formation and sepsis. Surgical repair of ureteral injuries usually involves debridement of devitalized tissue, performing a watertight anastomosis, and ureteral stenting and drainage. Upper ureteral injuries can usually be repaired with primary reanastomosis, while lower ureteral injuries (below pelvic vessels) usually require reimplantation into the bladder. If ureteral injuries are discovered in a delayed fashion, that is, more than 5 days, patients are managed with temporary urinary diversion with a nephrostomy tube and Foley catheter.
Traumatic Hematuria

A. Differential Diagnosis
1. Renal
2. Ureteral
3. Bladder
4. Urethral

B. Clinical Assessment
1. Physical Exam
2. Gross Hematuria
3. Microscopic Hematuria

Stable Blood at Meatus

Stable No Blood at Meatus

Retrograde Urethrogram

C. Urethral Injury

CAT Scan Abd/Pelvis

Observation

See renal, bladder or uerteral injury

D. Renal Injury

E. Bladder Injury

F. Ureteral Injury

Intraop 1 shot IVP

Suprapubic catheter or Foley by GU

Anterior
Suprapubic catheter possible delayed repair

Posterior
Suprapubic catheter with possible delayed repair vs. endoscopic catheter placement vs. immediate repair

Extraperitoneal
Long term bladder drainage

Intraperitoneal
Immediate laparotomy and repair

Surgery Indications
Uncontrollable bleeding
Expanding hematoma
Major vessel avulsion
Bilateral artery injury

Observe
Most injuries

Immediate Recognition
Prompt Repair
Reanastomosis

Delayed Recognition
Urinary Diversion
Future surgical repair

Intraop

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A. Introduction. Knee pain is a common orthopedic problem that can present in three different ways: (1) High-energy traumatic injuries (motor vehicle injuries or falls), which are the most serious in nature and warrant immediate treatment to ensure limb preservation, (2) Low-energy traumatic injuries (sports or occupational strains), which result in ligament or meniscal injuries that may require surgery in the ensuing weeks or months, and (3) the gradual onset of pain due to overuse or arthritic conditions. Accurate diagnosis is imperative to avoid misdiagnosis of limb or life-threatening conditions including septic arthritis, venous thrombosis, or popliteal aneurysm.

B. History. High-energy injuries or the inability to bear any weight on the affected limb should raise suspicion of a potential fracture. Sports or occupational noncontact injuries involving a twisting or cutting maneuver may cause ligament or meniscal injuries. An audible “pop” at the time of injury is commonly found in tears of the anterior cruciate ligament (ACL), patella tendon, and quadriceps tendon. The presence of swelling is also helpful; immediate swelling is typical of fractures, patella dislocations, and multiple ligamentous injuries. Swelling within 2–3 h is typical of isolated ligament injuries (ACL or medial collateral ligament) and within 12–24 h occurs with most meniscal injuries.

Patients with gradual knee pain may describe symptoms only with certain activities such as climbing stairs or prolonged sitting. Swelling that occurs with low levels of activity but resolve with rest are common with mild to moderate arthritic conditions. Swelling associated with warmth, increasing pain unrelieved by rest, and systemic symptoms including fever are found in patients with inflammatory arthropathies including septic arthritis. Patients with knee pain associated with leg swelling are at risk for venous thrombosis or a ruptured Baker’s cyst. Posterior knee and leg pain with diminished pulses are typical of arterial insufficiency or popliteal aneurysms.

C. Physical Examination. Physical examination of the knee includes visual assessment, inspecting tenderness, determining active and passive range of motion (AROM, PROM), and evaluating ligamentous stability. The hips and spine should be examined to rule out referred pain as a cause of knee symptoms. Obvious major limb deformity is typical of fracture or major dislocation, and examination should include accurate documentation of the neurovascular status of the limb, sterile dressing of open wounds, and splinting of the limb for immediate x-ray evaluation. The examiner should differentiate the presence of local swelling (associated with contusion or medial collateral ligament sprain) versus the presence of a knee joint effusion seen with intra-articular injuries (ACL tears or meniscal injuries). Point tenderness can also localize the injury to a specific site such as a hamstring muscle insertion, patella tendon, or medial collateral ligament. Loss of PROM is suggestive of an intra-articular mechanical block including a meniscus tear or a loose body. Loss of AROM including the inability to perform a straight leg raise or knee extension is seen with patella tendon or quadriceps tendon ruptures. Specific maneuvers will allow examination of individual ligaments to assess knee stability. The Lachman test will assess the ACL while the posterior drawer test is specific for posterior cruciate ligament injuries. Valgus and varus stress at 0° and 30° will assess the function of the medial and lateral collateral ligaments, respectively. Meniscal tears will typically display tenderness along the joint line and the pain will be elicited with meniscal compression signs including the McMurray’s and Apley’s test.

D. Diagnostic Tests. Knee radiographs are useful and are necessary whenever fracture or major injury is suspected. Anterior-posterior (A-P) and lateral views are the minimum required views in cases of acute injury. In cases of patella injury, the Merchant view will assess patella positioning and standing or weight-bearing views are used in patients with suspected arthritis. Computed tomography (CT) is necessary in fractures involving the weight-bearing surfaces of the tibia or femur. Magnetic resonance (MR) imaging is the exam of choice to diagnose soft tissue injuries and is commonly used to detect ligamentous injuries, neoplasms, avascular necrosis, and the presence of unusual cysts.
E. Treatment. Immediate treatment is required for limb/life-threatening conditions such as vascular occlusion/disruption, compartment syndrome, septic arthritis, and open fractures. Suspected popliteal aneurysms or patients with knee dislocations should undergo immediate arteriograms because of the high incidence of vascular injury. If limb ischemia is detected, revascularization within 6 h is necessary to avoid permanent sequela. If compartment syndrome is suspected, immediate compartment pressure measurements are indicated and fasciotomies performed within 6 h to avoid permanent damage. Septic arthritis is confirmed with knee joint aspiration (WBC count >50,000 and/or Gram stain + for bacteria) and is treated with immediate surgical/arthroscopic drainage. Patients with acute flare-ups of inflammatory arthritis due to gout or rheumatoid-like conditions are managed with a combination of oral medication, joint aspirations, and rest. Open fractures warrant surgical debridement within 6 h to diminish the risk of osteomyelitis. Most fractures about the knee and all fractures involving displacement of the articular surfaces warrant surgical stabilization. Complete tears of the quadriceps or patella tendon require immediate surgical repair to regain function.

Most contusion or direct blow injuries are treated with icing, short-term immobilization, and gradual return to function with light range of motion exercises. Symptoms related to osteoarthritis are treated with oral medications, exercise, weight loss, and occasional steroid injections. Patients with end-stage arthritis and bone-on-bone radiographic changes are treated surgically with total knee replacement. Low-energy meniscal and ligament injuries may be treated surgically or nonoperatively based on the demands of the patient. The majority of these patients are treated with outpatient arthroscopic procedures. Even in cases when surgery is chosen, it is often delayed days or weeks to allow the inflammatory response to subside. The majority of overuse conditions including patella tendonitis, iliotibial band syndrome, or chondromalacia are treated nonoperatively with combinations of exercise, activity modification, and oral medications.
Knee Pain

A. Introduction
- high energy trauma: treat promptly
- low energy trauma: gradual onset

B. History
- mechanism of injury
- swelling
- fever

C. Physical Examination
- visual assessment
- range of motion
- ligament stability
- neurovascular exam

D. Diagnostic Tests
- radiographs

E. Treatment

Acute, high energy
- reduce fractures, surgery if unstable or displaced
  - angiography for dislocations
  - fasciotomy for compartment syndrome
  - debridement if open fracture
Low energy
- Intra-articular (menisci, cruciates)
- Extra-articular (collateral ligaments, quadriceps, patellar tendon)
  - surgery if patient has high activity level, otherwise conservative (NSAIDS, physical therapy)
Gradual onset
- Osteoarthritis
- Rheumatoid arthritis
- Bursitis
- Anterior knee pain
- Internal derangement
Full mobility of the shoulder is critical to position the hand and upper extremity for the activities of daily living, work, and recreation. Pain about the shoulder may occur with acute trauma (fracture, joint subluxation/dislocation, or rotator cuff tear) or may develop gradually because of overuse (tendonitis, impingement), arthritic conditions, or idiopathic conditions (adhesive capsulitis).

A. History and Physical Exam. The mechanisms of injury can be helpful in determining the type and extent of injury in traumatic events. Falls directly onto the shoulder will typically lead to acromio-clavicular joint sprains (A-C joint separations). Lifting injuries typically will cause a strain or tear of the rotator cuff while falls on the outstretched hand will lead to rotator cuff injuries or fractures of the proximal humerus. Young patients are at risk for an anterior shoulder dislocation if the injury occurs with arm in an abducted externally rotated position. Posterior shoulder dislocations are rare (<5% of shoulder dislocations) but occur commonly in epileptic seizures, electric shock injuries, and alcohol-related falls. Axillary nerve injuries about the shoulder are rare but may occur with fractures or joint dislocations.

The patient’s age is often helpful in determining the cause of gradual shoulder pain. Younger patients (<35 years) may develop shoulder pain due to instability, as rotator cuff tears are unusual in this age group. Instability occurs as a result of repetitive overhead throwing and racquet sports but more typically occurs because of a specific injury. Patients complain of pain over the anterior aspect of the shoulder and have tenderness about the anterior glenohumoral joint and coracoid region. Glenohumoral dislocation occurs with a traumatic fall on the abducted externally rotated arm. The patients are in severe pain and hold the arm in an abducted position until reduction can be performed.

Instability is unusual in patients 40–60 years old but rotator cuff impingement is common. Anterolateral shoulder pain, pain with overhead activities, and night pain are common complaints in this age group. Patients with rotator cuff impingement but with normal strength will respond to conservative treatment but those with significant weakness should be considered to have a full thickness rotator cuff tear. Women over the age of 50 or diabetic patients with an insidious onset of global shoulder stiffness typically develop adhesive capsulitis (frozen shoulder). The acute onset of pain without injury or overuse should caution the examiner for the potential risk of infection or tumor about the shoulder.

Physical examination of the shoulder can aid in diagnosis. Critical elements of the exam include visual evidence of deformity, location of tenderness, active and passive joint range of motion (AROM and PROM), shoulder strength, and special tests for joint stability.

Visual inspection can reveal deformities including prominence of the distal clavicle (A-C joint separations), loss of the deltoid contour (anterior shoulder dislocation), or prominence of the coracoid (posterior shoulder dislocation). Atrophy of the deltoid and upper arm are seen with brachial plexus injuries while localized atrophy of the supra infraspinatus muscles are typical of suprascapular nerve injuries or chronic rotator cuff tears. Distal migration of the biceps muscle belly is a typical finding in patients with long head biceps muscle tear. Acute onset of swelling and development of ecchymosis or bruising about the shoulder are common with proximal humerus fractures and acute rotator cuff tears.

Pain to palpation in specific areas about the shoulder can localize the area of concern. Tenderness may be local or diffuse in nature. Local tenderness over the acromio-clavicular joint is specific to A-C joint pathology while tenderness over the lateral acromion is typical with impingement/rotator cuff conditions.

Range of motion and strength testing are the most critical areas in examining the shoulder. Patients with loss of AROM and PROM will have an intra-articular pathology (i.e., fracture, frozen shoulder, arthritis, infection). Patients with loss of AROM but normal PROM will have extra-articular conditions including rotator cuff injuries. Manual muscle testing is necessary to determine if specific muscle weakness is present. Weakness of external rotation of the shoulder with arm at the side is specific to the infraspinatus muscle. Patients with normal AROM and PROM and no localizing tenderness may
be considered to have referred pain from cervical spine conditions.

Special tests about the shoulder can also be helpful in localizing the source of shoulder pain. The apprehension sign and the relocation test are both sensitive and specific maneuvers to identify shoulder instability. The Neer and Hawkins’s signs are specific to impingement and rotator cuff conditions. A-C joint conditions can be localized with the cross-arm adduction maneuver. Spurling’s test (cervical spine conditions) and the Wright’s and Adson’s test are helpful in the diagnosis of the rare thoracic outlet conditions.

B. Imaging Studies. The history and physical examination should narrow the differential diagnosis, which can be confirmed or clarified by imaging studies. Plain radiographs are taken at the initial assessment and the “trauma series” (true A-P, axillary lateral, and scapular Y views) are the preferred techniques. An additional Zanca view (20° cephalad tilt AP with 50% kW) is used in patients with suspect A-C joint injuries. In cases of complex fractures about the shoulder, computed tomography is useful in treatment planning. In patients without fractures, magnetic resonance imaging (MRI) is the secondary diagnostic test of choice and allows accurate assessment of rotator cuff injuries, labral injuries, ganglion cysts, avascular necrosis, and the presence of soft tissue neoplasm.

C. Exclusionary Diagnoses. Several exclusionary diagnoses must be identified before proceeding with conservative treatment in most shoulder conditions. A warm, red, tender shoulder is indicative of infection (septic arthritis) and warrants aspiration. Neoplasms about the shoulder present with pain and are diagnosed with radiographs. Neoplasms are classified as benign (bone cysts, enchondromas, fibrous dysplasia) or malignant (metastatic or primary bone tumors). Fractures are associated with traumatic injuries and are classified as intra-articular or extra-articular and as stable or unstable. Most fractures are extra-articular, stable, and minimally displaced. These fractures are successfully treated with closed reduction and immobilization followed by ROM exercises after union is attained. Intra-articular fractures or those with significant displacement often require surgical stabilization. High-energy fractures associated with polytrauma injuries have a high risk of neurovascular injury warranting immediate treatment to ensure limb viability. Shoulder dislocations (95% are anterior) warrant immediate reduction in the emergency room setting and generally require intravenous (IV) sedation. Approximately one-third of shoulder dislocation patients will have an axillary nerve injury that is transient in nature.

D. Common Causes of Shoulder Pain. Once exclusionary diagnoses have been ruled out, most cases of shoulder pain fall into five categories. Rotator cuff impingement/tendonitis is secondary to overuse and degenerative changes about the shoulder or to repetitive use/micro trauma in throwing athletes and high-risk occupations such as carpenters. Initial treatment includes analgesics, ROM exercises, activity modification, and occasional subacromial injections. Surgery is considered if nonoperative measures fail. Rotator cuff tears may occur in young people (< age 50 years) as a result of a traumatic injury and warrant surgical repair for optimal results. In patients over the age of 50 years, a trial of rehabilitation is considered unless the patient has profound weakness. Frozen shoulder or adhesive capsulitis is often seen in diabetics, patients with renal disease, and idiopathic cases in women over the age of 50. Patients experience a painful period of global shoulder motion loss followed by a period of less painful gradual “thawing” lasting 6–9 months. ROM exercises, analgesics, and injections can lessen the symptoms but surgery is considered in the most refractory cases. A-C joint injuries are a result of trauma and most often treated with short-term immobilization. Severely displaced A-C separations or those that develop posttraumatic arthritis warrant surgical treatment. Glenohumeral arthritis is the end stage condition that may be posttraumatic, inflammatory, or degenerative in nature. Patients are treated symptomatically until pain becomes severe and total shoulder arthroplasty is the treatment of choice.
Shoulder Pain

A. History and physical exam
   - mechanism of injury
   - patient age
   - range of motion

B. Imaging studies
   - plain radiographs
   - CT, MRI

C. Exclusionary diagnoses
   - infection: aspiration, antibiotics
   - neoplasm: biopsy, CT, bone scan

D. Common causes
   - rotator cuff impingement
     - conservative therapy,
     - surgery if not better in 6 mo.
   - AC-joint separation
     - sling and rehabilitation,
     - surgery if severe

- rotator cuff tear
  - surgery if young, active
  - no surgery if older and has good function
- frozen shoulder
- glenohumeral instability
  - surgery if patient has pain or disability
- glenohumeral arthritis
  - conservative treatment
  - surgery for severe pain
  - joint replacement if end-stage
Hip fractures in the elderly are typically lower energy in nature than hip fractures in younger populations. Both, however, require prompt and appropriate surgical treatment to prevent significant morbidity. Even the 1-year mortality rate following hip fractures is elevated when compared to age-matched controls. Complications, in either case, include posttraumatic arthritis, avascular necrosis, and thromboembolic disease. The goal of treatment is to restore patients, as early as possible, to their prefracture level of function.

A. Initial Approach: In lower energy mechanisms, a complete history includes determination of loss of consciousness, syncopal episodes, cardiac symptoms, and prefracture ambulatory status; physical and radiographic exams are more important in higher energy accidents. The initial exam should consist of inspecting the limb for shortening, rotational deformity, skin condition, neurological function, and vascular status. Pain with axial compression or log roll may indicate an occult fracture. Routine radiographs include an AP Pelvis, an AP of the hip in internal rotation to best view the femoral neck, and a cross-table lateral.

B. Dislocation: Hip dislocations are usually caused by higher energy mechanisms, including falls from heights and motor vehicle accidents. Up to 90% are posterior dislocations and occur with the knee flexed. As with all high-energy accidents, concomitant chest, abdominal, and spine injuries must be ruled out. Then, an urgent closed reduction of the hip is performed and repeat radiographs, including internal and external oblique views (Judet views), are obtained to ascertain a concentric reduction. Computerized tomographic (CT) scans are routinely obtained following reduction in order to demonstrate the presence of small intra-articular fragments, to assess the congruence of the femoral head, and to assess femoral and acetabular fractures. Femoral head fractures occur in 10% of posterior dislocations.

C. Occult Fracture: Standard radiographs can usually identify most hip fractures. If the radiographs are negative and a high suspicion for injury persists, an MRI or bone scan can be performed to rule out an occult fracture. CT scans are not effective for ruling out an occult fracture in this setting. Non-displaced fractures discovered by advanced imaging can be pinned in place with three cancellous screws.

D. Femoral Neck Fracture: Femoral neck fractures disrupt the blood supply to the femoral head and frequently lead to osteonecrosis. Nondisplaced or slight valgus impacted fractures can be pinned in place with three cancellous screws. While it is appropriate to wait 12–24 h for an elderly patient’s medical clearance, reduction and operative stabilization of a displaced fracture should be performed as soon as possible. Hemiarthroplasty can allow earlier mobilization. If preexisting acetabular disease exists, a total hip arthroplasty can be considered. Displaced fractures in young patients (<60 years) should be emergently reduced and pinned because the likelihood of developing avascular necrosis increases with time.

E. Intertrochanteric Fractures: Intertrochanteric fractures are extra-capsular and thus do not tend to disrupt femoral head blood supply. These fractures are stabilized with a dynamic hip screw (DHS) or intramedullary fixation. Patients with intertrochanteric fractures are more likely to be older, in poorer health, and have more comorbid conditions. Mortality rates are higher in this population.

F. Subtrochanteric Fractures: Subtrochanteric fractures occur within 5 cm of the lesser trochanter and are stabilized with a dynamic compression screw (DCS) or intramedullary fixation.

All hip fractures should receive appropriate thromboembolic prophylaxis to avoid the high incidence of deep vein thrombosis and fatal pulmonary embolism.
Extremity Fractures

Eric M. Berkson

A. Initial Evaluation: Evaluation of extremity fractures focuses on the trauma in his/her entirety. Namely, the ABCs of a standard trauma resuscitation are first addressed and injuries to the head, chest, abdomen, pelvis, and spine are ruled out. In the secondary survey, appropriate radiographs are obtained, including any area where tenderness or deformity is noted. An anterior-posterior (AP) and lateral x-ray of the affected bone, as well as radiographs of the joint above and below the injury, are obtained. In addition, a complete history is obtained to elucidate the mechanism and forces that caused the fracture. The neurovascular status of the injured limb must be assessed, as well as the condition of skin and soft tissues. Finally, fractures should be monitored for the development of compartment syndrome.

Compartment syndrome results from the inability of the muscular compartments to accommodate increased tissue volume due to edema, vascular injury, or hematoma. As a result, the pressure in the compartment increases, compromising blood flow to the tissues, possibly causing irreversible damage to muscle and nerves if left untreated. This will occur within hours. The presence of a tense compartment, severe pain, or pain with passive range of motion suggests the possibility of a compartment syndrome and should be treated with an emergent fasciotomy. If there is doubt in the diagnosis or if the patient is unconscious, compartment pressures should be measured and monitored. Loss of sensory and motor function and loss of pulses in the involved extremity are very late signs of compartment syndrome. Prevention of this very serious problem is with prompt anatomic splinting of fractures and early surgical stabilization, combined with a high index of suspicion and measurement of compartment pressures.

B. Open Fractures: Open fractures are those in which the fracture and its hematoma communicate with the external environment. These require emergent surgical debridement and irrigation to reduce the likelihood of infection. The wound should be examined only once in the emergency department after which a sterile dressing is applied. The extremity should be splinted in an anatomic position and tetanus prophylaxis and intravenous antibiotics administered, according to the grade of the fracture. Angiography, if necessary, can be performed at this time, if the integrity of the blood supply is in question.

While historically grading an open fracture was described by the size of the skin opening, a more accurate grading system involves an assessment of the level of contamination. A Grade I fracture has minimal soft tissue damage, is clean with a small skin opening, and has only minimal muscle contusion. At the time of debridement, this wound should be extended to allow for thorough inspection of the fracture. After adequate sharp debridement, the wound is irrigated with 10L of saline containing antibiotics. The surgical portion of the wound is closed and soft tissue coverage of the fracture obtained. These fractures are usually treated similarly to a closed injury.

Grade II injuries have a moderate amount of contamination and soft tissue damage. They typically have soft tissue disruption from 1 to 10cm in length. Similar to a Grade I injury, the wound is meticulously debrided and irrigated. The presence of soft tissue damage may dictate that an external fixator be used as a temporizing device during soft tissue healing.

Grade III injuries have significant contamination and soft tissue disruption and typically have greater than 10cm of soft tissue injury (IIIA). If the injury occurred on a farm or has gross contamination (IIIB), or if there is an associated vascular injury (IIIC) additional antibiotics are added. Amputation of the limb should be considered for fractures with prolonged ischemia, tibial nerve injuries leading to an insensitive foot, and severe crush injuries. The Mangled Extremity Severity Score (MESS) can assist in this determination (Table 19.1).

C. Closed Fractures: Closed fractures of the extremities can be separated into those which are intra-articular and those which are extra-articular. Intra-articular fractures require anatomic reduction to reduce the likelihood of developing post-traumatic arthritis and to prevent arthrofibrosis. CT scans can often assist in determining intra-articular displacement. If the intra-articular stepoff is greater than 2mm, anatomical reduction via open reduction and internal fixation should be performed.
Occasionally, closed reduction using ligamentotaxis will produce a satisfactory articular surface. Limited internal fixation or percutaneous pins can be used in this instance. Fractures with less than 2 mm stepoff and a stable fracture pattern can be treated with a cast or hinged orthosis. All intra-articular fractures, especially articular fracture dislocations, must be assessed for neurovascular injuries. Early mobilization of the joint is absolutely important to provide nutrition to the injured cartilage and to maintain mobility.

**Table 19.1. Mangled Extremity Severity Score*.**

<table>
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<th>Points</th>
<th>Description</th>
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| 1 | Skeletal/soft tissue injury
| Low energy (stab, simple fracture, “civilian” gunshot wound) |
| 2 | Medium energy (open or multiple fractures, dislocation) |
| 3 | High energy (close-range shotgun or “military” gunshot wound, crush injury) |
| 4 | Very high energy (same as above but with gross contamination, soft tissue avulsion) |
| 1 | Limb ischemia
| Pulse reduced or absent but normal perfusion |
| 2 | Pulselessness, paresthesias, diminished capillary refill |
| 3 | Cool, paralyzed, insensate, numb |
| 0 | Shock
| Systolic blood pressure always >90mmHg |
| 1 | Hypotensive transiently |
| 2 | Persistent hypotension |
| 0 | Age (years)
| <30 |
| 1 | 30–50 |
| 2 | >50 |


D. **Diaphyseal Fractures:** Diaphyseal fractures may be stable (transverse with minimal comminution) or unstable (oblique, spiral, segmental, or comminuted). Unstable fractures or fractures in the trauma patient require surgical stabilization. In general, unstable fractures of the femur and tibia are treated with closed intramedullary nailing. Unstable fractures of the upper extremities are treated with open reduction and internal fixation. Stable long bone fractures without significant angular or rotational deformity may be treated with casting and close observation.

E. **Metaphyseal Fractures:** Metaphyseal fractures are usually unstable and require open reduction and internal fixation. They are generally not amenable to intramedullary nailing as the medullary canal is relatively wide at the metaphysis, and the nail itself takes relatively little purchase on the metaphyseal fragment. Metaphyseal fractures may be associated with neurologic or vascular injuries because of the tethering of these structures around joints, particularly the knee and elbow.
Extremity Fractures

A. Initial evaluation
   history, physical exam
   radiographs
   compartment syndrome?
   assess neurovascular status

B. Open fractures
   sterile dressing
   anatomic splinting
   IV antibiotics, tetanus
   angiography if needed

   grade I fracture
   (clean, small opening)
   → debridement, wound irrigation,
   fixation as for closed injury

   grade II fracture
   (moderate contamination,
   1-10 cm tissue disruption)
   → debridement, irrigation, internal
   or external fixation

   grade III fracture
   (> 10 cm soft tissue injury)
   → limb salvage vs. amputation

C. Closed fractures
   Intra-articular → CT scan
   > 2 mm stepoff,
   unstable pattern
   → open reduction
   internal fixation
   early mobilization
   < 2 mm stepoff
   stable pattern
   → closed reduction
   cast
   early mobilization

D. Diaphyseal fracture
   extra-articular
   unstable or trauma
   → intramedullary nail (femur, tibia)
   plating (radius, ulna, humerus)
   stable
   → cast
   unstable or trauma
   open reduction,
   internal fixation

E. Metaphyseal fracture
   stable
   → cast
   unstable or trauma
Burns
Laura J. Moore

A. Primary Survey: Each year nearly 2 million Americans seek medical care for burn injuries. These injuries range from minor burns sustained in domestic cooking accidents to extensive full-thickness burns with associated traumatic injuries. Burns can result from exposure to flames, chemical contact, electrical current, or exposure to hot liquids (thermal substances). Because of the frequent presence of associated injuries, the burn patient must be evaluated in the same manner as any trauma patient. This evaluation is based on a systematic approach that identifies life-threatening injuries and initiates prompt treatment. The primary survey, which uses the mnemonic “ABC,” focuses on A—securing an airway, B—assessing breathing, and C—assessment of circulation and establishing vascular access. Establishment of IV access in the burn patient should preferably be via two large bore catheters placed in unburned skin. Once the primary survey is completed, a more focused secondary survey should be performed. The secondary survey should determine the percent of the body surface area burned, the degree of burn, the presence of inhalation injury, and the presence of other traumatic injuries.

B. Burn Surface Area: One critical aspect in the evaluation of the burn patient is calculation of the total body surface area (TBSA) of the burn. The TBSA can be calculated using the “Rule of Nines.” In adults, the head and neck and each upper extremity count as 9% of the TBSA. The lower extremities, anterior trunk, and posterior trunk each account for 18% of the TBSA. For smaller burns, one can use the palm of the hand and assume that this represents 1% of the TBSA. Assessment of the TBSA allows for calculation of the proper amount of crystalloid to be given during the resuscitation phase. The Parkland Formula is the most commonly used formula for the calculation of fluid requirements in the burn patient. According to the Parkland Formula, 4 ml/kg/%TBSA of lactated ringer’s solution should be administered during the first 24h. Half of this fluid volume is given over the first 8h postburn. The remaining fluid volume is given over the next 16h. The fluid volume administered should be adjusted on an hourly basis to achieve a urine output of 0.5 cc/kg/h in adults and 1–2 cc/kg/h in children.

C. Burn Degree: Determination of burn depth is a critical aspect in the evaluation of the burn patient. First-degree or superficial thickness burns are characterized by erythema and pain. These burns blanch to the touch. Injury is confined to the epidermis. Examples of superficial burns include sunburns and minor scalds. Second-degree or partial thickness burns are characterized by erythema, pain, and bullae. By definition, second-degree burns involve the epidermis and dermis. These burns are subclassified into two types, superficial partial and deep partial. Superficial partial thickness burns affect the epidermis and superficial dermis. They are characterized by severe pain, moist erythema, and bullae formation. Superficial partial thickness burns usually heal within two weeks by reepithelialization from the hair follicles and sweat glands. Deep partial thickness burns involve the epidermis and the deep layer of the dermis (reticular dermis) with destruction of most of the hair follicles and sweat glands. These burns are more pale and mottled in appearance, remain painful, and do not blanch to the touch. They heal in 14–35 days, often with severe scarring due to loss of the dermis. These burns usually require split thickness skin grafting because of the slow rate of reepithelialization. Third-degree or full-thickness burns are characterized by dark, charred eschar with white dry patches and are not painful. All layers of the skin are destroyed by coagulation necrosis. All full-thickness burns require excision of the eschar followed by skin grafting.

Treatment of burn injuries utilizes both topical agents and surgical techniques. Circumferential deep partial and full-thickness burns of the extremity can compromise blood flow to the limb. Impaired venous outflow from edema in the tissues underlying the eschar eventually impairs arterial inflow. In these situations, emergent release of the burn eschar by incising the medial and lateral aspects of the extremity must be performed. Limb integrity can be assessed using digital and distal extremity Doppler studies and digital pulse oximetry. Performance of escharotomies should restore blood flow to the affected limb until formal excision of the burn wound can be performed. Early excision and grafting of deep partial thickness and full-thickness burns reduces the risk of inflammation.
and infection within the burned tissues. This point cannot be overemphasized. Excision of dead tissue avoids the complications of bacterial overgrowth in necrotic areas. A barrier, however, needs to be established otherwise the benefit of tissue excision will not be realized. Coverage of the excised burn can be accomplished with either allograft (cadaver) skin or autograft (preferred if donor sites are available).

A variety of topical agents are currently available for the treatment of burn wounds. (Table 20.1) These topical agents minimize bacterial and fungal growth within the wound, reduce evaporative heat loss, and decrease pain in the burn wound. Silver sulfadiazine (Silvadene) is a topical broad-spectrum antimicrobial salve commonly used in burn care. Silver sulfadiazine is painless upon application and has no metabolic side effects. Its one disadvantage is that it does not penetrate eschar. Mafenide acetate (Sulfamylon) is another topical agent frequently used in burn wound care. Unlike silver sulfadiazine, mafenide acetate penetrates eschar but is painful upon application and causes a metabolic acidosis. Silver nitrate 0.5% solution is also a topical broad-spectrum antimicrobial. It penetrates eschar poorly and can leech sodium and potassium from the wounds.

Inhalation injury is a frequent cause of morbidity and mortality in the burn patient. Inhalation injury should be suspected in any patient with a history of closed space smoke exposure, prolonged extrication time, singed nasal hairs, facial burns, or carbonaceous sputum. Patients suspected of having inhalation injury should be immediately intubated. Following intubation, a bronchoscopy should be considered and frequently reveals carbonaceous sputum and mucosal erythema or ulceration. The inhalation of toxins is the primary mechanism of inhalation injury, the sequela of which include upper airway obstruction due to progressive edema, bronchospasm, occlusion of small airways due to sloughing of endobronchial cellular debris, loss of the ciliary clearance mechanism, and loss of capillary integrity with subsequent interstitial and alveolar edema. Treatment of inhalation injury requires aggressive pulmonary toilet and positive pressure ventilation to reduce intrapulmonary shunting and improve lung compliance.

Nutritional support of the burned patient should not be overlooked. The enteral route is preferred and feedings are started on the second postburn day. Adequate caloric intake is associated with faster healing and fewer septic complications.
Burns

A. Primary Survey
   Airway
   Breathing
   Circulation

Secondary Survey

B. Calculation of % TBSA
   "rule of 9's"

   fluid resuscitation

   Parkland formula:
   4 ml/kg/%TBSA
   1/2 given in 1st 8 hrs
   1/2 given in next 16 hrs

   adjust fluid resuscitation on an hourly basis

C. Determination of Degree of Burn

   first
   second
   third

   superficial
   deep

   Topical therapy:
   sildavene
   sulfaamylon
   silver nitrate

   Surgical therapy:
   escharotomies
   burn excision
   skin grafting

   allograft
   autograft (preferred)

Evaluation of Airway Injury

Evaluation of Other Traumatic Injuries
Part III
Vascular Problems
Chronic Lower Extremity Ischemia

Chad E. Jacobs

A. History. The most common cause of chronic lower extremity arterial insufficiency is atherosclerotic occlusive disease. Several other disease processes may mimic lower extremity arterial insufficiency, such as osteoarthritis, neurospinal compression, and diabetic neuropathy. The clinical picture may be further clouded by the coexistence of several of these diseases. Chronic lower extremity ischemia is best described as being either “functional” or “critical.” A careful and thorough history will almost always provide key information to help differentiate either functional or critical arterial insufficiency from other causes of leg symptoms.

Intermittent claudication is the hallmark of functional limb ischemia. Claudication develops when blood flow to the exercising muscle mass is unable to meet the requirements of increased metabolic activity. Three essential features of claudication are that the pain is always felt in a functional muscle unit, the pain is reproducible with exercise, and the pain is relieved within minutes of exercise cessation. If these three conditions do not exist, another etiology is likely the cause of the leg pain.

Chronic limb ischemia is determined to be critical if a patient has ischemic rest pain, ulceration, or gangrene of the foot or toes. Rest pain is unlike claudication in that it is experienced in the foot or toes rather than in a functional muscle group. Rest pain often occurs at night after the leg has been elevated for a short time, and may be relieved by dangling the affected extremity over the side of the bed. The presence of rest pain implies that there is less blood flow to the extremity than is required for normal resting tissue metabolism. If left untreated, rest pain will usually progress to tissue necrosis. Rest pain in itself may be debilitating due to the constant pain, muscle paresis, and paresthesias that are often present.

Risk factor assessment is of paramount importance in evaluating the patient with chronic ischemia. Atherosclerosis is a systemic disease, and it is necessary to question the vascular patient about symptoms of coronary and cerebrovascular atherosclerotic disease and pursue further diagnostic work-up as needed. Medical control of hypertension, diabetes mellitus, and hyperlipidemia is also an important aspect of the history and management. The use of tobacco has conclusively been shown to accelerate the progression of atherosclerotic disease, so patients who use tobacco should be enrolled in smoking cessation programs early in their course.

B. Physical Exam. Because atherosclerosis is a systemic disease, the temptation to examine only the lower extremities must be avoided in the patient with chronic leg ischemia. One may hear a new carotid bruit, palpate an abdominal aortic aneurysm, or diagnose atrial fibrillation in the course of a thorough physical examination. In addition to palpation of pulses at the femoral, popliteal, dorsalis pedis, and posterior tibial locations, auscultation over the course of the major vessels to listen for bruits may provide important information regarding the location of stenoses. A patient with iliac stenosis, for example, will often have not only a diminished femoral pulse on the affected side but a femoral bruit as well.

Atrophy of the calf muscles and loss of hair on the dorsal surface of the foot and toes are relatively common signs of arterial insufficiency. Toenails may become thickened because of slow nail growth. In more advanced ischemia, the foot may develop a shiny “skeletonized” look because of atrophy of the skin and subcutaneous tissue. Delayed capillary refill beyond 2–3 s is also a sign of advanced ischemia. Areas of localized pallor or cyanosis are often a precursor of ulceration and gangrene. Edema of the extremity may often be present, either due to underlying congestive heart failure or from continuously keeping the leg in a dependent position in an attempt to relieve the rest pain.

C. Noninvasive Studies. A stenotic lesion within an artery increases resistance to arterial flow and results in a pressure drop across the lesion. It is this principle which makes the ankle-brachial index (ABI) a useful first-line test for patients with chronic ischemia of the lower extremity. To obtain the ABI, a pneumatic cuff is placed around the ankle and the pressure is measured at both the dorsalis pedis and posterior tibial arteries with the use of a Doppler probe. The higher of the two is divided by the brachial artery pressure and the index is obtained. The ABI in a normal, well-rested individual lying supine averages 1.1. Note that in patients with diabetes, calcification of the distal lower extremity vessels is common. This results in a falsely
in the toes. A decreased ABI indicates that occlusive disease is present and may indicate how many levels of obstruction are present, this measurement does not provide information regarding the location of the disease. Segmental lower extremity pressure measurement does provide this crucial information. To obtain this measurement, pneumatic cuffs are placed around the thigh at groin level, around the thigh above the knee, around the calf below the knee, and at ankle level. The Doppler probe is held at the dorsalis pedis or posterior tibial artery and each cuff is sequentially inflated to obtain a segmental pressure. The Doppler ultrasound probe is also used to obtain an arterial waveform at each level.

In patients with intermittent claudication, the segmental pressure measurements may sometimes be normal. To clarify the cause of symptoms in such patients, segmental pressure measurement before and after exercising on a treadmill may be quite useful. The exercise effectively stresses the peripheral circulation and may unmask lesions that were not detectable at rest. It may also enable the physician to assess the degree of disability that is produced by the patient’s occlusive disease.

Digital plethysmography is routinely used to obtain pressures in the toes. Toe pressures are significant because they are taken from the most distal portion of the extremity and reflect the physiologic status of all the proximal arteries. These measurements are particularly useful in patients with calcified distal vessels where the ABI and segmental pressures are spuriously elevated. A toe pressure of less than 30 mmHg signifies severe ischemia.

D. Angiography. Despite advances in less invasive modalities for evaluating the vascular system such as Duplex ultrasonography and magnetic resonance angiography, arteriography remains the gold standard and the most commonly used technique to plan arterial reconstruction. In addition to providing diagnostic information, the advent and rapid expansion of endovascular technology has allowed for therapeutic intervention to become a safe and common facet of angiography.

Abdominal aortography with bilateral lower extremity runoff is the standard means of preoperative evaluation. This study provides information regarding both the level and the severity of disease. Generally a lesion is not considered to be hemodynamically significant unless it causes greater than 50% reduction in luminal diameter. If there is question as to whether a lesion causes significant flow reduction, pressures can be measured both proximal and distal to the suspected lesion. A measured pressure gradient of 15 mmHg or more between two adjacent arterial segments is considered significant.

Since contrast agents are cleared from the body by glomerular filtration, particular attention should be paid to a patient’s renal function. In patients with diminished renal function, contrast use should be minimized and adequate hydration should be provided. There are a variety of contrast agents commercially available, several of which are less nephrotoxic and should therefore be used in patients with diminished creatinine clearance.

E. Treatment. It is important to decide prior to extensive testing who is a candidate for intervention. Patients with functional ischemia have little risk of limb loss. Approximately 75% of such patients will remain stable or improve over 2–5 years, and only 5–7% will eventually require amputation of the affected leg. If such patients are able to abstain from tobacco use, participate in a regular exercise program, and medically control associated diseases, they can expect a reasonable outcome without vascular reconstruction. However, if claudication is debilitating and prevents active participation in daily activities or employment, then surgery or other intervention should be considered. Patients with critical limb ischemia are candidates for revascularization provided their disease is sufficiently localized so as to allow intervention with a reasonable chance of success, and provided the patient’s comorbidities will not preclude safe intervention.

Once the decision has been made to intervene, several questions must be addressed. Some lesions are more amenable to percutaneous treatment, while others are more amenable to surgical correction. Several different types of conduit are available for bypass grafts. Most such decisions are based on current literature, experience, and institution variability.

Aortoiliac occlusive disease has historically been treated by aortobifemoral bypass graft, which has been shown to have excellent long-term patency. If laparotomy is prohibitive, then an axillofemoral bypass with cross-femoral bypass graft using ringed prosthetic can be performed. Unilateral and bilateral iliac disease can be treated in a variety of ways. Percutaneous transluminal angioplasty (PTA) and stenting of short segment iliac disease is a viable alternative that may provide adequate inflow and eliminate symptoms. This technique can also be used in conjunction with distal bypass. An extra-anatomic approach, such as axillofemoral or crossfemoral bypass, is an alternative in the setting of unilateral iliac disease, as is an ilioliac or iliofemoral bypass.

Surgical revascularization rather than percutaneous therapy is currently the mainstay of treatment for infrainguinal disease. Femoral popliteal bypass successfully treats occlusion of the superficial femoral artery, which most commonly occurs at the adductor or Hunter’s canal. For bypasses targeting the above-knee popliteal artery, vein grafts and prosthetic grafts have shown nearly equal long-term patency. For all below-knee bypass procedures, vein should be used as conduit since it has been shown to have superior long-term results. Bypass to the pedal vessels is an alternative for patients with trifurcation disease. Such bypass grafts can originate from the common femoral, profunda femoris, superficial femoral, or popliteal arteries.

The most important principles for surgical revascularization are meticulous attention to technical details, and ensuring that there is both adequate inflow and outflow for the bypass being performed.
Chronic Lower Extremity Ischemia

A. History
   functional vs. critical risk factors (coronary disease, hypertension, diabetes, smoking)

B. Physical Examination
   muscle atrophy, hair loss, bruits, pallor, cyanosis, ulceration, gangrene

C. Non-invasive Studies
   ABI
   segmental pressures
   toe pressures

   ABI < 0.5
   mild - moderate disease

D. Angiography
   level, severity of disease
   gradient across lesions
   angioplasty stenting

   inflow disease
   outflow disease

E. Surgery
   fem-pop bypass
   fem-distal
diabetic-distal endarterectomy

E. Surgery
   aortobifemoral bypass
   fem-fem bypass
   axillary-femoral bypass
   endarterectomy

E. Percutaneous Techniques
   angioplasty stenting

Medical Management
   stop smoking
   exercise programs
   control DM, HTN diet
Acute limb ischemia is one of the most common challenging problems faced by the vascular surgeon. Despite the advanced surgical and critical care, the morbidity and mortality have remained unchanged for the last two decades. The mortality rate is greater than 25% secondary to comorbid medical problems and the amputation rate is about 20% in survivors.

Acute arterial occlusion tends to propagate and occlude collaterals. The ischemic tissue starts to swell in a tight fascial compartment and eventually leads to compression of the neurovascular bundles, hence the “compartment syndrome.” The “no reflow” phenomenon is the terminal event when the arterioles, capillaries, and venules are occluded and there is no distal perfusion despite a successful revascularization. Skin and subcutaneous tissue tolerate ischemia better than peripheral nerves and muscles. Peripheral nerves and muscles start to develop irreversible damage within 6 h. Therefore, once the diagnosis is made, the patient should receive therapeutic intervention immediately.

In addition to possible limb loss, “reperfusion injury” from revascularization of the extremity may lead to multisystem organ failure. Superoxide and hydroxyl radicals, acid and potassium from injured cells, cardioprepressants, and harmful cytokines are incriminating factors in reperfusion injury. Moreover, myoglobin from dead muscle cells precipitates in the renal tubules, which leads to acute tubular necrosis and renal failure.

A. Etiology: There are two main causes of acute limb ischemia: thrombosis and embolism, the former outnumbers the latter by a ratio of 6:1. Regarding thrombosis, bypass graft thrombosis occurs at a slightly higher rate than native artery thrombosis by a ratio of 5:4. Atherosclerosis, low-flow states, and hypercoagulable states cause thrombosis of the native arteries. On the other hand, thrombosis of vascular bypass grafts is caused by technical or mechanical problems, intimal hyperplasia, and progression of atherosclerotic disease. Most arterial emboli originate from the heart (60–70%), usually after a myocardial infarction (MI) or cardiac arrhythmia. Other arterial emboli originate from valvular heart disease, aneurysmal disease, and paradoxical embolus from the right heart to the left heart through a patent foramen ovale.

B. Clinical Diagnosis: A patient with an acute cold leg, history of a recent MI or atrial fibrillation, no previous claudication or rest pain, and a palpable contralateral pulse has an arterial embolism until proven otherwise. The sudden onset of acute arterial occlusion is manifested by some or all of the following six cardinal signs, the “6 Ps”: Pulselessness, Pain, Pallor, Paresthesia, Paralysis and Poikilothermic.

Alternatively, a smoker or patient with a history of diabetes, who complains of pervers claudication or rest pain, is more likely to have thrombosis of the native artery. In patients with a previous bypass graft, who present with an acute cold leg, thrombosis of the bypass conduit is the most likely cause. Pulses are usually not palpable bilaterally and some of the “6 Ps” signs may be present on physical exam depending on the arterial collaterals.

C. Management: The following principles apply to the acute cold leg:

1. Patients presenting with the “6 Ps” and do not have a pulse or arterial Doppler signal have a vascular emergency until proven otherwise.
2. Patients diagnosed with acute limb ischemia should have full anticoagulation with an intravenous bolus of heparin (100 units/kg) followed by a continuous IV drip (10 units/kg). The goal of heparinization is to prevent proximal and distal arterial thrombus propagation, distal arterial thrombosis, and venous thrombosis.
3. Arterial embolization should be suspected in a patient with previous MI or atrial fibrillation with a palpable contralateral pulse. Patients with arterial embolization should proceed to the operating room (OR) immediately without further testing. An embolectomy is performed if the limb is viable or salvageable. A bilateral femoral artery exposure is mandatory for aortic saddle embolus for simultaneous balloon embolectomy. A femoral artery approach is used for iliac and femoral emboli. A below the knee popliteal artery
approach is used for the peroneal, anterior, and posterior tibial artery embolus.

4. Patients with irreversible ischemia (an anesthetic and paralyzed extremity) should be offered amputation. The level of amputation should be well demarcated by skin temperature and appearance.

5. Patients suspected of having native artery or bypass graft thrombosis should undergo angiography immediately; this can be performed in the OR if the interventional radiologist is not available. Arterial thrombosis secondary to atherosclerotic occlusion should be treated with arterial bypass. An artificial conduit should be used for an above the knee arterial bypass, while the autologous vein should be used for bypass below the knee.

Treatment for bypass graft thrombosis depends on the arterial outflow bed. If the arterial outflow bed is acceptable (patent distal vessels with flow to the rest of the extremity on arteriogram), a bypass graft replacement is the best treatment. For an inadequate or absent arterial outflow bed, the only treatment option is amputation.

6. A completion arteriogram should be done in the OR to assess technical problems of the bypass conduit or the completeness of embolectomy.

7. Consider four compartment (anterior, medial, superficial posterior, and deep posterior) fasciotomies if ischemia time is greater than 4h.

8. Consider treatment for myoglobinuria with IV hydration, mannitol, and alkalization of urine, and maintain greater than 100 cc/h urine output. Monitor for hyperkalemia and other organ injuries.

9. Thrombolytic therapy plays no role in emergency cases when the patient has no pulse or Doppler signals. Thrombolytic therapy generally takes more than 12h, as peripheral nerves and muscles tolerate ischemia only for 6h.
A. Etiology
Thrombosis
Embolism

B. Clinical Diagnosis
History recent MI
History atrial fibrillation
Claudication
Contralateral pulse

“6-P’s”
Pulselessness
Pain
Pallor
Paresthesia
Paralysis
Poikilothermic

C. Management
Anticoagulation with heparin

If embolism suspected, immediate surgery embolectomy
Amputation for anesthetic, paralyzed extremity
If thrombosis suspected, perform angiography then do arterial bypass
Completion arteriogram to assess adequacy of surgery
If ischemia time >4 hours, perform fasciotomies
Protect against myoglobinuria
Myocardial Infarction
Clifford J. Kavinsky and Paul W. McMullan

Coronary artery disease is the leading cause of death in North America and Europe. Each year in the United States, approximately one million people are admitted to hospitals with the diagnosis of acute myocardial infarction (MI), and another 200,000–300,000 die before reaching the hospital. Overall mortality is about 40%.

Recent advances in the understanding of the pathogenesis of MI indicate that the inciting event is most often coronary artery occlusion due to atherosclerotic plaque rupture followed by thrombosis. The majority of “culprit” lesions occlude less than 50% of the arterial lumen prior to the event. The thin, unstable fibrous cap overlying a lipid-laden core fissures at a weak point, exposing blood in the lumen to the plaque’s underlying thrombogenic contents. Platelet adhesion immediately occurs, followed by platelet aggregation and fibrin cross-linking. Once the thrombus is formed, blood flow in the artery stops and the myocardium in that arterial territory begins to die. The amount of that territory which can be rescued from necrosis is directly related to the rapidity with which reperfusion is established.

A. Diagnosis. Diagnosis of acute MI is based on three critical criteria: (1) Ongoing chest pain, (2) Characteristic EKG changes, and (3) Elevation in cardiac-specific markers. A targeted history should elicit time of onset of pain, which is typically of a squeezing or crushing nature, often associated with radiation to the left arm or jaw. Patients may also present with atypical symptoms or “anginal equivalents” such as shortness of breath, nausea, diaphoresis, or isolated arm or jaw pain. Diabetic patients and the elderly are particularly prone to atypical presentations.

The electrocardiographic (ECG) diagnosis of acute MI is made by demonstrating ST segment elevation of greater than or equal to one millimeter in two contiguous leads or the presence of a new left bundle branch block (LBBB) pattern in a patient experiencing characteristic chest discomfort. One should keep in mind that 50% of patients presenting with inferior MI (ST segment elevation in leads II, III, and aVF) will have right ventricular involvement and should have a right-sided ECG, which may demonstrate ST segment elevation or “Q” waves in the right-sided precordial leads, particularly V3R or V4R.

Also, 50% of ECGs in patients presenting with an acute MI do not display ST segment elevation and are nondiagnostic. They may demonstrate only ST depression, T-wave changes, or other nonspecific features. Since this is the case, recent changes have been made which designate infarctions with the aforementioned typical pattern as “ST-segment-elevation MIs” and others which present with elevations in cardiac markers but without ST elevation as “non-ST-segment-elevation MIs.” The terms Q-wave and non-Q-wave MI have been supplanted by this new terminology. Treatment of the patient with a non-ST-segment-elevation MI is evolving rapidly, and current therapy involves the use of heparin, glycoprotein IIb/IIIa inhibitors, and percutaneous intervention. Further elaboration on this topic is beyond the scope of this chapter, and the discussion that follows will focus on ST-segment-elevation MI.

Cardiac markers should be included in the immediate blood work. Troponin I is an extremely sensitive and specific indicator of myocardial necrosis but takes 6–12h from symptom onset to begin to rise. Myoglobin is a nonspecific indicator and can be elevated by any skeletal muscle or myocardial injury, but it rises within 1–2h of symptom onset, making it a valuable diagnostic tool for patients arriving early in the course of chest pain. CKMB is a fractionated portion of total creatine kinase, and an elevation in the MB index (depending on a given laboratory’s standards, over 2–5% of the total CK) is diagnostic of MI. Table 23.1 provides a time course for rise and fall of the markers in the bloodstream. Troponin I elevations can be prolonged in patients with renal insufficiency.

Echocardiography performed at bedside can also be of diagnostic assistance in patients with chest pain but nondiagnostic ECGs. The presence of a new left ventricular regional wall motion abnormality on two-dimensional imaging is indicative of ongoing ischemia or infarction.

B. Immediate Therapy. Immediate therapy in the first moments of evaluation should include the following:
1. 325 mg aspirin, chewed
2. Oxygen via nasal cannula
3. Cardiac and blood pressure monitoring
4. Intravenous access (with simultaneous blood draw for lab studies)
5. Nitrate therapy (sublingual followed by intravenous)
6. Analgesia (morphine or meperidine if morphine allergy present)
7. Consider beta blocker therapy (IV metoprolol up to 15 mg in 3 divided doses or IV atenolol up to 10 mg in 2 divided doses). Contraindications include heart rate below 60 and systolic blood pressure below 100 mmHg.

Once the diagnosis of acute MI is established, reperfusion therapy is the immediate goal. The possibilities are fibrinolytics, percutaneous transluminal coronary angioplasty (PTCA), and coronary artery bypass grafting (CABG). Patients are not candidates for fibrinolytics if chest pain has been continuously present for more than 12 h.

C. Fibrinolytics. Fibrinolytics are given systemically through the IV and serve to dissolve the newly formed thrombus in the coronary artery. Door-to-therapy goal time is 30 min. Approved fibrinolytics and their doses are listed in Table 23.2. Absolute contraindications include (1) previous hemorrhagic stroke at any time or other stroke or cerebrovascular event within one year, (2) known intracranial neoplasm, (3) active internal bleeding, and (4) suspected aortic dissection.

Relative contraindications include (1) uncontrolled severe hypertension (BP > 180/110 mmHg), (2) history of cerebrovascular event or other intracranial pathology not listed above, (3) anticoagulation (INR ≥ 2–3) or known bleeding diathesis, (4) recent (2–4 weeks) trauma, including head trauma, (5) noncompressible vascular punctures, (6) recent (2–4 weeks) internal bleeding, (7) prior allergic reaction to streptokinase, (8) pregnancy, (9) active peptic ulcer disease, and (10) history of chronic hypertension.

Intravenous heparin should be administered along with alteplase and reteplase. Reperfusion failure (i.e., persistent chest pain or ST elevation) is an indication for emergent PTCA.

D. PTCA. PTCA is often the first choice for reperfusion therapy depending on practices in the local medical community and availability of catheterization facilities and experienced operators. Goal for timing of PTCA is 60–90 min from the time of presentation. If this goal cannot be met, fibrinolytic therapy is preferential. The population that benefits most from PTCA as compared to fibrinolysis consists of those with anterior MI, persistent tachycardia, cardiogenic shock, or age over 70 years.

CABG is performed in the acute setting when reperfusion therapy by PTCA fails or is found to be impossible after visualization of the coronary arteries by angiography. It is reserved for such situations in the first 4–6 h after symptom onset and is not considered first-line therapy.

Prognosis. In the 1960s, in-hospital mortality rates for MI were as high as 35%. Since that time, evolution in understanding the disease process coupled with advances in therapy have produced a steady decline from 10–15% in the 1980s to the current low of 7–10%. In general, indicators of a poorer prognosis are advanced age, cardiogenic shock, tachycardia, and location of infarction in the anterior left ventricular wall.

### Table 23.1. Time course for rise and fall of the markers in the bloodstream.

<table>
<thead>
<tr>
<th>Marker</th>
<th>Onset of rise</th>
<th>Peak</th>
<th>Return to normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoglobin</td>
<td>1–2 h</td>
<td>6–12 h</td>
<td>24–36 h</td>
</tr>
<tr>
<td>CKMB</td>
<td>3–6 h</td>
<td>24 h</td>
<td>48–72 h</td>
</tr>
<tr>
<td>Troponin I</td>
<td>6–12 h</td>
<td>24–48 h</td>
<td>7–10 days</td>
</tr>
</tbody>
</table>

### Table 23.2. Approved fibrinolytics and doses.

<table>
<thead>
<tr>
<th>Fibrinolytic</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Streptokinase</td>
<td>1.5 MU in 30–60 min</td>
</tr>
<tr>
<td>Alteplase</td>
<td>100 mg in 90 min</td>
</tr>
<tr>
<td>Reteplase</td>
<td>10 U × 2 over 30 min</td>
</tr>
</tbody>
</table>

(continued)
Myocardial Infarction

A. Diagnosis
- ongoing chest pain
- EKG changes: ST elevation, new LBBB
- elevated cardiac markers
- echocardiography

B. Immediate Therapy
- chewed 325 mg ASA, oxygen, cardiac monitoring, intravenous access, nitrate therapy, analgesia, beta blocker

reperfusion therapy

C. Fibrinolytics
- goal is 30 minutes
- see contraindications

resolution of chest pain and elevation of ST segment?
- no → rescue PTCA
- yes

D. PTCA
- goal is 60-90 minutes

successful?
- no → consider CABG within 4-6 hours
- yes

admit to CCU for care
Abdominal Aortic Aneurysm

Bernadette Aulivola

A. Aneurysms are defined as a focal enlargement of the artery to greater than 1.5 times its expected normal diameter. A segment of abdominal aorta larger than 3 cm in diameter is therefore considered to be an abdominal aortic aneurysm (AAA). The prevalence of AAA is highest in white males. The pathogenesis is multifactorial, likely involving aortic wall degeneration by atherosclerosis, chronic inflammation, and increased proteolytic enzyme activity. Risk factors for AAA include advanced age, male gender, tobacco use, emphysema, atherosclerosis, and hypertension (HTN). First-degree relatives of those diagnosed with AAA have a 15–20% incidence of AAA and should therefore be advised to undergo ultrasound screening. Current screening recommendations also include performing abdominal ultrasound in all males over 65 years of age with a history of smoking. Approximately 80% of AAAs are associated with smoking.

Aortic aneurysms are most commonly located in the infra-renal aorta (90%). Only 5% of aneurysms requiring repair are suprarenal, necessitating reimplantation of at least one renal artery. Thoracoabdominal aneurysms are rare, accounting for only 2% of aortic aneurysms. Approximately 25% of AAAs are associated with concomitant iliac artery aneurysms. Although only 10% of patients with AAA will also have a popliteal artery aneurysm, 40% of those with popliteal aneurysms are found to have AAA, making screening for AAA in these patients essential.

The risk of AAA rupture is best predicted by the cross-sectional diameter of the aneurysm (Table 24.1). Patients with COPD and HTN have a higher rupture risk. The natural history of AAAs is such that, if left alone, they will enlarge and eventually rupture. Up to 80% of patients with ruptured AAA will not survive the event. Only 50% of rupture patients make it to a hospital alive and 50% of these will die before or after operative repair.

B. Most AAAs are asymptomatic until rupture. Approximately 80% are discovered incidentally by abdominal ultrasound, CT, MRI, or plain radiograph. Less than 5% of patients present with distal embolization. Physical examination detects only 50% of AAAs 4–5 cm in diameter and 75% of those >5 cm.

Ultrasound remains the simplest and most cost-effective modality for screening and initial diagnosis of AAA. CT and MRI both provide excellent images of the aorta and mesenteric and renal vessels and can be processed to provide 3-D images of the vasculature. Patients with known AAA should undergo spiral CT with 2–3 mm cuts to obtain accurate information regarding aneurysm size and extent. Fine-cut CT scan plays an important role in operative planning. MRI may be used if a contraindication to administering intravenous contrast exists. Angiography demonstrates aortic lumen caliber but does not accurately demonstrate aneurysm diameter given the variable amount of thrombus lining the aneurysm sac. It provides the most accurate image of branch vessel anatomy including renal artery anatomy, patency of the inferior mesenteric artery (IMA), and iliac artery aneurysm or occlusive disease. Angiography should be considered in patients with juxtarenal or suprarenal AAA, presumed renovascular HTN, chronic mesenteric ischemia, or lower extremity ischemia. It is also useful in planning for possible endovascular stent-graft AAA repair.

Decisions on elective aneurysm repair should take into account comorbid medical conditions and the risk of aneurysm rupture. AAAs less than 4 cm are unlikely to rupture and may be followed with ultrasound or CT scan on an annual basis to determine growth rates. AAAs with diameter growth rates of greater than 0.5 cm in 6 months or 1 cm in a year should be considered for repair. AAAs larger than 5–5.5 cm warrant elective repair, as the risk of rupture outweighs the operative risk in patients who are reasonable operative candidates. Because of the high incidence of rupture of large AAAs (>6 cm), even patients with elevated cardiac risk should be considered for repair.

C. Coronary artery disease (CAD) is the most accurate predictor of perioperative morbidity and long-term mortality and it is present in almost two-thirds of AAA patients. Since a large proportion of perioperative and late deaths are due to underlying CAD, careful preoperative assessment of cardiac status is essential prior to elective AAA repair. Stress echocardiography should be considered, particularly in patients with
a history of MI, ventricular arrhythmias, diabetes, or angina. Stress testing should be followed by cardiac catheterization to further investigate the severity of CAD in those with reversible defects. When indicated, coronary artery bypass grafting or angioplasty +/- stenting prior to AAA resection leads to improved survival.

D. Symptomatic AAs should be repaired urgently. Patients with a known aneurysm who present with abdominal or back pain and/or shock should be taken directly to the operating room. Hemodynamically stable patients with nonspecific complaints can be evaluated with a contrast-enhanced CT scan to rule out leaking aneurysm.

E. Patients undergoing elective AAA repair should receive preoperative antibiotics, usually a cephalosporin, to reduce the risk of graft infection. Ampic intravenous access, usually in the form of a central venous line, as well as arterial line and urinary catheter placement are routine. Epidural catheter placement for postoperative pain management should also be considered. Preoperative bowel preparation is generally not necessary. Operative approach is usually via a vertical midline transperitoneal incision or a left retroperitoneal dissection. The left retroperitoneal approach provides improved pararenal exposure and has been noted to be associated with decreased rates of postoperative respiratory complications and ileus. Alternatively, a right retroperitoneal approach may be used in cases where a stoma would interfere with this approach from the left. Relative indications for the retroperitoneal approach are a “hostile abdomen” after multiple previous transperitoneal surgeries, presence of an abdominal wall stoma, horseshoe kidney, inflammatory AAA, and expected need for renal revascularization. Proximal (usually infrarenal aorta) and distal (iliac artery) exposure is performed with minimal manipulation of the aneurysm to avoid distal embolization. Heparin (80 units/kg) is administered intravenously prior to vascular clamping. Most surgeons do not administer heparin in cases of ruptured AAA. In order to minimize the risk of embolization, distal vascular clamps are placed prior to application of the proximal clamp. The aneurysm sac is then opened, thrombus is removed, and lumbar arteries are ligated from within the sac to control backbleeding. Auto-transfusion devices are helpful in minimizing banked-blood transfusion intra-operatively. The IMA is assessed for possible reimplantation; however, assuming adequate collateral flow, it is usually ligated. Typically, a tube graft is placed, but a bifurcated graft may be required if iliac artery aneurysms or external iliac artery occlusive disease are present. When extensive external iliac occlusive disease is present, distal graft limbs may be tunneled to the groin for femoral anastomotic. Once the graft is sutured in place, flow is restored first to the internal, then to the external iliac arteries to divert emboli from the lower extremities. The aneurysm sac is closed over the graft to help prevent aortoenteric fistula formation. Visual inspection of the left colon should be performed to evaluate for ischemia. Distal flow is assessed by palpating the femoral and distal pulses and abdominal closure is performed.

Postoperative care includes ICU monitoring and cardiac afterload reduction to reduce the risk of bleeding and myocardial ischemia. Beta blockade should be considered to reduce postoperative cardiac morbidity. Potential postoperative morbidity of open AAA repair includes myocardial infarction (3–5%), stroke, renal failure, limb ischemia, colonic ischemia (2–6%), pulmonary insufficiency, infection, and spinal cord ischemia. Colonic ischemia usually presents within 3 days with blood-tinged loose stools, left lower quadrant pain, fever, and tachycardia. These findings should trigger evaluation with flexible sigmoidoscopy followed by exploratory laparotomy if transmural ischemia is suspected. Perioperative mortality with elective open AAA repair is 5%, two-thirds of which is attributable to cardiac disease.

Endovascular stent-graft repair avoids an abdominal incision and its associated morbidities. This repair involves endovascular graft placement into the infrarenal aorta via the femoral arteries. The repair can be performed either with bilateral femoral cut downs or percutaneously. Favorable vascular anatomy for endovascular repair is summarized in Table 24.2. The long-term benefits of endovascular repair in patients considered high risk for open AAA repair are questionable. Reduction in perioperative morbidity and potential for discharge from the hospital on the day following surgery have helped this technique to gain popularity. Complications of endovascular repair include groin wound complications, intravenous contrast-induced nephropathy, and endoleaks. Endoleaks are the presence of persistent blood flow within the aneurysm sac. Four main types of endoleaks have been described. Type I consists of leaks into the sac via the proximal or distal sealing zone of the graft. Type II endoleak is defined as a leak into the sac via AAA branches such as the lumbar arteries or IMA. Type III endoleaks originate from junctions between graft components or tears in the graft wall. Type IV endoleaks originate from graft wall fabric porosity or suture holes. In addition to these four types, an entity referred to as endotension consists of high AAA sac pressure without a demonstrable endoleak. Approximately 70% of

<table>
<thead>
<tr>
<th>Cross-sectional AAA diameter (cm)</th>
<th>Rupture risk (% per year)</th>
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<tbody>
<tr>
<td>&lt;4</td>
<td>&lt;1</td>
</tr>
<tr>
<td>4–5</td>
<td>1–3</td>
</tr>
<tr>
<td>5–7</td>
<td>6–10</td>
</tr>
<tr>
<td>&gt;7</td>
<td>&gt;20</td>
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</table>

**Table 24.1. Risk of AAA Rupture.**

**Table 24.2. Favorable vascular anatomy for endovascular stenting.**

1. A normal infrarenal segment of aorta at least 15 mm in length and no more than 30 mm in diameter
2. Minimal iliac artery inner diameter of 7 mm
3. Angulation of proximal aortic neck of less than 60°
4. Angulation of iliac arteries of less than 90°
all endoleaks resolve spontaneously within a month of graft placement. Type II leaks are thought to be the most benign; however, even these are repaired in cases where the AAA continues to increase in size on follow-up. Endovascular repair requires lifelong follow-up with CT scanning, plain radiographs, and/or ultrasound. Approximately 30% of endovascular stent-grafted AAAs will require a secondary procedure at some point in time.
Abdominal Aortic Aneurysm

D. Symptomatic AAA
1. 50% die before making it to hospital
2. Large bore I.V.
3. Type & Cross
4. O.R. without delay

CAT scan to rule out leak
O.R. see AAA Surgical Repair

A. AAA Risk Factors
1. Advanced Age
2. Male Gender
3. Tobacco use
4. Emphysema
5. Atherosclerosis
6. HTN

Nonspecific Complaints

(-) leak
O.R. see AAA Surgical Repair

B. Asymptomatic AAA
1. 80% discovered incidentally
2. Ultrasound, CAT scan or MRI
3. <4cm follow by ultrasound or CAT scan

4-5cm
Consider Elective Repair

(-) leak
O.R. see AAA Surgical Repair

4-5cm
Consider Elective Repair

Young low risk
Rule out associated aneurysms
HTN control
Smoking cessation
Ultrasound 3-6mos

Expansion >0.4cm/6mos

>5cm
C. Cardiac work-up

CABG/PTAC

E. AAA Surgical Repair
1. Laparotomy
2. Endovascular stent

Minimal CAD

Significant CAD

*Angiogram is performed preoperatively if endovascular repair is considered
1. To evaluate renovascular HTN
2. To assess IMA patency
3. To assess iliac disease/distal runoff

*Angiogram is performed preoperatively if endovascular repair is considered
1. To evaluate renovascular HTN
2. To assess IMA patency
3. To assess iliac disease/distal runoff

*Angiogram is performed preoperatively if endovascular repair is considered
1. To evaluate renovascular HTN
2. To assess IMA patency
3. To assess iliac disease/distal runoff
Part IV
Endocrine Disorders
In iodine-deficient countries, goiter is endemic and the incidence of thyroid nodules is high. In the United States where table salt is iodinated, thyroid nodules are clinically detectable in only 4–7% of the general population. An aging population and incidental findings from usage of unrelated radiographic studies have however resulted in a higher prevalence of this endocrine entity. High-resolution ultrasonography, which is being used with increasing frequency to image the neck for a variety of disorders, detects thyroid nodules in 16–67% of unselected patients. In autopsy series, 30–60% of thyroids harbor nodules. Many of these micronodules are less than 1 cm in diameter and are usually not clinically relevant in asymptomatic patients.

A. Differential Diagnosis. The vast majority of thyroid nodules are benign. They represent an array of nonfunctional and functional lesions that challenge the physician to differentiate benign from malignant nodules. Often these represent various stages of nodule formation and degeneration within a nodular thyroid gland. Commonly seen entities include colloid lesions, degenerative cysts, and follicular adenomas. Thyroid nodules are frequently due to glandular hyperplasia resulting from spontaneous or compensatory growth of the thyroid gland. Lymphocytic infiltration, as well as granuloma formation, may be the sequelae of inflammatory processes such as Hashimoto’s and subacute thyroiditis. The sudden, rapid, painful growth of a nodule may indicate malignancy, but the likely cause is hemorrhage into a preexisting colloid nodule.

Thyroid cancers usually grow slowly, constitute 5% of palpable thyroid nodules, and are responsible for 0.5% cancer-related deaths annually. Risk factors for thyroid carcinoma include the extremes of age (young and old patients), male gender, a family history of thyroid or other endocrine tumors, and a history of radiation exposure.

The most common thyroid malignancy is papillary cancer, which has the best prognosis of all thyroid tumors. This type of neoplasm is typically multifocal, not well encapsulated, and has a tendency to spread in a loco-regional fashion. Follicular cancers in contrast are often solitary, encapsulated, and grow via vascular invasion. Epidemiological studies indicate that the incidence of papillary cancer is increased in iodine-rich regions, whereas follicular cancer occurs more commonly in iodine-deficient regions of the world.

B. History and Physical Exam. Most patients with thyroid nodules are asymptomatic. Those who present with dypsnea, dysphonia, or dysphagia may have extrinsic compression either from a goitrous thyroid or from loco-regional invasion of a malignant lesion to the trachea, recurrent laryngeal nerve, and esophagus, respectively.

The thyroid gland is best examined by standing behind the patient with the neck slightly extended. Having the patient swallow will help differentiate thyroid nodules from other neck masses, as the thyroid gland moves up and down during swallowing. The size, texture, and consistency of the thyroid gland are assessed during the examination. The clinician should attempt to differentiate whether a thyroid nodule is solitary or part of a multinodular gland. The presence of cervical lymphadenopathy is also noted. Vocal cord mobility is routinely assessed by indirect laryngoscopy on all patients referred for surgery since a nonmobile vocal cord can be a heralding sign of a locally invasive thyroid cancer.

C. Evaluation of Thyroid Nodules. For the euthyroid patient having a solitary thyroid nodule, fine needle aspiration (FNA) has emerged as the procedure of choice. With good technique in experienced hands, this cytologic evaluation is highly accurate with very low false-positive and false-negative rates. With the patient in the supine position and the neck slightly extended, the thyroid nodule is palpated and aspirated with a 23- or 25-gauge needle. Typically, several passes are made with the needle to increase the yield of thyroid cells for cytologic evaluation. Cytologic interpretation is classified into four groups: inadequate, benign, suspicious, or malignant. The main limitation of this technique is the differentiation of follicular and Hurthle adenomas from carcinomas due to inability to assess capsular and vascular invasion. One in every four “suspicious” lesions may harbor malignant tumor and, for this reason, surgical resection is justified.
The FNA sample must contain an adequate number of follicular cells for an accurate interpretation. Having an experienced cytopathologist assess the specimen is essential. If the initial FNA produced an inadequate sample, another attempt at FNA is warranted and will likely yield the diagnosis. Patients with benign cytology can be followed. If FNA is indeterminate or suspicious, thyroidectomy is indicated. Alternatively, one could obtain a thyroid scan in this instance; if the nodule demonstrates increased uptake, suppression may be chosen. If the nodule does not regress with suppression, surgery should be undertaken. Surgery is also indicated for patients with malignant FNA and for patients who have a history of radiation exposure regardless of the FNA result.

Thyroid ultrasonography plays an important role in the management of thyroid nodules. Ultrasound is an extension of the physical examination, allowing a thorough, cost-effective survey of the neck, including the thyroid gland. The precise size and location of thyroid nodules are easily determined as well as the presence of additional thyroid pathology (i.e., nonpalpable nodules, Hashimoto’s thyroiditis). Although ultrasound cannot definitively differentiate benign from malignant nodules, certain sonographic features raise the index of suspicion of malignancy including the visualization of punctate calcifications, irregular borders, absence of a hilar echo, and presence of enlarged cervical lymph nodes.

Thyroid scintigraphy may yield useful information in equivocal circumstances. The ability to concentrate radioactive iodine isotopes on these studies demonstrates whether the lesion is nonfunctioning (“cold”) or functioning (“hot”). About 20% of cold nodules with solid components harbor malignancy. Cystic and hot nodules are usually benign.

D. Treatment of Thyroid Cancer. Consensus on the exact surgical management for papillary neoplasms has not been reached by experts in this field. Some surgeons perform only a lobectomy (vs. total thyroidectomy) for a papillary lesion under 4.0 cm as survival is not altered. Others feel quite strongly that the minimal operation of choice is total thyroidectomy as it facilitates postoperative follow up with thyroglobulin and radioiodine ablation. Near total thyroidectomy preserves adjacent vital structures such as the recurrent laryngeal nerves and parathyroid glands. The treatment of follicular neoplasms is equally controversial. Lesions less than 2.0 cm are considered “low risk,” and if histology did not reveal transcapsular or vascular invasion, some surgeons will perform a lobectomy only. Since up to 40% of follicular cancers identified by FNA are shown to be follicular variants of papillary cancers on final pathology, there are some surgeons who advocate a total thyroidectomy for lesions greater than 2 cm. Thorough examination of the central and lateral compartments of the neck and removal of any enlarged lymph nodes are part of the intraoperative metastatic evaluation.

Anaplastic cancers are poorly differentiated, fast growing, and highly fatal, with an average life expectancy of 4–6 months after diagnosis. These tumors usually affect older patients. Some studies suggest that these tumors represent a transformation of longstanding, untreated papillary and follicular carcinomas. If complete resection is not possible, surgery is directed toward palliation (i.e., tracheostomy).

Medullary cancers are unique in that they are tumors of the laterally located parafollicular cells and secrete calcitonin. These can be sporadic or familial with associated endocrinopathies, as is seen in multiple endocrine neoplasia II syndrome. Patient and family members should be screened for the RET protooncogene to identify this autosomal dominant disease. Surgical management should include total thyroidectomy, central neck dissection, and unilateral modified radical neck dissection.
A. Manifestations. Cushing’s syndrome is a collection of diseases of chronic glucocorticoid excess. Signs and symptoms include truncal obesity (95%), facial fullness (80%), diabetes (80%), amenorrhea (75%), hypertension (70%), proximal myopathy (65%), skin atrophy and bruising (60%), and osteoporosis (40%).

B. Causes. The etiologies of Cushing’s syndrome include exogenous glucocorticoid administration (most common) and endogenous hypercortisolism. Causes of the latter include Cushing’s disease (microadenoma of the pituitary) (70%), ectopic adrenocorticotropic hormone (ACTH)-secreting tumor (12%), adrenal adenoma and hyperplasia (10%), and adrenal carcinoma (8%). Ectopic ACTH-secreting tumors are usually located in the lung and may be malignant or benign. Other possible ectopic sources include pancreatic or thymic cancers, medullary thyroid cancer, or other neuroendocrine tumors.

C. Diagnosis. The initial step in evaluating a patient with suspected hypercortisolism is to collect a 24-h urine sample for free cortisol. If an overnight 1 mg dexamethasone suppression test reveals an 8 AM cortisol > 5µg/dl, it should be confirmed with a 24-h urinary-free cortisol. The vast majority of patients with Cushing’s syndrome will have a 24-h urinary-free cortisol greater than three times the upper range of normal. Caution must be taken to clinically recognize the two common causes of “pseudo-cushing’s,” namely, depression and alcoholism.

D. Adrenal versus Nonadrenal Source. The next step is to measure ACTH by immunoradiometric assay. If the value is less than the lower range of normal (<5 pg/ml) then an adrenal source is confirmed and an adrenal CT should be performed to determine unilateral versus bilateral disease. Surgical therapies may include unilateral or bilateral adrenalectomy. Perioperative and postoperative glucocorticoids should be administered. Medical therapies will include ketoconazole or mitotane.

E. Localizing Nonadrenal Sources. In Cushing’s disease, ACTH levels may range from normal (5–50 pg/ml) to modest elevations (50–150 pg/ml). The highest plasma levels of ACTH have been observed in patients with ectopic ACTH sources and may be greater than 1,000 pg/ml. Patients with small cell lung cancer may present acutely with extreme hypercortisolism, hypokalemia, hypertension, and weakness. Patients with benign bronchial carcinoids have a clinical picture which is indistinguishable from that of patients with pituitary Cushing’s disease; both have a chronic presentation and normal or modestly elevated ACTH levels. In order to delineate pituitary from nonpituitary sources of ACTH, the first step is a dynamic enhanced MRI scan of the pituitary. Most patients with pituitary Cushing’s will have a microadenoma and may be treated with transsphenoidal surgery.

If the dynamic MRI scan of the pituitary is negative, the excess ACTH may still be secreted from a pituitary microadenoma or from an extra-pituitary source usually in the lung. At this juncture it is necessary to perform bilateral inferior petrosal sinus sampling (IPSS) of ACTH before and after corticotrophin-releasing hormone (CRH) stimulation. A central/peripheral ACTH ratio of greater than 2 basal or greater than 3 stimulated is diagnostic of a pituitary source. Such patients are referred for transsphenoidal surgery. If there is a left/right IPSS ratio greater than 2, this will correlate 80% of the time with correct lateralization of the microadenoma at the time of surgery.

Patients who do not have a central/peripheral ACTH ratio of greater than 2–3 may have a false-negative result due to the technical vagaries of IPSS. However, a ratio less than 2 usually points to a pulmonary tumor and a thin section spiral CT of the chest with IV contrast is the next step in localization. If this is negative then an enhanced MRI scan, octreotide nuclear scan, and PET scan have all been shown to have important utility. Additionally, octreotide scan, if positive, yields a functional assessment as somatostatin receptors are often present on bronchial carcinoids and may predict a clinical response to octreotide therapy. All patients in whom a pulmonary target can be localized are referred for surgical resection. Second-line therapies include octreotide, ketoconazole, or bilateral adrenalectomy.

It must be kept in mind that 2–10% of all patients may harbor nonfunctioning pituitary or adrenal “incidentalomas”
identified on CT or MRI. Thus it is critical that the diagnosis and localization of a surgical target in Cushing’s syndrome be established on the basis of hormonal testing as outlined above. Patients in whom uncertainty exists should be studied further with CRH stimulation of ACTH in peripheral blood and/or iodocholesterol adrenal scanning. Finally, low-dose and high-dose dexamethasone suppression tests are only about 80% accurate and therefore are no longer recommended.
Cushing's Syndrome

A. Manifestations
   - truncal obesity (95%)
   - facial fullness (80%)
   - diabetes (80%)
   - amenorrhea (75%)
   - hypertension (70%)

B. Causes
   - exogenous glucocorticoids
   - pituitary adenoma
   - ectopic ACTH tumor
   - adrenal hyperplasia
   - adrenal adenoma
   - adrenal cancer

C. Diagnosis
   - 24 hour urine for free cortisol
   - normal elevated
     - stop

D. Adrenal vs. Nonadrenal source
   - ACTH level
     - low ACTH
       - adrenal source
         - adrenal CT
     - high ACTH
       - Nonadrenal source
         - pituitary Cushing's
         - ectopic ACTH
         - small cell lung Cancer
         - benign bronchial tumor

E. Localizing
   - dynamic MRI pituitary
     - transsphenoidal surgery
     - PET scan
     - octreotide scan
     - Look for pulmonary tumor
     - Petrosal Sinus Sampling
     - measure central: peripheral ratio of ACHH
     - (+) pituitary source
Hyperthyroidism

Subhash Patel

A. History. Hyperthyroidism is a state of health caused by increased levels of serum thyroxine (T₄), or triiodothyronine (T₃) or both. Clinical symptoms and signs include weight loss, intolerance to heat, insomnia, loss of hair, flushing, anxiety, tremors, chest pain, palpitation, increased appetite and gastrointestinal motility, amenorrhea, and gynecomastia in males. Metabolic abnormalities include an increase in basal metabolic rate (50%–100%), glycogenolysis, gluconeogenesis, protein catabolism, lipolysis, and free fatty acids. A thyroid storm is a severe state of hyperthyroidism characterized by a rapid increase in temperature, cardiac arrhythmias, circulatory shock, multiorgan failure, and it carries a mortality rate of 10%. It is usually precipitated by stressful conditions such as surgery, infection, and child birth.

B. Physical Examination. Physical signs include increased resting heart rate, wide pulse pressure, palmar erythema, onycholysis, warm moist skin, and hyperreflexia. The gland is diffusely enlarged, nodular, or not palpable at all.

Diffuse enlargement: Graves’ disease (Basedows disease) is the most common cause of hyperthyroidism associated with diffuse thyroid enlargement (80% of all reported cases in the USA). In this disease, thyroid-stimulating antibody (TSI) binds with the thyroid-stimulating hormone (TSH) receptor site on the thyroid gland eliciting a response similar to TSH. A specific gene has not been identified; however, Graves’ disease may be found in siblings of the affected family. The peak incidence of Graves’ disease is in the fourth decade. Female to male ratio is 4:1 to 5:1. Patients with Graves’ disease may have ophthalmopathy characterized by proptosis, lid lag, paradoxical dilatation of pupil to light (Marcus Gunn sign), corneal ulcers, and varying degree of visual defects. Nearly 50% of patients will have clinical evidence and 90% will have radiographic evidence of ophthalmopathy. Other physical signs that may be present include pretibial myxedema, dermopathy, clubbing, and acropachy.

Other causes of diffuse thyroid enlargement include Hashimoto’s thyroiditis (painless), subacute thyroiditis (painful), pituitary tumor, and HCG-mediated hyperthyroidism (molar pregnancy, choriocarcinoma, hyperemesis gravidarum). The effects of HCG on the thyroid gland are similar to those of TSH.

Nodular Enlargement. Multinodular toxic goiter is the second most common cause of hyperthyroidism. Autonomous production of thyroid hormone develops in patients with long-standing multinodular goiter after usually more than 10 years. Underlying mechanisms are not clearly understood; however, IGF and epidermal growth factors have been implicated. Ingestion of iodine or the use of iodine-containing compounds (amiodarone, contrast material) may precipitate hyperthyroidism in patients with preexisting multinodular goiter (Jod Basedows disease). Nodular goiters may cause aerodigestive compression. On physical examination, the thyroid gland is not tender and has multiple nodules of varying size.

Plummer’s disease is hyperthyroidism associated with a solitary nodule (toxic adenoma). A somatic mutation at the TSH receptor site in the nodule causes autonomous hormone overproduction, while the remaining gland becomes atrophic and inactive. Other causes of nodular goiter include thyroid cancer and nodular Graves’ disease. Less than 2% of patients with thyroid cancer will have a hyperfunctioning enlargement of the gland. Cold nodules in Graves’ disease should be evaluated for cancer.

Nonpalpable Thyroid. Not all patients with Graves’ disease have thyroid enlargement. Hyperthyroidism in the absence of palpable gland may be due to exogenous administration of thyroid hormone (factitious hyperthyroidism), or ectopic secretion of thyroid hormone (Struma ovarii, metastatic thyroid carcinoma)

C. Diagnosis. The diagnosis of hyperthyroidism is confirmed by suppression of thyrotropin (TSH <0.03) associated with an increase in serum T₄ and/or T₃. The only exception is the rare patient with a TSH-secreting pituitary tumor, where T₄, T₃, and TSH are all elevated. T₄ has a greater affinity for binding with protein (2–6 times more than T₃). Only 0.024% of T₄ and 0.36% of T₃ are free. It is the free hormone which is responsible for its biological activity. Normal T₄ and T₃ levels with low serum TSH is often seen in elderly patients and is referred as subclinical thyrotoxicosis. Decreased binding
due to low protein accounts for this laboratory anomaly. In these patients, free $T_4$ (free thyroxin index; FTI) is measured to make the diagnosis. Following successful treatment of thyrotoxicosis, TSH remains suppressed for a variable period of time and should not be confused with subclinical thyrotoxicosis. In patients with $T_3$-thyrotoxicosis (<5%), $T_3$ is elevated but $T_4$ remains normal. Because of the rarity of this event, free $T_3$ is not measured normally.

For the patient with classical signs and symptoms, no additional diagnostic tools are necessary. For patients with suspected iatrogenic or factitious hyperthyroidism, a low serum thyroglobulin level confirms the diagnosis. For patients with suspected molar pregnancy, choriocarcinoma, or transient hyperthyroidism of pregnancy, serum HCG is elevated. In doubtful cases, measurement of TSI (thyroid-stimulating immunoglobulin) can confirm the diagnosis of Graves’ disease. It is especially useful during the last few weeks of pregnancy where high levels of TSI in maternal blood are associated with an increased incidence of transient hyperthyroidism in the newborns.

Nuclear medicine scanning with technetium-99m ($^{99m}$Tc) can distinguish hyperfunctioning from nonfunctioning areas. In the former cases, there is increased uptake, either diffusely as in Graves’ disease or in an isolated area such as a toxic adenoma. Decreased uptake can be seen in thyroiditis (diagnosis confirmed by presence of antithyroid antibody) and cancer where FNA may establish the diagnosis. Extra-thyroidal uptake is confirmed by presence of antithyroid antibody) and cancer.

D. Treatment. The three major treatment options include pharmacotherapy, radio iodine ablation, and surgical resection. Pharmacotherapy is the preferred treatment for Graves’ disease in pregnancy and in children, and as the initial form of therapy for other causes of hyperthyroidism. Beta blockade has become an important adjuvant; even though it does not reduce release of thyroid hormone, it provides symptomatic relief by blocking target organ response to increased catecholamines. Alleviation of symptoms like tachycardia, palpitation, sweating, nervousness, and tremors makes it tolerable for the patient to await definitive therapy. For individuals with transient hyperthyroidism of pregnancy, it may be the only drug that may be needed.

Antithyroid drugs [propylthiouracil (PTU) and methimazole] block oxidation of iodine and its conjugation with tyrosine and are effective in most patients. PTU is given at an initial dose of 100 mg every 8 h and is the preferred drug during pregnancy and thyroid storm. Methimazole is given at an initial dose of 20–40 mg once a day and is preferred by most patients because of its once a day dose, fewer side effects, and lower cost. Side effects include allergic reaction, arthralgias, hepatitis, and agranulocytosis (noted in less than 1% of the patients). Adverse reactions tend to occur in elderly patients and after prolonged use. In pregnant women the dose is reduced (after 2–6 weeks of therapy) to maintain high normal levels of $T_4$ and is eventually tapered off by 34–36 weeks. In most other patients, the duration of therapy may be as long as 12–16 months. Recurrence rate is common even after 1–2 years of therapy. There is increasing evidence that addition of thyroxine not only avoids hypothyroidism but also reduces recurrence following antithyroid drugs.

Iodide is often used prior to surgical resection and in patients with thyroid storm. Potassium iodide, 5 drops three times a day, acutely inhibits the release of the thyroid hormone from the gland. It also reduces the vascularity of the gland. In patients with thyroid storm, in addition to antithyroid drugs (PTU), beta blockers, and iodide, dexamethasone 0.5 mg is given every 6 h. It reduces the conversion of $T_4$ to $T_3$. Volume replacement and symptomatic treatment of hyperthermia are other measures undertaken for this form of thyrotoxicosis. Hyperthyroid state with thyroiditis is usually transient and is managed with beta blockers, steroids, and analgesics.

Ablative therapy with radioactive iodine is emerging as the treatment of choice for most patients with Graves’ disease, especially in elderly patients. It should not be used in pregnant patients. Success rates range from 70% to 100%; if an initial dose of 5–10 mCi of $^{131}$I is not effective after 4–12 weeks, it should be repeated. If patients have been treated with antithyroid drugs, higher doses of $^{131}$I may be needed. Lithium carbonate can be used to improve the effectiveness of radioactive iodine therapy. Lithium enhances iodine uptake and retention in the thyroid gland and reduces its release. Pretreatment with 1600 mg of lithium one week prior to therapy may lower the failure rate following radiotherapy. Patients with nodular goiter respond poorly to radiotherapy. Potential side effects include thyroiditis and thyroid cancer (controversial).

Even though surgical ablation is the most effective therapy, it is often considered the least preferred treatment option because of potential complications and the possibility of precipitating thyroid storm. Major complications reported include injury to the laryngeal nerves, injury to parathyroid glands, bleeding, and hypothyroidism. Experienced endocrine surgeons have very low morbidity rates and surgical resection is becoming the treatment of choice for nodular goiter. The use of preoperative beta blockade, iodine, and a short course of antithyroid drugs has dramatically reduced the risk of thyroid storm in the perioperative period. The extent of thyroid resection is dependent upon the underlying disease. For toxic adenoma, a lobectomy will be sufficient; however, total thyroidectomy is preferred for most other conditions. Some surgeons have used near total or subtotal thyroidectomy to reduce the incidence of surgical complications and permanent hypothyroidism. Following resection of the thyroid gland patients are placed on replacement hormone therapy for the rest of their life.
Hyperthyroidism

A. History
weight loss, heat intolerance, insomnia, hair loss, flushing, anxiety, tremors, etc.

C. Diagnosis
TSH suppressed to < 0.03
increased T3, T4
exceptions:
1. if thyroglobulin decreased, consider factitious or iatrogenic cause
2. if thyroglobulin and TSH increased, consider pituitary tumor

Technetium-99m scans
increased uptake
Grave's disease
multinodular goiter
molar pregnancy
choriocarcinoma
toxic adenoma
decreased uptake
cancer
thyroiditis
solitary cold nodule
cancer
nodular Grave's multinodular goiter

D. Treatment

Grave's disease, pregnant
beta blocker, PTU for 2-6 weeks, stop by week 34-36

Grave's, not pregnant
beta blocker, radiotherapy is the preferred treatment, alternatives are available

Thyroiditis
beta blocker, steroids, wait for disease to run its course

Toxic nodule, cancer, goiter
beta blocker, antithyroid drugs then surgery

Thyroid storm
IV fluids and beta blocker, IV steroids, iodide, PTU, support

Molar pregnancy, choriocarcinoma
beta blocker, antithyroid drugs, treat underlying disease

B. Physical Examination
diffuse enlargement-Grave's nodular enlargement non-palpable gland
Calcium (Ca) homeostasis is maintained by the action of two hormones on three organ systems. The majority of Ca is stored in bone as hydroxyapatite, a compound that helps maintain structural integrity. Although the intravascular concentration of Ca is small, an imbalance can produce life-threatening symptoms. In plasma, 50% of Ca is unbound or ionized, whereas the remainder is bound to albumin. A change of 1 gm/dl in serum albumin is associated with a 0.8 mg/dl change in total serum calcium. Serum phosphate levels vary inversely with calcium, maintaining a normal concentration product between 30 and 40.

Parathyroid hormone (PTH) is an 84 amino acid polypeptide secreted by the chief cells of the parathyroid glands in response to a decrease in the plasma concentration of ionized Ca. PTH elicits three responses. First, it stimulates bone resorption, which leads to release of Ca into the blood. In the kidneys, PTH facilitates the conversion of 25-hydroxycholecalciferol (calcifediol), which is produced in the liver, to 1,25-dihydroxycholecalciferol (calcitriol), the active form of vitamin D. Finally, PTH enhances the absorption of Ca and subsequent excretion of phosphate in the distal tubules and collecting system of the kidney.

Vitamin D is a fat-soluble vitamin that is found in all living animals. In the presence of ultraviolet light, 7-dehydrocholesterol, which is located in the skin, is converted to vitamin D3 (cholecalciferol). Vitamin D3 then undergoes hydroxylation in the liver to form 25-hydroxycholecalciferol, the precursor of calcitriol. The endogenous production of calcitriol requires PTH to facilitate the final conversion in the kidney. Calcitriol increases absorption of Ca in the intestine, stimulates resorption of bone in conjunction with PTH, and decreases excretion of Ca in the kidney.

A. Presentation. Hypercalcemia is usually detected during chemical screening for unrelated reasons. Patients usually do not have symptoms until serum calcium exceeds 11 mg/dl. Symptoms are variable and can involve multiple organ systems (Table 28.1).

If untreated, hyperparathyroidism (HPT) may lead to premature death from cardiovascular disease; there is an increased frequency of hypertrophic cardiomyopathy even without hypertension. These patients have an increased incidence of renal calculi, ulcer disease, renal failure, hypertension, osteopenia, gout, and pancreatitis. Following parathyroidectomy, older patients still have an increased risk of premature death. Treatment is warranted even if the patient is asymptomatic.

B. Differential Diagnosis. HPT is the most common cause of hypercalcemia. Cancer is the next most common cause either by the secretion of a PTH-like substance or by its bony metastases. Tumors associated with hypercalcemia include multiple myeloma, lymphoma, leukemia, and tumors of the breast, lung, or kidney. Other causes of hypercalcemia include sarcoidosis, tuberculosis, fungal infections, excessive use of thiazide diuretics, and excessive intake of calcium and/or vitamin D.

C. Diagnostic Studies. Serum calcium, PTH, and phosphate levels should be obtained. High calcium and low normal phosphate levels are seen in HPT. The PTH level will be low in all cases other than those due to primary or ectopic HPT. If tumors are causing a paraneoplastic syndrome, a PTH-related peptide can be identified by the PTH-RP serum test. The use of imaging studies is dictated by the specific diagnosis being considered; these are discussed below.

D. Hyperparathyroidism. HPT is a disorder that is characterized by an excess production of PTH and a normal to high plasma Ca concentration. The most common cause of this disease is a single parathyroid adenoma (80–85%), although 10–20% of patients will have multigland disease. Carcinoma of the parathyroid gland is responsible in 1% of cases. The peak incidence of HPT is in the sixth decade of life, with women being affected twice as often as men. Parathyroidectomy is the only known cure for HPT.

High-resolution ultrasound and/or technetium 99-sestamibi scintigraphy play an important role in determining the operative approach. If a solitary adenoma is visualized on preoperative imaging, a focused neck exploration is performed (unilateral neck exploration under local anesthesia, video-assisted or endoscopic parathyroidectomy). Intraoperative PTH monitoring confirms excision of the hypersecreting...
gland, as demonstrated by a greater than 50% reduction in the highest baseline value following parathyroidectomy. If there is an inappropriate reduction of PTH or if the levels remain high, bilateral neck exploration is performed. The advantages of a targeted approach include lower incidence of postoperative hypocalcemia, use of local anesthesia, same day surgery, improved cosmetic results, and decreased duration of surgery. If preoperative localization is negative, conventional bilateral neck exploration is performed whereby all 4 glands are identified and frozen-section biopsy of one gland is performed. If it is hyperplastic, 3-1/2 glands are removed. If only 3 normal glands are found, a thyroid lobectomy is performed on the side with one gland.

E. Malignancy. Hypercalcemia of malignancy is usually indicative of advanced disease. These patients are typically symptomatic and require rapid diagnosis and definitive treatment. Both osteolytic processes and PTH-related factor are responsible for the hypercalcemia of malignancy. Hematologic malignancies affecting bone marrow and breast cancer are known to cause hypercalcemia through the local invasion of bone via local factors produced by the tumor cells that stimulate osteoclasts to reabsorb bone. PTH-related factor is associated with significant hypercalcemia but the low intact PTH tumors that produce this humoral response include squamous cell cancer of the lung, head and neck, renal, bladder, and cervix/vulva cancers.

F. Hypercalcemic Crisis. Hypercalcemic crisis (HCC) is a life-threatening condition that warrants immediate diagnosis and treatment. Causes include dehydration, HPT, vomiting, diuretics, immobilization, parathyroid carcinoma, and underlying malignancy. A serum Ca level greater than 14 mg/dl is often considered a medical emergency, even in the mildly symptomatic patient. Symptoms may include severe dehydration, mental status changes, and cardiac arrhythmias.

G. Medical Treatment of Hypercalcemia. Treatment of hypercalcemia is usually based on symptoms, although some physicians will treat an asymptomatic patient with Ca levels greater than 12 mg/dl. The first line of treatment in these patients is vigorous hydration with normal saline solution (2–6 liter/day). The hypercalcicuric state inhibits the resorption of Na in the kidney, exacerbating the dehydration and hypercalcemia. In addition, hypercalcemia may become symptomatic in the setting of dehydration or itself cause dehydration by inducing diarrhea and vomiting, thus leading to a decrease in Na and Ca excretion.

Several medications play a role in the treatment of hypercalcemia. The first line is the loop diuretics (furosemide), which block the resorption of Ca in the kidney. This medication should be used with caution, for its use may be counterproductive if given before the patient is adequately rehydrated. Diuretics may be particularly efficacious in patients at high risk for developing heart failure with large volume fluid resuscitation. Bisphosphonates, which inhibit osteoclasts and resorption of bone, are an important treatment modality. There are several classes of bisphosphonates with varying side effects. Pamidronate has been shown to decrease serum Ca to near normal levels within 2–3 days when given in a dose of 60–90 mg over 4–24 h. Other bisphosphonates like etidronate are also effective but may induce the complication of bone demineralization. This class of medication may normalize the serum Ca for weeks to months. Calcitonin (0.5 mg SC q; 6–12 h) has an immediate effect by limiting osteoclast activity. Although its initial effects are seen within minutes, sustained results are not seen because of tachyphylaxis. In addition, calcitonin has not been shown to decrease Ca by more than 2–3 mg/dl. Its use may be warranted in an emergent situation or when awaiting the effects of more effective and longer lasting medications. Glucocorticoids may be effective in hypercalcemia associated with hematologic and breast malignancies by inhibiting the cytokine effects of the tumor on bone. In addition, they also have a role in treatment by increasing Ca excretion and decreasing intestinal absorption in sarcoidosis and vitamin D intoxication. The usual dose is 40–100 mg of prednisone/day in divided doses for up to 5 days. In severe, refractory cases, or in renal failure, hemodialysis is a viable treatment modality. Phosphate levels are also decreased during dialysis and should be monitored closely. Finally, if the diagnosis is HPT, emergent parathyroidectomy for cure may be considered once adequate hydration and initial correction of life-threatening serum Ca levels have been achieved.
Hypercalcemia

A. Presentation
usually found on chemical screening
asymptomatic until calcium 11 mg/dl

B. Differential Diagnosis
hyperparathyroidism most common
cancer, sarcoidosis, tuberculosis, thiazide
diuretics, calcium or vit D excess

C. Diagnostic Studies
serum Ca, serum PTH, 24 hr urinary Ca

D. Hyperparathyroidism
localization studies
solitary adenoma - targeted
parathyroidectomy
(endoscopic, video-assisted, unilateral)
multigland disease - bilateral neck
exploration, biopsy, if hyperplastic then
remove 3-1/2 glands

E. Malignancy
ectopic PTH
osteolytic lesions
surgery
vs.

F. Hypercalcemic Crisis

G. Medical Treatment
saline hydration
loop diuretics
bisphosphonates
calcitonin
steroids
hemodialysis
emergency parathyroidectomy
A. Clinical Presentation. In patients with suspected hypoglycemia, low plasma glucose concentrations must be documented at the time of symptoms, as symptoms alone may be nonspecific. Symptoms can be divided into either adrenergic (anxiety, sweating, palpitations) or neuroglycopenic (headache, dizziness, confusion), the latter being more specific for true organic hypoglycemia. Hypoglycemic symptoms tend to occur with plasma glucose concentrations at or below 50 mg/dl with CNS disturbances becoming apparent below 45 mg/dl. Clinical hypoglycemic disorders have been divided into two broad categories: those occurring in seemingly healthy patients and those occurring in patients who are obviously ill. The diagnosis of endogenous hyperinsulinism is dependent on the satisfaction of Whipple’s triad. The triad includes neuroglycopenia, documented hypoglycemia (plasma glucose levels < 50 mg/dl), and symptoms relieved (often within 5–10 min) with the administration of glucose. Patients who satisfy this triad warrant further evaluation.

The causes of hypoglycemia in seemingly healthy patients include insulinoma, noninsulinoma pancreatogenous hypoglycemia syndrome (NIPHS), insulin factitial hypoglycemia, drugs, severe exercise, and ketotic hypoglycemia. Insulinomas make up the large majority of these cases.

The incidence of insulinomas is ~4 per one-million persons per year. Well over 90% are benign and solitary, and most are less than 2 cm in diameter. Women outnumber men ~3:2. They are equally distributed throughout the pancreas. Ten percent occur within the context of the multiple endocrine neoplasia type I (MEN I) syndrome, and in this setting may be multiple, in association with parathyroid hyperplasia, and occasionally pituitary neoplasia (i.e., prolactinomas).

B. Diagnosis. The 72-h fast, during which the patient drinks calorie and caffeine-free beverages until the plasma glucose reaches <45 mg/dl and the patient has signs or symptoms of hypoglycemia, forms the basis for the diagnosis of an insulinoma. A witnessed neuroglycopenic event with documented plasma glycopenia can obviate the need for a formal supervised fast, if all the appropriate end of fast (EOF) studies are obtained (Table 29.1). A positive supervised fast is virtually pathognomonic of an insulinoma.

C. Localization. Once the diagnosis is secure, reasonable attempts at preoperative localization should be made to facilitate operative planning and allay patient and surgeon anxiety. Options include selective arterial calcium stimulation (>90% sensitive), selective angiography (50–60%), endoscopic ultrasonography (>90%), somatostatin receptor scintigraphy (60%), helical CT (60–80%), intraoperative ultrasound (90%), transabdominal ultrasound (65%), and palpation and intraoperative ultrasound (98%). We generally obtain transabdominal ultrasonography by a radiologist experienced in recognizing insulinomas, as well as a triple-phase spiral CT. One or both of these studies will be truly positive in about two-thirds of patients. Endoscopic ultrasonography can also be very helpful. In the past, no further studies were performed, regardless of the outcome of these imaging studies, because the operative success, in the hands of experienced endocrine surgeons, aided by intraoperative ultrasonography (IOUS), was reported in the range of 98%. IOUS is very useful in not only identifying small tumors but also in providing valuable information with regard to the proximity of the tumor to major ducts and blood vessels. In recent years, we have become increasingly aware of a clinical syndrome that we have termed NIPHS. This syndrome is characterized by postprandial hyperinsulinemic hypoglycemia, negative 72-h fast, negative perioperative radiologic localization studies, positive selective arterial calcium stimulation test (SACST), islet hypertrophy/nesidioblastosis (but no insulinoma), and relief of symptoms by gradient-guided partial pancreatectomy. It is becoming increasingly recognized in the post-bariatric surgery population.

D. Surgery. Two-thirds of insulinomas are amenable to surgical enucleation without pancreatic resection. One-third of insulinomas require distal pancreatectomy, which can often be performed with splenic preservation, for smaller tumors of the body and tail. Rarely, Whipple procedures are required for large tumors of the pancreatic head encroaching on the pancreatic or bile ducts. At the Mayo Clinic, out of 185 patients with endogenous hyperinsulinism, 62% underwent enucleation, 34% underwent distal pancreatectomy, and 3% required a Whipple operation. Malignant tumors (<10%) require formal
pancreatic resection with regional lymphadenectomy. Isolated and limited hepatic metastases can and should be treated with resection and/or thermoablation techniques, often providing excellent palliation for extended periods of time. Patients with MEN I syndrome and NIPHS are generally treated with an extended distal pancreatectomy. MEN I patients may require additional enucleations from the pancreatic head remnant. Most MEN I patients will have symptomatic hyperparathyroidism preceding or concurrent with the diagnosis of endogenous hyperinsulinism. A normal ionized serum calcium level virtually rules out MEN I syndrome. Subtotal parathyroidectomy and transcervical thymectomy can be performed for hyperparathyroidism at the same time as the pancreatic procedure in MEN I kindred members. Laparoscopic techniques are now available for insulinoma surgery in select patients.

E. Results. In patients with sporadic, solitary, insulinomas, the likelihood of finding the tumor and curing the patient is ~98–99%. Long-term maintenance of euglycemia is 93%. Diabetes has developed in ~7% of surgically resected patients. Perioperative mortality rates are less than 1% overall. Pancreatic fistulas occur in ~18% of cases and can generally be managed with closed-suction drainage. Patients with NIPHS, following gradient-guided pancreatectomy, have an 80–90% chance for successful early palliation of symptoms. Ninety-five percent of MEN I patients achieve satisfactory glycemic control early after surgery with 20% recurrence rates reported. Survival for malignant insulinomas is stage-dependent like with other islet cell carcinomas. The median disease-free survival after curative resection is 5 years, but recurrences develop in over 60% at a median interval of 3 years. Patients with multiple hepatic metastases can be palliated for prolonged periods of time utilizing hepatic resections, radiofrequency ablations, chemoembolization, systemic chemotherapy, diazoxide, and Sandostatin LAR®.

Table 29.1. Diagnostic criteria (72-h fast).*

<table>
<thead>
<tr>
<th>Neuroglycopenia</th>
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<tbody>
<tr>
<td>• Glucose (plasma)</td>
<td>≤45 mg/dl</td>
</tr>
<tr>
<td>• Insulin (ICMA)</td>
<td>≥3 μu/ml</td>
</tr>
<tr>
<td>• C-peptide (ICMA)</td>
<td>≥200 pmol/L</td>
</tr>
<tr>
<td>• Proinsulin (ICMA)</td>
<td>≥5 pmol/L</td>
</tr>
<tr>
<td>• Sulfonylureas screen (1st and 2nd generation)</td>
<td>Negative</td>
</tr>
<tr>
<td>• B-hydroxybutyrate</td>
<td>&lt;2.7 mmol/L</td>
</tr>
<tr>
<td>• Δ glucose with 1 mg IV glucagon</td>
<td>≥25 mg/dl @ 30 min</td>
</tr>
<tr>
<td>• Glycated hemoglobin</td>
<td>&lt;4.1 mg/dl</td>
</tr>
</tbody>
</table>

* Plasma values at end of fast (EOF)
Insulinoma

A. Clinical Presentation
Neuropathic hypoglycemia
Plasma glucose

B. Diagnosis
(+) 72-hour fast

C. Localization
Triple-phase spiral CT,
TUS or EUS

D. SURGERY
Small Tumors
(<2.5 cm)
Enucleation

Large Tumors (>2.5 cm)
Body & Tail:
Distal
Pancreatectomy
± splenic
Preservation
Head & Uncinate
Enucleation vs
Whipple procedure

E. RESULTS
Survival is stage dependent
Median disease-free survival for curative resection: 5 years
<1% perioperative mortality
12% fistula rate
98% success rate for solitary benign sporadic insulinomas

Malignant Tumors
Distal pancreatectomy or
pancreatoduodenectomy with regional
lymphadenectomy. Debulking
(metastasectomies or partial
hepatectomy) or radiofrequency ablation
for symptomatic hepatic disease.
Nonoperative therapy includes:
chemotherapy, chemoembolization,
diazoxide and sandostatin LAR®

NIPHS
GGPR
(usually extended distal
Pancreatectomy)

MEN I
Distal subtotal
pancreatectomy plus
additional enucleations
as indicated

Successful palliation in 80-90%
Satisfactory glycemic control: 95%
Recurrence rate: 6%
A. Zollinger Ellison syndrome (ZES) is classically characterized by the presence of hypergastrinemia secondary to a gastrin-secreting tumor otherwise known as a gastrinoma. ZES occurs sporadically in 75% of the cases and is associated with multiple endocrine neoplasia-1 (MEN-1) in 25% of cases identified. ZES usually occurs after the onset of hyperparathyroidism in MEN-1 and occurs 50% of the time. Rare causes of hypergastrinemia with acid hypersecretion include retained gastric antrum syndrome, chronic gastric outlet obstruction, chronic renal failure, massive small bowel resection, and gastric G-cell hyperplasia. A careful history and physical, along with other ancillary tests, will help discern these causes from ZES.

B. Gastrinomas constitute the second most common islet cell tumor of the pancreas but are the most common symptomatic malignant endocrine tumor of the pancreas. Although this is the case, approximately half of all gastrinomas occur in the duodenum. Tumors at other locations in the body have been associated with gastrin secretion, ovarian cancer is the only other type associated with hypergastrinemia. Hypergastrinemia is responsible for hypertrophy of the gastric mucosa, including the gastric enterochromaffin cells, which in turn increases the number of parietal cells. This increase causes a greater maximal gastric acid output. A secondary action of hypergastrinemia is the stimulation of acid secretion resulting in increased basal acid secretion.

C. Presentation. Patients with ZES often present with abdominal pain indicative of ulcer disease. However, they tend to have ulcers that recur frequently, require large doses of medications to produce healing, occur in the absence of Helicobacter pylori or intake of nonsteroidal anti-inflammatory drugs, and fail to heal after either H. Pylori therapy or H₂ blockade. ZES may also be suspected in patients who demonstrate severe gastroesophageal reflux disease or heartburn. A secretory diarrhea that persists and that is halted by nasogastric suction is another common presentation. This secretory diarrhea may occur in up to 15% of patients with ZES without ulcer symptoms. Malabsorption and weight loss may also occur secondarily. In ZES patients with MEN-1 syndrome, elevated calcium levels, elevated parathyroid hormone levels, or a pituitary tumor may also be appreciated. In this group of patients, ZES usually occurs in the third to fourth decade of life, with evidence of hyperparathyroidism (hypercalcemia and kidney stones) preceding the ZES diagnosis and occurring during the second decade of life. Hyperparathyroidism occurs in ~90% of these patients, with four-gland pathology being common. All patients with MEN-1 and ZES should be asked about a personal or family history of endocrine diseases, tumors, and kidney stones. Patients with MEN-1 should also have plasma calcium, parathyroid hormone, growth hormone, prolactin, luteinizing hormone, and follicle-stimulating hormone measured. Although values that are normal are more suggestive of sporadic ZES, occasionally manifestation of MEN-1 can occur several years after the ZES. Peptic ulcers are most commonly found in the duodenum. Also, gastric fundic argyrophil cell carcinoids are frequently observed in MEN-1 patients.

D. Diagnosis. Both hypergastrinemia and acid hypersecretion must be confirmed to establish the diagnosis of ZES. Serum gastrin determination can be initially made without having a fasting state or without antisecretory medications. Gastrin levels of 100 ng/L are considered to be the upper limit of normal. If the plasma gastrin is < 100 ng/L, ZES is virtually excluded. Equivocal values between 100 and 1000 ng/liter should prompt a measurement of gastrin level following a fasting state. If the plasma gastrin is >1000 ng/L, gastric acid secretion assays can be pursued to confirm a diagnosis of ZES. If the patient is on antisecretory medication then achlorhydria as a cause of hypergastrinemia must be ruled out. Fast ing gastric pH measurements will help discern the cause of elevated gastrin. A pH <2.0 is strongly suggestive of ZES while a pH>2.0 indicates achlorhydria.

A diagnosis of hypergastrinemia in a patient suspected of having ZES requires a more formal measurement of gastric acid secretion to confirm the diagnosis. A nasogastric tube placed in the dependent part of the stomach and aspirated for as little as 30 min, or more classically for 1 h with 15 min intervals, can be used to calculate acid output. Basal acid output
(BAO) > 15 mmol/h is reported to have the best sensitivity and specificity. If MEN-1 has absolutely been ruled out, a BAO > 18 mmol/h has a greater sensitivity. If the patient has had a previous acid reducing procedure, then a BAO > 5 mmol/h is confirmatory. Basal acid concentration (BAC) when compared to pentagastrin-stimulated maximum acid concentration (MAC) is extremely useful when the BAC/MAC ≥ 0.6. Basal gastric volume of > 160 ml/h and > 140 ml/h in patients with and without previous acid reducing procedures also have excellent sensitivities and specificities for ZES.

Similar to measurements of serum gastrin, the initial measurement of gastric acid output can be made without the patient discontinuing medications. If the BAO measurements are inconclusive initially, the dosage of antsecretory medications can be decreased or discontinued to allow the demonstration of acid hypersecretion. It is imperative that whether the dosage is decreased or discontinued, the subsequent assay should be performed 7 days after the alteration. H₂ blockers can be used as an alternative form of therapy during the discontinuation of proton pump inhibitors.

Historically, the secretin stimulation test has been used in circumstances where it is not possible to measure gastric acid output. A bolus injection of secretin (2 U/kg) in patients suspected of having ZES typically causes a rise of plasma gastrin of greater than 200 ng/L. Unfortunately, the sensitivity of the secretin test in patients with ZES is less than 90%, false positives have occurred with hypochlorhydria, and false-negative rates have been as high as 50%. These limitations underscore the importance of not making the diagnosis of ZES in the absence of a gastric acid assay. The secretin stimulation test may be useful in helping to distinguish ZES from other causes of hypergastrinemia with acid hypersecretion. Similarly, the use of the MAO alone or in the ratio BAO/MAO has not proven to be useful.

E. Location. Approximately 60–90% of gastrinomas occur in the gastrinoma triangle defined by the junction of the cystic and common bile duct, the junction of the inferior margin of the second and third parts of the duodenum, and the junction of the head and neck of the pancreas. The duodenum is the site of gastrinomas in 45–80% of patients with a higher predisposition for this area in patients with MEN-1. There is no universally accepted localization study of choice for a primary gastrinoma. Somatostatin receptor scintigraphy (SRS) is one study available for localizing gastrinomas. The use of radio-labelled octreotide in this study is based on the fact that 90% of gastrinomas have receptors for somatostatin. The SRS is proving to be the superior imaging modality when compared to ultrasound, CT, and MRI. It is also described as being better than portovenous sampling or intra-arterial sampling of the right hepatic vein. Detection is thought to be dependent on size. Endoscopic ultrasound is described as another preferred technique in detecting endocrine pancreas tumors. The combination of endoscopy with transillumination and endoscopic ultrasound may be more effective in detecting duodenal gastrinomas at the time of surgery. This technique can also localize 90–98% of pancreatic gastrinomas. Once discovered they are often described as firm, reddish tan.

F. Medical Treatment. Important to the management of ZES is the initial suppression of high gastric acid. Antisecretory medications can be used with relative ease and can start with a simple regimen of oral proton pump inhibitors. If oral proton pump inhibitors are unavailable, oral H₂ antagonists can be used as an alternative. If a more rapid regimen of treatment is required, then intravenous H₂ antagonists can be used. Since the complications associated with the ulcer diathesis can occur over a relatively short period of time, initiating treatment concurrently with the workup of ZES is appropriate. Gastric secretion should be controlled in the perioperative period with proton pump inhibitors or H₂ antagonist adjusted to decrease gastric acid output to ~5 mEq/h. Acid secretion fluctuates. Therefore, it must be tested often (3 times/day). Overall long-term treatment with proton pump inhibitors provides excellent results but requires lifelong daily medications.

G. Surgical Treatment. The surgical management of ZES remains a controversial area particularly since control of the acid hypersecretion may be achieved with medication. Gastrinomas identified in the pancreas should be enucleated if possible. If they are adjacent to a duct, injury to the duct should carefully be avoided. If injury is unavoidable, it should be repaired and a drain placed. Large tumors distally should be removed with distal pancreatectomy. Pancreaticoduodenectomy has been described, but the increased morbidity and mortality prohibits its routine use. Tumors in the duodenum can be difficult to both localize and resect. Tumors identified endoscopically should be excised with a full thickness elliptical incision protecting the papilla. Palpation can sometimes help to identify larger tumors. Duodenotomy is essential for identification of duodenal gastrinomas, which occur more common proximally than distally. Transillumination using the endoscope allows placement of the duodenotomy incision without incising into tumor tissue and avoids injury to the papilla. When MEN-1 patients with ZES have multiple tumors, these tumors are often microadenomas scattered throughout the pancreas. In patients with ZES and MEN-1 without scattered tumors, but life-threatening nongastrinoma tumors such as an insulinoma, limiting the surgery to the resection of the insulinoma has been advised.

Adjuncts to surgery either at the time of the initial operation or in lieu of other operations include total gastrectomy or parietal cell vagotomy. Total gastrectomy may be indicated in patients unable to take antisecretory medications or in MEN-1 patients with multiple fundic carcinoids. Parietal cell vagotomy can produce a reduction of ~40% in BAO. This may decrease drug requirements, particularly in those patients who ultimately will fail surgical excision. Also, a parietal cell vagotomy can be considered in patients who are found to have unresectable disease or in women of childbearing age wishing to have children. A subtotal gastrectomy should never be an option. During abdominal exploration,
special attention should be paid to the right subhepatic and paraduodenal area to the pelvic cul-de-sac and ovaries. The entire small bowel and colon should also be examined. If all tumors are removed, the immediate cure rate is 60–90%. But ~30–50% of patients initially free of disease show symptoms or biochemical recurrences by 5 years. In patients with MEN-1, the management of hypercalcemia is important in the control of acid hypersecretion. Hyperparathyroidism exacerbates hypergastrinemia and acid hypersecretion and increases antisecretory medication requirements. Therefore all MEN-1 patients with hyperparathyroidism should undergo parathyroidectomy.

Approximately 75% of ZES will remain benign, while 25% will become more aggressive indicated by larger tumors and liver metastasis, shorter history of symptomatic onset, higher serum gastrin levels, tumors to the left of the superior mesenteric artery, and a greater female predilection. Gastrinoma that metastasizes to the liver is associated with the presence of tumors >3 cm and the sporadic form of ZES. Liver involvement portends a decreased long-term survival. Lymph node involvement is not a determinant of decreased long-term survival. The treatment for metastatic disease has undergone several changes, but is unsatisfactory. Radiation and chemotherapy are largely ineffective.
Zollinger Ellison Syndrome

A. Zollinger Ellison Syndrome
75% sporadic
25% MEN-1

B. Gastrinomas
2nd most common islet cell tumor
50% in duodenum
Hypergastrinemia with increased acid output

C. Presentation
1. Abdominal pain
2. Ulcer on EGD
3. Secretory diarrhea
4. ↑ calcium, ↓ PTH or pituitary tumor with MEN-1
5. Recurrence of ulcer despite adequate treatment

D. Diagnosis
1. ↑ Gastrin >1000ng/L
2. Fasting gastric pH <2.0
3. Basal acid output at >15 mmol/hr
4. Secretin stimulation test

E. Location
1. Gastrinoma triangle 90%
2. Somatostatin receptor scintigraphy
3. Ultrasound, CT, MRI unreliable
4. Endoscopic ultrasound adjunct at surgery

F. Medical
1. PPI
2. H2 antagonist

G. Surgical
1. Enucleation
2. Resection
3. Total gastrectomy
4. Vagotomy
A. Signs and Symptoms. Hypertension affects 40–50 million people in the United States; the majorities (90–95%) of the cases are classified as essential hypertension. Secondary hypertension is clinically important because it may be reversible and hence, a search for the cause is warranted. Secondary hypertension has several causes. *Renal parenchymal disease is the most common cause* followed by renovascular disease and various other disorders, including hyperaldosteronism, pheochromocytoma, and coarctation of the aorta. One should have a high index of suspicion for secondary hypertension in patients with resistant or severe hypertension, onset in younger (<30 years) or older patients (>55 years), abrupt onset, rapid worsening of pressure after initially good control, those with renal bruits, or absent or delayed femoral pulses. Although the end result is hypertension, the presenting signs and symptoms of each of the conditions listed below may vary greatly.

B. Renal Parenchymal/Vascular Disease. Renal parenchymal diseases include polycystic kidneys, diabetic nephropathy, glomerulonephritis, and obstructive uropathy. The cause of hypertension is multifactorial and includes disturbances in salt balance, neurogenic factors, and vasodepressors. Historical clues suggestive of renal parenchymal disease include weight change, frequent urinary tract infections (UTI's), and a family history of polycystic kidney disease. Laboratory screening tests include creatinine level and urinalysis. An elevated serum creatinine level, proteinuria, hematuria, or glucosuria warrant further testing. A 24-h urine collection for protein, creatine, and creatinine clearance is helpful in diagnosing nephropathy. A renal ultrasound examination is useful to detect polycystic kidneys, measure renal size, assess for hydronephrosis, and identify renal tumors. Once the cause for parenchymal disease is identified, various treatment modalities may then be employed for treatment.

Renovascular hypertension, that is, renal artery stenosis, is present in less than 1% of the general hypertensive population. It should be suspected in patients with hypertension and an upper abdominal bruit. The two main causes are atherosclerosis (75%) and fibromuscular dysplasia (25%). These include unilateral artery stenosis (Goldblatt’s kidney), bilateral renal artery stenoses, and stenosis in a solitary kidney. The type may be important in determining therapy, for example, angiotensin-converting enzymes (ACE) inhibitors are useful in patients with unilateral artery stenosis but are contraindicated in the other two types of disease.

Obstruction of the renal artery creates a pressure gradient which stimulates the secretion of renin. Renin is a proteolytic enzyme that acts on angiotensinogen to produce angiotensin I. Angiotensin I is then converted to angiotensin II by a converting enzyme located in the pulmonary endothelial cells. Angiotensin II is a potent vasoconstrictor that also enhances adrenal production of aldosterone causing an increase in sodium and intravascular volume.

The best screening test for renal artery stenosis is the captopril (Capoten) renal test which has a sensitivity and specificity of 90%. Captopril blocks the conversion of angiotensin I to angiotensin II, which is needed for the regulation of blood flow in an ischemic kidney. A 50mg dose of captopril is given after baseline plasma renin activity and blood pressure are measured. Blood pressure and plasma renin activity measurements are repeated after 60 min. A reactive rise in renin and fall in blood pressure are diagnostic.

Arteriography is the gold standard for the diagnosis of renal artery stenosis, it can be used to differentiate fibromuscular dysplasia from atherosclerosis, and it provides an opportunity to perform angioplasty. Stenoses due to fibromuscular dysplasia respond better to angioplasty than those caused by atherosclerosis. Midrenal artery lesions may be treated with angioplasty; ostial lesions, however, do not respond as favorably. Stents may be used for ostial lesions with some success. When conservative measures fail, bypass or endarterectomy may be beneficial.

C. Coarctation of the Aorta. Coarctation of the aorta is narrowing or constriction of the medial layer of the aorta and can occur along any of its portion. It most commonly occurs distal to the origin of the left subclavian artery. Coarctation of the aorta is the most common congenital cardiovascular cause of hypertension, affecting more males than females in a ratio of 2:1. It occurs in 1 of every 2000 live term births.
The clinical diagnosis of coarctation is made by comparison of the pulses and blood pressures of the extremities. If the obstruction is proximal to the ligamentum arteriosum or is at the left subclavian artery, a stronger pulse is felt in the right arm as compared to the left arm. If the obstruction is distal to the subclavian, then no difference between the upper extremities is observed. However, lower extremity pulses are weaker than upper extremity pulses.

Electrocardiographic findings are usually nonspecific and show left ventricular strain with left ventricular hypertrophy. Chest X-ray may show cardiomegaly with a dilated ascending aorta or subclavian artery and poststenotic dilation of the descending aorta. Rib notching is due to erosions and indentations by tortuous collateral vessels. Echocardiography is useful for the diagnosis as are CT and MRI. Cardiac catheterization will define the anatomy of the arch, the collateral blood flow, and the hemodynamic status of the patient.

Coarctation must be treated because of possible future cardiac decompensation, aortic rupture, endocarditis, and cerebrovascular accidents. Initial attempts should be made to decrease blood pressure with beta-blockers and ACE inhibitors. Diuretics should be used for volume overload. Definitive management involves surgical repair. Aortoplasty or resection of the narrowed segment with graft interposition may be used. Axillary to femoral bypass grafting may be used in high-risk patients.

D. Primary Hyperaldosteronism. The classic presentation of hyperaldosteronism includes hypertension, sodium retention, hypokalemia, and volume expansion. Primary hyperaldosteronism originates from the adrenal glands without exogenous stimulation. Causes include adenomas, bilateral adrenal hyperplasia, carcinomas, and ectopic aldosterone-producing tumors. It is characterized by elevated aldosterone and decreased renin levels. Secondary hyperaldosteronism is characterized by elevated renin levels. Causes of secondary hyperaldosteronism include congestive heart failure, cirrhosis, ascites, and nephrosis.

The renin-angiotensin system is an important regulator of aldosterone. Renin is synthesized in the juxtaglomerular cells of the kidney and is secreted into the blood where it acts on angiotensinogen, a substrate made in the liver. Reduction in renal arterial pressure increases renin secretion from juxtaglomerular cells and an elevated pressure decreases it.

Aldosterone acts primarily at the kidney where it enhances reabsorption of sodium and secretion of potassium by the distal tubule. The clinical symptoms of primary aldosteronism are related to the hypertension and the hypokalemia. Measuring plasma aldosterone to renin ratio may confirm the diagnosis; a ratio greater than 50 strongly suggests primary aldosteronism. Urinary aldosterone excretion greater than 14 g/24 h is also suggestive. The saline suppression test consists of the infusion of 2 L of isotonic saline over 4 h. In patients with essential hypertension, aldosterone is suppressed to less than 7 ng/dl and is not suppressed in patients with primary hyperaldosteronism.

It is important to distinguish between aldosteronomas and idiopathic hyperaldosteronism. Patients with adenomas may be cured or markedly improved with surgery, whereas those with idiopathic hyperaldosteronism do not respond to either unilateral or bilateral adrenalectomy. Postural testing and measurement of 18-hydroxylated steroid (elevated in patients with adenomas) can be helpful in differentiating these two.

CT scan is used to localize the adrenal lesion. If after biochemical testing and CT scanning the subtype of primary aldosteronism is still uncertain, bilateral adrenal vein sampling may be used. This test relies on the fact that aldosterone secretion is high on the side of an adenoma and is suppressed on the opposite side. The ratio in aldosterone-producing adenomas is generally 10:1. In contrast, both adrenal glands have increased secretion in idiopathic hyperaldosteronism.

The treatment of choice in patients with aldosterone-producing adenomas is unilateral adrenalectomy. Hypokalemia is corrected in nearly all patients and 70% of the patients will have improvement in their blood pressure within 1 year. Medical management may be used for those patients who are poor surgical candidates.

E. Pheochromocytoma. Pheochromocytoma is a catecholamine-secreting tumor of neural crest cell origin. The most common site of pheochromocytoma is the adrenal medulla (90%). Extraadrenal sites include the paraaortic region, organ of Zuckerkandl at the bifurcation of the aorta, hilum of the liver or kidneys, urinary bladder, and sympathetic ganglia. The rule of 10’s is applied to pheochromocytomas; they occur at a rate of 10%, 10% involve both glands, 10% occur in children, and 10% are malignant. They occur at all ages but most commonly are seen in the young to mid-adult life and with a slight female predominance.

Pheochromocytomas secrete norepinephrine (predominates), epinephrine, and dopamine. Pheochromocytomas generally are benign (90%). Dopamine secretion is usually associated with malignant tumors. Secretion of catecholamines may be precipitated by anxiety, trauma, drugs, food, surgery, or anesthetics. Symptoms associated with the release of catecholamines have been described as the four H’s—hypertension, headache, hyperhidrosis, and heart palpations. Ten percent of pheochromocytomas may be associated with multiple endocrine neoplasia (MEN) type II syndrome (pheochromocytoma, medullary carcinoma of the thyroid gland, and hyperparathyroidism). All patients with pheochromocytoma should be screened for the presence of MEN II by measuring serum calcitonin levels and mutations of the RET protooncogene with genetic testing.

The initial screening tests include a 24-h measurement of urinary normetanephrine or metanephrine and urinary vanilmandelic acid (VMA) and plasma catecholamine levels (norepinephrine and epinephrine). If levels are markedly elevated, the diagnosis is established. However, if the tests are inconclusive pharmacologic testing is then necessary.

The clonidine suppression test is most commonly used to make the diagnosis and is based on the fact that clonidine may suppress neurogenically mediated catecholamine release but not catecholamine excess from a pheochromocytoma. Usually a 0.3 mg dose of oral clonidine is given followed by the
measurement of catecholamine levels. If the plasma catecholamines fail to fall by 3 h, the diagnosis is made. The glucagon test is rarely used because of the hypertensive episodes that can occur. Plasma catecholamine levels are measured after glucagon is given to stimulate the pheochromocytoma.

A pheochromocytoma is best localized with three imaging modalities: CT, MRI, and metaiodobenzylguanidine (MIBG 131) scanning. CT scan is most commonly used because of its accurate detection of lesions 1 cm in diameter. MRI may be used to differentiate benign adrenal disease from pheochromocytomas. MIBG is a precursor for catecholamine synthesis and MIBG 131 scanning has the advantage of tagging chromaffin cells. It is therefore useful in the detection of extra-adrenal tumors.

The goal of treatment is to cure the hypertension and reduce the symptoms associated with the tumor. Long-term medical therapy is reserved for patients who are either unwilling to undergo surgical treatment or are poor surgical candidates. Unilateral adrenalectomy is the treatment of choice for patients with pheochromocytoma. The anterior transabdominal approach is preferred for the reason that 10% are malignant and 10% are bilateral. However, posterior or flank incisions are becoming more popular with the increasing sensitivity of imaging modalities.

Preoperative control of blood pressure is essential. Almost all patients should have alpha-blockade. Phenoxybenzamine is the drug of choice for its long half-life. Phentolamine is a short-acting alpha-blocker used mainly for control of paroxysms of hypertension during the operation. For patients with persistent tachycardia, beta-blockers may be used. Beta-blockers should be used with great care because they can precipitate a hypertensive crisis if the alpha receptors are unblocked.
Surgical Hypertension: Evaluation and Treatment

Suspect when:
- resistant hypertension
- extremes of age
- abrupt onset
- rapid worsening
- renal bruits

A. Signs and Symptoms

- severe hypertension
- very young or old
- malignant hypertension
- bruits

B. Renal parenchymal
Renovascular

- creatinine urinalysis
- 24 hr urine protein
- captopril test
- ultrasound angiography
- beta blockade diuretic
- ACE inhibitor angioplasty or surgery

C. Coarctation of the Aorta

- EKG, CXR
- CT, MRI, echo
- beta blocker diuretics
- ACE inhibitor
- surgery

D. Hyperaldosteronoma

- plasma renin and aldosterone
- low renin
- clonidine test
- CT scan
- vein sampling
- surgery

E. Pheochromocytoma

- 24 hr urine for normetanephrine, metanephrine, VMA
- plasma catecholamines
- radiographic CT scan
- localization
- alpha blockade then beta blockade surgery
A. Presentation and Evaluation. Acute pancreatitis usually presents with severe abdominal pain. Over 80% of all cases of acute pancreatitis are due to cholelithiasis or alcohol abuse. Other causes include medications, recent surgery, endoscopic retrograde cholangiopancreatography (ERCP), autoimmune, viral, trauma, cancer, hyperlipidemia, pancreatic divisum, scorpion venom, hereditary, pregnancy, and idiopathic. Based on the clinical and radiographic evidence, acute pancreatitis is classified as severe or mild. Laboratory findings include hyperamylasemia or hyperlipasemia greater than three times normal values. If these are not present initially but pancreatitis is still suspected, a CT scan of the abdomen is indicated. CT scan is more than 95% sensitive and specific in making the diagnosis of acute pancreatitis.

B. Determining the Severity of Disease. Severe cases have three or more Ranson’s criteria, an Apache score of 8 or more, shock (BP < 90 mmHg), organ failure (pulmonary or renal), or massive GI bleeding. Ranson’s criteria are used to help determine the severity of disease (Table 32.1).

C. Management. If the patient has acute pancreatitis, he/she should be admitted to an intensive care unit, receive nothing by mouth, have a nasogastric tube placed if he/she is vomiting, and receive vigorous intravenous (IV) hydration to maintain renal perfusion. Intubation may be necessary if there is respiratory compromise and IV antibiotics should be started to cover gram-negative and anaerobic organisms. Coverage of gram-positive organisms will also be needed as IV lines, arterial lines, Foley catheters, NG tubes all lead to entry points from skin surface bacteria. Imipenem has been found to be the broad-spectrum antibiotic of choice as it penetrates the pancreatic parenchyma at acceptable concentration levels. Antifungal coverage may also be added if the clinical course becomes prolonged.

A CT scan with dynamic infusion of contrast should be obtained in the first 48 h to rule out pancreatic necrosis, identify fluid collections, and confirm the presence of an inflamed pancreas. Consideration should be given for the placement of a peritoneal dialysis catheter for continuous peritoneal lavage over a 7-day period. This should be done within the first 48 h to clear the pancreatic toxins responsible for hemodynamic instability and respiratory and renal failure. Long-term lavage has also been shown to markedly reduce pancreatic sepsis and reduce the overall mortality rate from severe acute pancreatitis.

D. Pancreatic Necrosis. If the CT scan reveals necrosis, one must be concerned about infection; this is suggested by seeing air within the necrotic material or is proven by fine needle aspiration. Once the diagnosis of infected necrosis is confirmed, the patient should undergo operative debridement. Both open and closed approaches have been described in the literature; the choice is dependent upon the individual surgeon’s expertise and the ability to adequately debride the majority of the infected necrotic material. Noninfected necrosis can be observed (as long as the patient is improving), but if the patient’s status worsens, debridement is indicated.

E. Fluid Collections. Fluid collections should be followed with serial ultrasounds every 2–4 weeks, and if the fluid collections develop a mature wall, are larger than 5 cm, and have persisted for longer than 4–6 weeks, drainage should be performed. There is debate as to whether drainage should be performed percutaneously with radiographic guidance or internally by an operative approach. Simple pseudocysts can be internally drained with an endoscopic or laparoscopic approach. Complicated pseudocysts still require open cystgastrostomy or cystjejunostomy. Most surgeons would agree that internal drainage is the procedure of choice for a large (greater than 5 cm) persistent pancreatic pseudocyst.

F. Uncomplicated Acute Pancreatitis. If the CT scan shows only inflammation, one should continue conservative treatment and go to the mild pancreatitis pathway when the process resolves.

G. Mild Pancreatitis. If the patient does not have any of the criteria for severe acute pancreatitis, then an ultrasound should be performed after the patient has been admitted, given nothing by mouth and hydration instituted. Eighty to
eighty-five percent of all cases of pancreatitis are mild and resolve with conservative therapy. Most failures are due to physician impatience and eagerness to start oral feedings too early. Patients with severe acute pancreatitis will probably not be able to eat for at least 7 days, and most likely much longer; therefore hyperalimentation should be started early. Placement of the appropriate central venous lines or access for nutritional support should be performed with strict attention to sterile technique.

If the ultrasound does not reveal cholelithiasis but an etiology such as medically induced pancreatitis (steroids, diuretics), autoimmune vasculitis, or hyperlipidemia is suspected, then one would treat the underlying condition and no further therapy instituted other than supportive care. Antibiotics are not necessary in mild pancreatitis without cholangitis. If the etiology is unknown and there are no gallstones, then an ERCP should be performed after the pancreatitis resolves. Five percent of pancreatic cancers present as attacks of pancreatitis secondary to pancreatic duct obstruction.

If ultrasound confirms the presence of cholelithiasis and the pancreatitis resolves (return of amylase and lipase to normal levels), a cholecystectomy with cholangiography should be performed during the same hospital admission. In these instances only 20% of patients will have choledocholithiasis; therefore, ERCP should not be routinely performed preoperatively. The 20% who have positive intraoperative cholangiograms can have their stones extracted by a common bile duct exploration at the time of cholecystectomy. If the patient has pancreatitis and cholelithiasis on ultrasound but the process is worsening, an emergent ERCP with stone extraction is indicated; an interval cholecystectomy can be performed after the pancreatitis resolves. If the ERCP is unsuccessful in extracting the common bile duct stones, then the patient should have an emergent cholecystectomy and common bile duct exploration.

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<thead>
<tr>
<th>Table 32.1. Ranson’s criteria.</th>
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<td><strong>On admission to hospital</strong></td>
<td><strong>Within 48 h of admission</strong></td>
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<tr>
<td>Age &gt; 55 years</td>
<td>Hematocrit fall &gt; 10%</td>
</tr>
<tr>
<td>WBC &gt; 16,000</td>
<td>BUN rise &gt; 8 mg/dl</td>
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<tr>
<td>Serum LDH &gt; 350 IV/dl</td>
<td>Base deficit &lt; 4 mEq/liter</td>
</tr>
<tr>
<td>Blood glucose &gt; 200 mg/dl</td>
<td>Serum calcium &lt; 8 mg/dl</td>
</tr>
<tr>
<td>AST &gt; 250 IV/dl</td>
<td>pO₂ &lt; 60 mmHg</td>
</tr>
<tr>
<td>Blood glucose &gt; 200 mg/dl</td>
<td>Fluid sequestration &gt; 4 liter</td>
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Acute Pancreatitis

A. Acute Pancreatitis
   pain
   amylase, lipase > 3x

B. Determine Severity
   Ranson's criteria
   (Table 1)

C. Management
   resuscitation
   possible intubation
   antibiotics
   CT scan

D. Pancreatic Necrosis
   sterile
   infected
   observation as long as condition improves
   debridement

E. Fluid Collections
   follow with ultrasounds
   - drainage if persistent, have mature wall, larger than 5 cm

F. Uncomplicated Acute
   Gall stones
   cholecystectomy
   cholangiogram

G. Mild Pancreatitis
   no stones
   treat cause
A. **Etiology**: Long-term alcohol abuse is the most common cause of chronic pancreatitis. Other causes include high-fat and protein diets and untreated hyperparathyroidism with hypercalcemia. *Chronic pancreatitis usually presents with continuous, daily pain* which may require analgesics or increased alcohol ingestion for relief. Occasionally, exocrine and/or endocrine insufficiency are the first signs of chronic pancreatitis but these usually become manifest only after pancreatitis has been present for a long time.

B. **Physical Examination**: Physical examination is for the most part unremarkable. There may be signs of malnutrition since food stimulates pancreatic enzyme release, thereby worsening pain. Consequently, patients are afraid to eat. Since smoking is a habit that frequently accompanies alcohol abuse, patients may have chronic obstructive pulmonary disease.

C. **Diagnosis**: The pain from chronic pancreatitis may be caused by a pseudocyst, obstruction of the pancreatic duct, or replacement of the gland by a diffuse fibrocalcific process. Other causes of upper abdominal pain must be excluded; tests which assist in this process include upper gastrointestinal endoscopy to rule out peptic ulcer disease and an ultrasound to rule out cholelithiasis. A plain radiograph of the abdomen will reveal pancreatic calcifications in 30–40% of patients. Since the pancreas is usually not well visualized by ultrasound, a CAT scan of the abdomen is generally recommended. This may reveal multiple calcifications, a dilated pancreatic duct (>7 mm), a localized phlegmon in the head or tail, diffuse involvement of the entire gland, or a pseudocyst. To image the duct, ERCP (endoscopic retrograde cholangiopancreatography) is performed; this may be useful if a biliary obstruction (30% of cases) is also suspected. If ERCP is technically difficult or the endoscopist is not able to cannulate the pancreatic duct, MRCP (magnetic resonance cholangiopancreatography) can be performed. Duodenal obstruction is present in 10–15% of cases and may preclude successful ERCP. In these instances, upper gastrointestinal (UGI) barium exam may reveal a stricture usually in the second portion of the duodenum. Tumor markers (Ca19-9, CEA) should be obtained if an occult malignancy is suspected.

D. **Pseudocysts**: If a pseudocyst is found in conjunction with chronic pancreatitis, observation for a period of 4–6 weeks may allow the cyst wall to either mature or spontaneously resolve. Cysts which persist and are greater than 5 cm or are associated with proximal ductal obstruction are drained internally into the stomach, duodenum, or a Roux-en-Y limb of jejunum depending on the location of the cyst in the pancreas. For *simple* pseudocysts without proximal ductal obstruction, an ERCP endoscopist can first attempt endoscopic internal drainage. If unsuccessful, surgery should be considered. For *complicated* pseudocysts or those with proximal ductal dilatation and obstruction, an open surgical internal drainage procedure will be necessary. For pseudocysts, which are not adherent to the stomach or duodenum, a cystjejunostomy via a Roux-en-Y limb is required. External drainage is preferred only when a mature cyst wall is lacking or if the cyst has become an abscess.

E. **Dilated Duct, No Pseudocyst**: For the patient with severe unremitting pain and a pancreatic duct which is dilated greater than 7 mm, there are three common drainage procedures. The most popular is the modified *Puestow* procedure, which is a lateral pancreaticojejunostomy; this drains the main pancreatic duct into a Roux-en-Y limb of jejunum over a distance of at least 10 cm. This procedure relieves pain in 80% of patients. The *Beger* procedure is popular in Europe and consists of a duodenal-preserving resection of the head of the pancreas and drainage of the body and tail duct and a small rim of pancreatic head along the duodenum into a Roux-en-Y limb. The success rates obtained in Europe with the Beger procedure have not been duplicated in the United States. The *Frey* procedure is a modification of the Puestow and consists of coring out of the head of the pancreas in conjunction with a lateral pancreaticojejunostomy extended onto the tail. This procedure combines both the Puestow and the Beger and is gaining popularity in the United States. If biliary (30%) and/or duodenal (15%) obstruction are present, choledochoduodenostomy or choledochojejunostomy and/or gastrojejunostomy may be required. Some surgeons will perform a pyloric-preserving pancreatico-duodenectomy with a Puestow procedure of the body and tail when biliary and/or duodenal obstruction exists.
F. Localized Phlegmon: If the workup reveals a localized inflammatory process of either the head or the body or the tail of the pancreas, a resective procedure of the localized area is the appropriate therapy. For an inflammatory mass of the head of the pancreas, a pyloric-preserving pancreaticoduodenectomy is recommended. For an inflammatory mass of the body or tail of the pancreas, a distal pancreatectomy with splenectomy is performed.

G. Normal Duct, Fibrotic Gland: For a diffusely fibrocalcific pancreas with a normal sized duct, the first line of therapy includes analgesics and pancreatic enzyme replacement to enhance negative feedback on the pancreas. If this is not successful, a celiac nerve block can be performed through a percutaneous injection of phenol or alcohol. These blocks relieve pain for only a period of 6 months and have been used mainly for patients with pancreatic cancer. Splanchnicectomy has been popularized in France with very promising results that have not been duplicated in the United States. The procedure most commonly performed for a diffusely fibrocalcific pancreas with a normal sized duct is near total pancreatectomy. Its disadvantage is that it causes exocrine and endocrine insufficiency, namely, brittle diabetes and its complications. Some authors believe that near total pancreatectomy is overly radical and that the pancreas should be allowed to burn itself out (the pain eventually resolves). What is frequently overlooked is that these patients are disabled, out of work, and, if their pain cannot be relieved, are burdens to society. Pancreatic autotransplantation has been attempted but has many complications, including moving the pain and the location of pancreatitis to the site of autotransplantation. Islet cell transplants have also been attempted but two to three pancreas organs are required for successful islet function. A limited supply of transplantable pancreas organs has kept this procedure from gaining wide acceptance.

The chronic pancreatitis patient is very difficult to manage. One must spend an enormous amount of time, effort, and medical resources in their care. Pain medication must be regulated, abstinence from alcohol should be emphasized, and each patient’s treatment must be individualized according to the diagnostic workup. It must be remembered that frequently failure or persistent pain after surgical treatment for chronic pancreatitis can also be the result of a missed pancreatic cancer.
Chronic Pancreatitis

A. History
- pain
- alcohol abuse
- high fat, protein diet
- hyperparathyroidism
- exocrine/endocrine insufficiency

B. Physical exam
- signs of chronic alcoholism
- malnutrition

exclude other causes of upper abdominal pain:
- EGD (r/o ulcer disease)
- ultrasound (r/o gallstones)
- confirm chronic inflammatory changes:
  - abdominal X-ray

C. Imaging studies

assess duct, extent of pancreatic damage:
- CT, ERCP, MRCP
- tumor markers:
  - CEA, Ca19-9

D. Pseudocyst
- observation for 4-6 weeks
- internal drainage for large cyst or obstructed duct
- external drainage if there is infection

E. Dilated pancreatic duct
- pyloric preserving Whipple
- or pancreaticojunostomy
- Frey procedure
- Beger procedure
- add biliary and/or gastric bypass for biliary, duodenal obstruction

F. Localized phlegmon
- resection

G. Diffuse calcifications
- normal duct
- analgesics and enzyme replacement
- nerve block spleenectomy
- total pancreatectomy, transplantation
Small bowel obstruction (SBO) is one of the most common conditions encountered by the general surgeon. In the majority of patients a thorough history and physical exam, complemented by plain abdominal radiographs, is all that is required to make the diagnosis. Postoperative adhesions are the leading cause of SBO (70%) followed by incarcerated hernias (inguinal, femoral, incisional, or ventral). Other less likely causes include primary or metastatic tumors, intussusception, radiation enteritis, and inflammatory bowel disease.

A. Diagnosis: Patients present with colicky abdominal pain accompanied by nausea and vomiting, and obstipation. One should elicit from the history if there has been prior abdominal or pelvic surgery, or treatment for cancer or inflammatory bowel disease. Physical exam usually reveals a distended abdomen; however, if the obstruction is proximal in the small bowel, distension may not be prominent. Tenderness may or may not be present. Hyperactive or “tinkling” bowel sounds may be heard upon auscultation of the abdomen, particularly in early obstruction, while hypoactive or absent bowel sounds are more likely to be found in long-standing cases. Laboratory studies may reveal abnormal electrolyte levels and leukocytosis, but these findings are nonspecific. Four view abdominal x-rays (including upright or decubitus views), in conjunction with the history and physical exam, usually confirm the diagnosis. Common radiographic findings include dilated loops of small bowel (≥3 cm) and air-fluid levels (best seen on upright views). Alternatively, these may be seen on decubitus views if the patient cannot assume the upright position. Paucity or absence of air in the distal gastrointestinal tract may also be noted. If these x-rays are unsuccessful in establishing a firm diagnosis, a computed tomography (CT) scan may reveal evidence of complex problems such as tumors, abscesses, or a variety of inflammatory processes, such as diverticular disease of the colon or pelvic inflammatory disease, which may cause extrinsic compression of the small bowel. CT scan may also show a transition point between dilated proximal and decompressed distal bowel or changes consistent with strangulation and infarction of the bowel. In most cases, however, abdominal plain films are the only radiologic studies needed.

B. Initial Resuscitation: Immediate intravenous volume replacement with an isotonic fluid such as normal saline should be instituted, as patients are typically dehydrated and depleted of sodium, potassium, and chloride. A nasogastric (NG) tube should be placed and intermittent suction applied to relieve intestinal distention and reduce the risk of aspiration. In addition, urine output should be monitored with a bladder catheter to assess the adequacy of resuscitative efforts. For extremely ill or unstable patients, a central catheter may help manage the patient’s hemodynamic status. Once the diagnosis of SBO is made and resuscitative measures are in place, the question of operative versus conservative management must be addressed.

The majority of patients with SBO have partial or incomplete obstructions without bowel compromise. Often IV fluids and NG suction alone are adequate treatment for these patients; resolution of symptoms can be seen in up to 80%. Patients should be monitored carefully with serial abdominal examinations and abdominal x-rays. Clinical improvement is demonstrated by a decrease in pain, nausea and vomiting, and bowel distension, or by the return of bowel function as indicated by the passage of stool or flatus. An improvement in the patient’s clinical condition usually correlates with radiographic findings as well, with fewer air-fluid levels and decreased bowel distension. Once bowel function returns, the NG tube is removed and the patient is given a liquid diet which is advanced as tolerated.

C. Surgery. Operative intervention is required for patients presenting with signs of peritoneal inflammation (rebound, guarding, and rigidity), free intraperitoneal air, worsening pain, and strangulating obstruction, and for patients who fail to improve with conservative management after a brief period of observation. The concern in any of these situations is compromise of the blood supply to segments of the intestine and subsequent intestinal infarction. Symptoms suggestive of intestinal compromise include persistent abdominal pain with tachycardia, fever, and leukocytosis, though these signs are by no means specific. During laparotomy, the small bowel is examined from the ileocecal valve to the ligament of Treitz, and adhesive bands are lysed. In cases where bowel viability is
in question, the color, peristalsis, and arterial pulsations of the intestine (by palpation and Doppler ultrasound) may help determine if bowel resection is necessary. Fluorescein staining and second-look operations 24 hours after the initial surgery are additional options for assessing bowel viability. The decision to proceed with a second-look operation is made at the first operation, usually when bowel of marginal viability is encountered and additional resection of bowel, which may in fact be normal, may jeopardize subsequent nutrition and absorption.

Following a nonurgent adhesiolysis, the intestinal contents are milked proximally into the stomach and aspirated by the NG tube. This relieves distention, improves blood supply to the intestine, and facilitates closure of the abdomen. The patient is then maintained on IV fluids and NG tube decompression until bowel function returns.

D. Special considerations: Hernias may cause an SBO. If a hernia is found and is easily reduced, then IV fluid replacement and NG tube suction are all that are required; this is followed by elective herniorrhaphy after obstructive symptoms resolve. Spontaneous resolution of symptoms occurs in the majority of these cases. If the hernia is incarcerated, emergent surgery should be pursued, as strangulation is likely to occur.

There are certain situations in which all conservative measures should be undertaken before considering surgery. Early postoperative SBO is usually caused by soft adhesions which often disappear in 7–14 days. These patients typically do well solely with NG tube decompression and IV fluids. Ischemic changes are rarely encountered. In cases of recurrent SBO, operative treatment may predispose the patient to continued adhesion formation and should thus be done judiciously, though in cases of complete obstruction, operation is warranted. Conservative management may also be preferable for patients with inflammatory bowel disease, particularly Crohn’s disease, given the risk of fistula formation with bowel manipulation. Furthermore, repetitive operations and resections may precipitate malabsorption; therefore caution should be exercised. Short strictures can be treated with stricturoplasty without resection; multiple such procedures can be performed in an individual patient. Leak rates are acceptable and this form of treatment for obstruction is generally considered safe and highly efficacious.

Radiation enteropathy should also be approached judiciously because of the inherent difficulty in surgically managing such bowel. Short intestinal strictures may be resected or treated with stricturoplasty. Longer segments may be better treated with bypass especially if the responsible segment of bowel is tethered deep within the pelvis.

Finally, the patients with known metastatic intraperitoneal carcinoma may be a challenge because they are usually in the later stages of their disease when they present and have a poor prognosis even with surgical intervention. In such cases, medical management may be all that is indicated for relief of symptoms.

With SBO, the course of treatment—whether conservative or operative—depends upon sound clinical judgment. The treating surgeon must understand the importance of resuscitation, bowel decompression, and most important, if and when to operate in order for the patient to have the best possible outcome.
Small Bowel Obstruction

A. Diagnosis
   history and physical exam
   flat and upright abdominal X-ray
   (dilated bowel, air-fluid levels)

   negative X-rays → CT scan

B. Resuscitation
   IV fluids
   correct electrolytes
   monitor urine output
   nasogastric decompression

   free air peritonitis pain → no hernia
   no hernia → no pain
   serial physical exams, X-rays
   no improvement after 24 hours

C. Surgery

   viability questioned
   check bowel color
   check peristalsis
   check pulsations
   fluorescein injection

   second look laparotomy

D. Special Considerations

   hernia present
   early postoperative obstruction
   Crohn's disease
   history of radiation
   probable metastatic disease

   exhaust non-surgical treatment

C. Surgery
A. Introduction. Mesenteric ischemia from occlusive disease of the mesenteric vessels is a relatively uncommon problem but may have catastrophic consequences if not immediately recognized and treated. Most cases appear in the elderly, although any age patient may be affected. Patients with ischemic bowel may present with “pain out of proportion to physical findings.” Although presentation may vary based on the exact etiology of the condition, the degree and acuity of ischemia, and other concurrent medical conditions, it must be stressed that classic clinical manifestations of intestinal ischemia (fever, leukocytosis, peritonitis, and acidosis) are the result of systemic signs of bowel necrosis and as such signify advanced disease. Regardless of the exact cause of mesenteric insufficiency, ischemic bowel must be identified earlier rather than later, as failure to recognize and treat this condition in its early stages decreases the chance for bowel salvage and adversely affects survival. Management in an intensive care setting is mandated in the patient suspected of having mesenteric ischemia.

The causes of ischemic bowel include acute mesenteric arterial occlusion from either emboli or thrombosis, nonocclusive mesenteric insufficiency, mesenteric venous occlusion, and chronic mesenteric insufficiency.

B. Acute mesenteric arterial occlusion is often caused by embolus to the superior mesenteric artery (SMA), though the celiac artery may be affected as well. The embolus is frequently of cardiac origin and occludes the SMA just distal to its origin off the aorta, commonly at the division of the middle colic artery and jejunal branches. Thrombosis at the site of an atherosclerotic plaque, usually at the origin of the SMA within 2.5 cm of the aortic ostium, is another cause of acute mesenteric arterial occlusion. With either embolus or thrombosis, acute arterial occlusion may lead to thrombosis further downstream within the mesenteric vessels, compounding the problem. Abrupt, severe periumbilical pain is the predominant symptom (i.e., “pain out of proportion to physical exam”). However, in its early stages, the pain may be mild and unimpressive. As ischemia progresses to the point of full-thickness necrosis, pain worsens and bowel perforation and peritonitis ensue. Leukocytosis and a persistent, progressive metabolic acidosis are the most common laboratory abnormalities seen in this condition. Radiographic findings are nonspecific, consisting of edematous, fluid-filled bowel loops on abdominal films. Acute mesenteric ischemia should be suspected in patients with cardiac disease, particularly arrhythmias, valvular disease, or recent myocardial infarction, as well as in those with severe atherosclerosis or prior history of embolic events. Diagnosis is confirmed on mesenteric angiography, with an embolus typically appearing as an abrupt filling defect lodged at the point of an arterial branch bifurcation. If the patient has no signs of peritonitis and the angiogram reveals a mesenteric embolus or thrombosis, it may be possible to dissolve the clot via direct intra-arterial infusion of tissue plasminogen activator (TPA). Systemic heparinization is also started at this time. The patient is kept in the intensive care unit for aggressive supportive management and observation. If lytic agents are ineffective in dissolving the clot, or if the patient develops peritonitis, surgery is the next step. It must be stressed that surgical intervention should not be delayed to pursue further studies if the patient is clinically decompensating. Operation may reveal necrosis of the entire bowel, in which case no further interventions should be pursued given the extremely poor prognosis in such cases. In the face of segmental necrosis, the SMA is exposed at the base of the transverse mesocolon and is evaluated for blood flow via palpation and intra-operative Doppler ultrasound. An arteriotomy is made over the presumed site of occlusion and embolectomy is performed. A number 3 or 4 Fogarty catheter is placed into the SMA and passed upstream and downstream from the arteriotomy until the clot is removed and blood flow reestablished. Alternatively, in cases of thrombosis due to atherosclerotic disease, a retrograde bypass graft from either the aorta or the iliac artery may be performed. Clearly necrotic bowel must be resected. Viability in the remaining bowel is assessed by the color of the intestine, arterial pulsations in the vascular arcade, and peristalsis. With questionably ischemic bowel, examination of the bowel under a Wood’s lamp after injection of 1 gm of sodium fluorescein is a useful adjunct. Areas greater than
5 mm in diameter that fail to fluoresce indicate nonviability. In equivocal cases, a “second-look” operation 24–48 h after the initial surgery allows reassessment of bowel viability. The decision to restore intestinal continuity is based on the overall condition of the patient, the presence of gross intraperitoneal contamination, and the state of bowel perfusion after arteriotomy. Any doubt about these issues suggests that reconstruction should be deferred and stomas be created. The mortality rate for patients undergoing surgery can be as low as 25% with early intervention but may reach 50–90% in most patients with arterial occlusion.

C. Nonocclusive mesenteric ischemia (NOMI) results from severe vasoconstriction of the splanchnic blood supply and is usually seen in cardiogenic, hemorrhagic, or septic shock. Use of vasopressors to maintain hemodynamic stability, while preventing cardiovascular collapse, decreases the already compromised blood flow to the ischemic bowel. These patients are typically extremely ill, often in an intensive care setting, and may be intubated or obtunded, making diagnosis difficult. As with acute mesenteric arterial occlusion, diffuse abdominal pain unaccompanied by proportionate physical findings may be seen in responsive patients. In the stable patient without a clear surgical abdomen, angiogram of the mesenteric vasculature is both diagnostic and therapeutic. Absence of a large vessel occlusion, coupled with “beading” of mesenteric branches from focal vasospasm, is the classic finding on angiogram. The mesenteric vasculature may also resemble a “pruned tree” as smaller branches vasoconstrict, preventing flow of contrast agent distally. Once the diagnosis is made, angiography enables treatment of NOMI with continuous, selective infusion of papaverine, tolazoline, or nitroglycerine into the SMA. At the same time, the factor that precipitated the episode of vasospasm should be addressed. Peritonitis suggestive of bowel infarction mandates immediate surgical intervention, with the intra-arterial infusion of vaso dilators continued during surgery. Necrotic or nonviable bowel is resected as needed. The methods employed to assess bowel viability and issues regarding intestinal reconstruction are identical to those discussed in the case of acute mesenteric occlusion. Given that patients with NOMI usually have a severe primary cause triggering splanchnic vasospasm, mortality rate with this condition remains high.

D. Mesenteric venous thrombosis results in bowel ischemia due to reduced venous drainage of the bowel. Predisposing factors are multifactorial and include portal hypertension, intra-abdominal inflammatory processes, pancreatitis, trauma, hypercoagulable states, and low-flow states. Patients present with vague, often insidious abdominal pain. Stool samples are often hemoccult positive. Diagnosis is difficult due to the nonspecificity of the symptoms, although CT scan with IV contrast may reveal a poorly filling or non-opacifying portal vein. Angiography confirms the presence of venous congestion and delayed filling of the portal system. Systemic heparinization is the mainstay of treatment once the diagnosis is made, with eventual conversion to coumadin once the hypercoagulable condition is identified. After anticoagulation is started, therapy is primarily supportive, unless progression to peritonitis occurs, which mandates surgery. In contrast to its efficacy in arterial occlusive disease, thrombectomy in mesenteric venous thrombosis is usually ineffective. If the patient survives the initial episode, splanchnic venous congestion may regress as the occlusion recanalizes or as venous collaterals develop.

E. Chronic mesenteric insufficiency typically presents in older patients with atherosclerotic disease of the aorta and its branches. Usually, two of the three major splanchnic vessels (celiac, superior mesenteric, and inferior mesenteric arteries) must be occluded before symptoms occur, given the extensive collateral network between these vessels. After meals, the increased need for mesenteric blood flow, coupled with the presence of fixed, occlusive plaque within the vasculature preventing adequate mesenteric circulation, causes severe, crampy pain aptly described as “intestinal angina.” Periumbilical pain and nausea appear 30 min after a meal and resolve gradually, with these symptoms inevitably reappearing at the next meal. Patients subsequently develop “food fear,” leading to abstinence from food and weight loss. An abdominal bruit may be heard during physical examination in up to 75% of these patients. Angiography remains the diagnostic study of choice, often revealing blockage of at least two of the major splanchnic arteries. Definitive treatment entails surgery, with either transaortic endarterectomy of the occluding plaques or bypass grafting performed to relieve the state of relative ischemia. Bypass grafting using autologous vein or prosthetic graft bridges the supraceliac aorta and both the celiac and superior mesenteric arteries. Typically both affected vessels undergo bypass. The infrarenal aorta or iliac arteries are also potential proximal vascular anastomotic sites if a retrograde bypass is performed. Surgery is highly effective in patients with chronic mesenteric ischemia, with most patients achieving a resolution of their pain and a rapid return to normal weight. In a properly selected patient, long-term graft patency may be as high as 90%.
Ischemic Bowel

A. Introduction
- Pain out of proportion to exam
- Abdominal Xrays
- Fever
- Leukocytosis
- Acidosis

Stable Patient
- 1. Resuscitation
- 2. Angiogram
- 3. Heparinization if occlusive disease found

B. Acute mesenteric arterial occlusion
- 1. Start thrombolytics
- 2. Serial exams
- 3. Repeat angiogram
- Peritonitis
- Or Free air on Xray
- Laparotomy Bowel resection
- 2nd look operation
- Clot dissolving With lytic agent
- Peritonitis
- Continue Thrombolitics and heparin

C. Non-occlusive mesenteric ischemia
- 1. Infuse vasodilators
- 2. Treat precipitating cause
- Vasospasm resolves
- Peritonitis
- Laparotomy Bowel resection

D. Mesenteric venous thrombosis
- 1. Heparin
- 2. Supportive therapy
- Patient improves
- Convert to long term coumadin therapy
- Peritonitis

E. Chronic mesenteric insufficiency
- 1. Food fear, weight loss
- 2. Intestinal angina
- 3. Angiogram reveals stenosis or occlusion of 2 or more vessels
- Poor operative candidate
- Angioplasty Or Stent placement
- Operative candidate
- Endarterectomy Or Bypass

Laparotomy
- Bowel resection
- Clot dissolving
- With lytic agent
- Peritonitis
- 2nd look operation

2
- nd
- look
- operation
Crohn’s Disease
Jacqueline Harrison

A. General Considerations. Crohn’s disease is an idiopathic inflammatory granulomatous disease that may involve any area of the gastrointestinal tract from the mouth to the anus. It has a bimodal age of onset, with the larger peak occurring in the second and third decades and a smaller peak of onset in the elderly. Males and females are equally affected, with an annual incidence of about 5 per 100,000 in the United States. Ileocolic involvement is the most common pattern affecting patients with Crohn’s disease (41%), followed by isolated ileal disease (29%) and colonic disease (27%). Three percent of patients with Crohn’s disease have isolated anorectal disease. Up to 80% of patients have some form of bowel resection within 10 years of diagnosis.

B. History. Physical symptoms depend on the area of involvement. Abdominal pain secondary to partial obstruction is one of the most common presentations of small bowel disease. Diarrhea may or may not be present depending on whether or not the patient has rectal or distal colonic involvement or an enteroenteric fistula. Bloody diarrhea may also occur but is more characteristic of ulcerative colitis. Over half of the patients have weight loss. On physical examination, a palpable abdominal mass may be present. Perianal pain may be present and clues to Crohn’s involvement include anal fissures not located in the midline, multiple fleshy skin tags, bluish perianal skin discoloration, stricturing of the anal canal, or multiple fistulae in ano (i.e., “watering can perineum”). Various extraintestinal manifestations such as pyoderma gangrenosum, oral aphthous ulcers, large joint arthritis, hepatic disease, hypercoagulability, and ocular problems may be present and usually wax and wane with the activity of the intestinal disease.

C. Diagnosis. Several modalities are available for confirming a diagnosis of Crohn’s disease. First, stool should be tested for ova and parasites, enteric pathogens, and Clostridium difficile toxin to rule out these causes of diarrhea. If these studies are negative, colonoscopy is very useful in identifying mucosal edema, fibrotic strictures, aphthous ulcers, and deep linear (“rake”) ulcers. Classically, the distribution is patchy, but continuous involvement may be seen. Proctosigmoidoscopy is inadequate, as these areas may be spared, with disease seen proximally. Biopsies can be taken at the time of endoscopy; often these do not differentiate Crohn’s disease from ulcerative colitis, but a diagnosis of inflammatory bowel disease can be established. A giant cell granuloma may be seen in Crohn’s disease, but this finding is not considered pathognomonic.

Contrast studies are also extremely helpful. Double contrast barium enema can demonstrate cobblestone-like mucosa, skip lesions, longitudinal and transverse ulcers, fistulae, and involvement of the terminal ileum. However, in early or mild disease, all of these features may be absent, making contrast enemas inferior to colonoscopy in this setting. Upper gastrointestinal series with a small bowel follow through or enteroclysis can be extremely helpful in establishing the presence of small bowel disease by demonstration of a “string sign” in the terminal ileum, strictures, and fistulae.

Indium-111-labeled leukocyte scanning may aid in detecting active inflammatory Crohn’s disease (97% sensitivity, 100% specificity). On the basis of this test, patients are identified as to who may be helped with aggressive medical treatment rather than surgery. A patient with a negative scan and a stricture on contrast study likely has a fibrotic stricture that will need surgical correction.

D. Medical Treatment. The mainstays of medical management include mesalamine, corticosteroids, azathioprine, metronidazole, and infliximab. Mesalamine is the first line of therapy, with patients maintained on this even after surgical resection of all gross disease. There is evidence that it decreases the recurrence rate of active disease in this situation. Metronidazole is especially useful as an adjunct to surgical therapy in the patients with colitis or perianal disease. Outpatient dosages should be the minimum effective dosage (250 mg tid), as prolonged use is associated with peripheral neuropathy. Patients hospitalized with exacerbation of active disease should be on intravenous corticosteroids, which may result in dramatic improvement. Of course sepsis is a contraindication to instituting treatment with steroids.
Infliximab, a recent addition to the medical treatment of Crohn’s disease, is a monoclonal antibody directed toward TNF-α. It has been found to be useful in inducing remission (33% at 4 weeks) in active disease. However, studies suggest this is short-lived and the disease quickly returns after infliximab is discontinued, with a mean duration of remission of 18 weeks. Infliximab has also proven useful in inducing healing of enterocutaneous fistulae and in improving severe perianal disease.

E. Surgery. Indications for surgical therapy include failure of medical management, obstruction from fibrotic strictures, free perforation, symptomatic fistulae, anorectal disease, hemorrhage, carcinoma, malnutrition, failure to thrive in children, and severe extraintestinal manifestations. Preoperatively, the patient’s nutritional status should be optimized with parental support, if necessary. Abscesses should be percutaneously drained if possible before surgery. A midline incision should be used so that present or future stoma placement is facilitated. Preoperatively, stoma marking and education by trained enterostomal therapy nurses are indicated in nonemergent cases. Intraoperatively, the goal is conservation of as much bowel length as possible while resecting gross disease. Surgical treatment of Crohn’s disease is palliative, not curative, and many patients will need future bowel resections. Preoperative mechanical bowel cleansing should be performed in elective cases.

Small bowel obstruction may be secondary to acute inflammatory edema of the bowel wall, fibrotic strictureing in chronic cases, mass effect from an abscess or phlegmon, adhesive disease from previous laparotomy, or carcinoma. Obstruction should be treated initially with nasogastric decompression, resuscitation, and a trial of intravenous steroids unless there is an abscess, in which case the abscess should be drained and antibiotics should be administered. For patients who have failed nonoperative therapy and have not had prior surgery, the diseased bowel should be resected. Stricturesplasty is appropriate in some circumstances, especially if there has been previous resection of over 100 cm of small bowel, recurrence within a year of previous resection, and multiple fibrotic strictures. Contraindications to stricturesplasty include malnutrition with a low serum albumin, multiple strictures in a short segment, perforation, and fistulization or phlegmon at the proposed striclesplasty site.

Fistulization is one of the features of Crohn’s disease that distinguishes it from ulcerative colitis and results from the fact that the inflammation of Crohn’s is transmural. Involvement of the serosa of the diseased intestine leads to its adherence to nearby bowel or other viscerla and subsequent fistulization into these structures. The most commonly involved organs are small bowel, sigmoid colon or rectum, urinary bladder, uterus, and vagina. About one-third of patients with Crohn’s disease have internal fistulae. Small bowel to small bowel fistulae can be left in situ if they are asymptomatic. Ileosigmoid fistulae are some of the most common internal fistulae in Crohn’s disease and their surgical management depends on whether or not the sigmoid is actively inflamed. In most cases, the sigmoid colon is an innocent bystander and can be closed primarily after the fistula is resected. The ileum is usually primarily involved in these fistulae and should be resected. Endoscopy or intraoperative frozen section can be helpful in determining whether or not there is active disease of the sigmoid in these cases, in which case, sigmoid resection needs to be performed. Enterovesical fistulas, usually arising from the terminal ileum, should be treated with resection of the involved bowel, repair of the bladder defect, and interposition of omentum between the bladder repair and the bowel.

Enterocutaneous and colocutaneous fistulae are also common in Crohn’s disease and may arise postoperatively or after spontaneous abscess drainage. Initially, these fistulae are treated with bowel rest, antibiotics to control sepsis, and percutaneous drainage (by CT) of persistent or residual abscess. Those arising postoperatively may heal with conservative therapy, but those arising spontaneously and those arising from areas of active Crohn’s disease usually will not. In these cases, surgical therapy with resection of the involved bowel and primary anastomosis should be undertaken.

Fulminant colitis should be treated with bowel rest, broad-spectrum antibiotics, and intravenous steroids. If the patient’s condition deteriorates or fails to improve within 5 days, operative intervention should be undertaken. In this case, subtotal colectomy with ileostomy and mucus fistula or Hartman’s pouch are appropriate. Hartman’s procedure allows resection of more bowel and avoids a second stoma. If disease in the remaining rectum continues to be symptomatic, a proctectomy can be performed at a later setting. Minimal disease in the rectum allows construction of an ileorectal anastomosis in an elective setting. Fulminant colitis may cause a toxic clinical picture whereby the patient is tachycardic, has abdominal distension, and appears critically ill. In this setting, surgery should be undertaken within 24 h if improvement is not seen.

For intractable colitis in a nonurgent setting, one option is proctocolectomy with ileostomy for those patients with severe rectal involvement and/or perianal disease or incontinence. Subtotal colectomy with ileorectal anastomosis may be appropriate for those with rectal sparing. Total proctocolectomy with an ileal pouch anal anastomosis is contraindicated for patients with known Crohn’s disease. Segmental colectomy may be considered for those patients with limited colitis or stricture with the understanding that they may need more surgery in the future. However, segmental colectomy can provide relief of symptoms and avoidance of a stoma for several years before further intervention is required.

Hemorrhage should be worked up as in non-Crohn’s patients, with emphasis on mesenteric angiography for localization as the most common site of bleeding is the ileum. Resection of the area is usually required for control.

Crohn’s disease discovered at operation for presumed appendicitis has traditionally been dealt with by appendectomy if the cecum is uninvolved, leaving the disease in situ for
postoperative medical treatment. However, in some studies, 90% of these patients returned to operation for ileal resection within a year despite medical treatment so that some consider resection of disease in this setting acceptable.

Severe extraintestinal manifestations usually improve after resection of diseased bowel. Exceptions are cirrhosis, ankylosing spondylitis, and primary sclerosing cholangitis.

Perianal disease is treated in much the same way as in patients without Crohn’s disease, with the addition of medical treatment. Infliximab in severe disease can be very helpful after adequate drainage of abscesses. Also, noncutting Setons are liberally employed to maintain drainage of complex and/or multiple fistulae rather than multiple or aggressive fistulotomies which may jeopardize continence.
Crohn's Disease

A. General Considerations
   idiopathic
   ileocolic in 41%, small bowel only in 29%, colon in 27%, anal only in 3%
   bimodal age of onset

B. History and Physical Exam
   abdominal pain, diarrhea, weight loss

C. Diagnosis
   endoscopy, radiographs

D. Medical Therapy
   mesalamine
corticosteroids
   metronidazole
   azathioprine
   infliximab

E. Indications for Surgery
   obstruction
   failure to thrive
   hemorrhage
   cancer
   perforation

- total proctocolectomy with ileostomy
- subtotal colectomy with ileorectostomy for rectal sparing
- subtotal colectomy with ileostomy if fulminant
- segmental colectomy for limited disease

small bowel disease

resection indicated
for initial operation,
if disease abuts
ileocecal valve

stricturoplasty for
re-operative cases,
multiple strictures

colonic disease

anal disease

drainage

seton sutures

inflammab
Ulcerative Colitis

Nadav Dujovny

A. Clinical Presentation. Chronic ulcerative colitis (CUC) is an inflammatory condition of the colon which begins in the rectum and extends proximally for variable distances. Typical clinical presentation is diarrhea, accompanied by blood and mucus. Patients often have abdominal cramps, urgency, and tenesmus associated with their bowel movements. Patients’ symptoms vary based on the severity of the disease. Physical exam findings may include fever, tachycardia, dehydration, malnutrition, abdominal tenderness, and blood on digital rectal examination. Extracolonic manifestations include arthritis, iritis, oral ulcerations, primary sclerosing cholangitis, and skin conditions such as pyoderma gangrenosum and erythema nodosum. The cause of ulcerative colitis is unknown, is probably multi-factorial, and may include autoimmune phenomena, environmental factors, and genetic predisposition. A family history of inflammatory bowel disease is often usually present.

B. Work-up. Diagnostic workup depends on the severity of the disease. Stool studies should be done to exclude infectious diarrhea. Serologic and genetic markers can help to diagnose inflammatory bowel disease and may be useful in differentiating between Crohn’s disease (CD), CUC, and indeterminate colitis. A rigid or flexible proctosigmoidoscopy with biopsies can be done easily and safely during initial consultation. Colonoscopy requires mechanical bowel preparation and generally should not be done initially in the ill patient; however, it is superior in terms of defining the extent and pattern of disease and the ability to intubate the terminal ileum allows for further biopsies. Also, the pattern of disease with respect to contiguous retrograde inflammation versus skip lesions or patchy involvement may help differentiate CUC from CD. Histologically, there is distortion of crypt architecture with inflammation or crypt abscess formation. Laboratory evaluation includes a complete blood count, electrolyte profile, renal and liver function tests, and possibly nutritional parameters. Radiographic studies should be done if the patient appears ill. These include plain abdominal films and/or a CT scan of the abdomen and pelvis to assess for perforation, obstruction, or inflammation.

C. Medical Treatment. Therapy for ulcerative colitis depends on disease extent and clinical severity. CUC is initially treated medically; the goal is to induce remission and then to maintain the patient symptom free. Aminosalicylates are intra-luminal anti-inflammatory medications which can induce remission and are used chronically for maintenance therapy. These can be given orally or per rectum depending on disease location. Steroids are used to control disease flare-ups and should not be used as a long-term maintenance therapy. These can be given intravenously, orally, or per rectum. Side effects include truncal obesity, hypertension, glucose intolerance, mood changes, osteoporosis, cataracts, ulcer disease, and growth retardation. Immunosuppressants, such as azathioprine, 6-mercaptopurine, and methotrexate, are used more frequently for maintenance therapy. Cyclosporine may be considered for severe steroid refractory ulcerative colitis. Monoclonal antibodies against tumor necrosis factor-alpha (Infliximab) may be used in maintenance therapy via intravenous therapy every 6–8 weeks. The main benefit of the immunosuppressive and biologic therapy is to decrease or eliminate chronic steroid use. Broad-spectrum antibiotics are also a useful adjunct in the treatment of fulminant or toxic colitis.

D. Urgent Surgery. Surgery can be curative and involves removing the entire colon and rectum. The operation is done in one to three stages depending on the patient’s presentation and the urgency of the surgery. Occasionally, a patient will present with unrelenting bleeding and diarrhea, which is treated with hospitalization, bowel rest, intravenous hydration, and immunosuppression, typically in the form of intravenous steroids. Parenteral nutrition and antibiotics should be considered as well. If these patients do not improve with medical therapy then a colectomy should be considered. Urgent colectomy is often necessary for toxic colitis. These patients present with fever, tachycardia, hematochezia, along with abdominal pain and distention. They may have a leukocytosis and colonic dilatation on abdominal radiographs. Maximal medical therapy should be instituted and these patients should be observed closely. If these patients do not improve within 24–48h (reduced distention, fever, tachycardia), they should
undergo surgery. Colonic perforation is accompanied with up to 50% mortality; prompt surgical intervention can reduce this mortality to less than 5%. After successful medical management of toxic megacolon, 30% will suffer a second episode, and half will require surgery.

The goal of urgent surgery is to remove the colon while leaving future options for sphincter preservation. Surgery will allow the patients to restore their overall health, strength, and nutrition. In the urgent setting, proctectomy is not advised because of potentially more complications and a longer operation. Moreover, attempts at restorative proctocolectomy or an ileal pouch anal anastomosis (IPAA) are unwise and contraindicated in a toxic patient. The rectum is transected at the sacral promontory and left in situ. Some surgeons advocate bringing the rectosigmoid stump through the abdominal wall. This is done to decrease the risk of a rectal stump leak and postoperative pelvic sepsis. A completion proctectomy with an IPAA can be performed in a few months once appropriate homeostasis has been achieved and the patient is off immunosuppression.

E. Elective Surgery. Elective indications include colitis refractory to medical management (most common), intolerance or noncompliance with medical management, prolonged steroid dependence, increased risk or diagnosis of cancer, growth retardation, and extraintestinal manifestations. The risk for colon cancer increases after 8–10 years of disease, especially for those with pancolitis. These patients should undergo periodic surveillance colonoscopy with random biopsies for dysplasia or cancer, the presence of which mandate colectomy. A colonic stricture found during endoscopy or barium enema has a high risk of harboring an occult malignancy and is an indication for surgery. Total proctocolectomy (TPC) may be necessary in the pediatric population because of growth retardation. Also, removing the colon usually improves a patient’s extraintestinal manifestations.

The physiologic state of the patient is clearly the most important factor in determining which surgery should be performed. The other important consideration is sphincter function. If continence is impaired, then the functional outcome of the IPAA will be poor. Prior anorectal surgery, obstetrical trauma, and advanced age may predispose to incontinence and sphincter preservation should be avoided.

Total proctocolectomy with permanent end ileostomy should be considered in the elderly and incontinent patients. Complications include a nonhealing perineal wound, bladder and sexual dysfunction, and small bowel obstruction from adhesions.

Another option is a continent ileostomy or a Kock pouch. Intussuscepting the efferent limb of the pouch into itself creates an internal one-way valve that essentially provides continence for the patient. Intubating the stoma with a catheter several times daily empties the pouch and eliminates waste. Most patients do not wear an appliance (bag). Valvular dysfunction (slippage) is the main complication of this procedure and there is a 20% reoperative rate for obstruction (inability to intubate the pouch) or incontinence.

Total abdominal colectomy with ileorectal anastomosis is another option for CUC. This operation is technically easier and avoids the pelvic dissection. However, the rectum is retained and it must be watched closely for dysplasia and cancer. Up to 40% of the patients will ultimately require proctectomy because of proctitis or cancer risk. The functional results depend on the capacity and compliance of the residual rectum.

Restorative proctocolectomy (IPAA) removes the entire colon and rectum, while maintaining intestinal continuity and preserving fecal continence. This operation can be done in one to three stages depending on clinical presentation and it has become the operation of choice for continent, medically fit patients. Usually, a diverting loop ileostomy is done in conjunction with the IPAA. Following ileostomy closure, patients will have six bowel movements per day. Main complications of the procedure include pelvic sepsis (5%) and small bowel obstruction (15%). Delayed complications include pouchitis, anastomotic stricture (4%), and pouch failure/excision. Pouchitis is a nonspecific inflammation of the ileal reservoir which is usually treated with antibiotics and/or probiotics. At 10-year follow-up, ~50% of the IPAA patients will have had at least one episode. The pouch failure/excision rate is 9% at 10 years. Usually, this occurs because of a subsequent diagnosis of CD, pelvic sepsis/fistula, poor function, or chronic pouchitis.
A. Clinical Presentation
- Bloody diarrhea
- Cramps, urgency
- Extra-colonic manifestations

B. Work-Up
- Avoid colonoscopy in the acute setting

C. Medical Therapy
- Surgery

D. Urgent hemorrhage perforation toxic colitis not responding

E. Elective Surgery
- Refractory to medicine
  - Medical side effects
  - Steroid dependence
  - Fear or rise of cancer
  - Extra-intestinal manifestation

Total proctocolectomy with permanent ileostomy
- Advanced age
- Incontinence

Colectomy with ileostomy
- 40% require proctectomy

Continent ileostomy
- 20% revision rate

Restorative proctocolectomy (IPAA)

Colectomy without proctectomy

Total proctocolectomy with permanent ileostomy

Colectomy without proctectomy

40% require proctectomy

20% revision rate

Restorative proctocolectomy (IPAA)
Appendicitis

John Butsch

Although the clinical presentation of appendicitis has not changed considerably since its first description in 1886 by Reginald Fitz, its mortality has diminished dramatically since William Packard performed the first surgery in 1867. It is one of the most common surgical diseases, annually affecting 233 per 100,000 people in the United States. The highest incidence is in 10–19-year-olds and is more common in men (8.6% lifetime incidence vs. 6.7% in women). The appendix is retrocecal in 65% of patients and is in the pelvis in 20%. This variability contributes to the difficulty in diagnosing appendicitis. Appendicitis is caused by luminal obstruction leading to edema, venous congestion, arterial insufficiency, ischemia, necrosis, and ultimately perforation. Stimulation of the visceral nervous system is transmitted through slow C fibers to T-10 and is inferred as originating from the umbilicus. Eventually, the parietal peritoneum is irritated and the somatic nervous system interprets the location in the right lower quadrant (RLQ).

A. History and Physical Exam. The history and physical exam of the patient is very important. The typical picture is that of a young man with mild periumbilical pain and nausea. There may be one or two bouts of emesis occurring a few hours after the onset of pain. The pain then localizes to the RLQ and progressively worsens. Usually there is anorexia and a low grade fever. Infants are more likely to have vomiting, diarrhea, and irritability. Older children may have had a recent bacterial/viral illness, which causes enlargement of the appendiceal lymphoid follicles and obstruction. The elderly may present with malaise, atypical pain, constipation, or even mental status changes.

Pain from an inflamed retrocecal appendix may not localize to the RLQ and pelvic appendicitis may present with dysuria, diarrhea, or tenesmus. The pain may even be in the right upper quadrant depending on the relationship of the appendix to the cecum. The physical exam should be thorough in its approach. There may be low grade fever and mild tachycardia. The patient should have tenderness around McBurney’s point (one-third of the distance between the umbilicus and the superior iliac spine). There may be a Rovsing’s sign (pain at McBurney’s point when palpating the left side). Patients may have a positive psoas sign (hip extension) or a positive obturator sign (internal rotation of the hip). There might be a mass felt on the rectal exam especially if perforation has led to a pelvic abscess. The pelvic exam might elicit right-sided pain if the appendix is near the ovary or cervical tenderness if it is near the uterus. There might be hypersensitivity of the skin overlying the appendix, suggesting peritoneal inflammation.

B. Diagnosis. Pain elicited on palpation of the RLQ should raise suspicion for appendicitis. Perforation is suggested by the presence of involuntary guarding, a board-like abdomen, a temperature greater than 103 °F, and a white blood cell count higher than 15,000. All patients should have a urinalysis and all women should have a beta-HCG to rule out pregnancy. The presence of microscopic hematuria and pyuria does not rule out appendicitis; these can be caused by any inflammatory mass located in close proximity to the ureter or the bladder.

To improve clinical diagnostic sensitivity, Alvarado’s number system, using the pneumonic MANTRELS, has been proposed. This system is based on a score using Migratory RLQ pain 1; Anorexia or acetone in the urine 1; Nausea and vomiting 1; Tenderness (RLQ) 2; Rebound tenderness (RLQ) 1; Elevated temperature 1; Laboratory leukocytosis 2; Shift to the left 1, for a total of 10 points. A score of 9 or 10 is 100% sensitive while a score of 4 has a sensitivity of less than 4%.

CT scanning is greater than 96% sensitive and 95% specific when using rectal contrast. This imaging modality is being used with increasing frequency and in many instances is obtained even before surgical consultation. CT scans are probably not necessary in clear-cut cases; however, they are helpful in cases where the history and/or physical exam are not entirely consistent with appendicitis. Ultrasound has been shown to have a sensitivity and specificity of 75%–90% and 85%–95%, respectively, but this is observer and body habitus dependent. Sonographic findings suggestive of appendicitis are an appendiceal diameter greater than 6mm and noncompressibility. CT scanning is less observer dependent, has a high negative predictive value, and is more likely to detect other abdominal
pathology. The disadvantages are its exposure to radiation (~300–500 mrads) and its higher cost.

C. Treatment. Symptoms less than 36 h. For all patients, the use of Alvarado’s number and clinical judgment will decrease the incidence of false-negative diagnoses and improve the efficient use of radiological tests. Children with signs and symptoms highly suggestive of appendicitis and a high Alvarado’s number should be taken to the operating room. Those with a low score and/or less clinical suspicion should undergo CT scanning. Prospective studies have shown that CT scans in children decrease the negative appendectomy rate and are cost effective by decreasing hospital stay.

In women of childbearing age, the negative appendectomy rate is as high as 25%; imaging studies are therefore indicated before surgery. The first test should be an ultrasound, which provides information about the ovaries and uterus as well as the appendix. In thin women a transabdominal ultrasound is sufficient, while in obese patients or those with distended loops of bowel, transvaginal scanning is preferred. If the ultrasound is negative or nondiagnostic, then a CT scan should be considered. If all imaging studies are negative, it is not unreasonable to observe a woman with an atypical presentation. Persistent pain after a period of observation should be assessed laparoscopically, at which time the appendix should be removed whether it is abnormal or not.

This algorithm holds true with pregnant women as well. All pregnant patients should be evaluated with an ultrasound, followed by a CT scan if the ultrasound is not helpful. The risk of CT scanning is minimal as compared to that of missed appendicitis in this population. The radiation dose from a focused CT scan is 250 mrads and from a regular CT scan is only 300 mrads, while the maximal allowed dosage for a fetus is 5 rads. If appendicitis is suspected, laparoscopy can be performed in pregnant patients. In young men, the negative appendectomy rate is less than 10%, and therefore if the clinical suspicion and/or Alvarado’s number are consistent with appendicitis, one should proceed to the operating room. If there is doubt then a CT scan should be performed. Again if the CT scan is nonconfirmatory, the patient should be observed and taken to the operating room if he worsens clinically. Laparoscopic exploration can be performed if the patient has persistent pain.

The elderly have higher morbidity and mortality rates and should be treated more aggressively. The highest mortality rate in appendicitis lies in this age group especially when there has been a perforation. The elderly are more likely to have other intra-abdominal pathology, including cancer. Complications such as perforation are more likely to occur as well. A CT scan should be obtained early during one’s presentation. The threshold for surgical exploration should be lower in the elderly, since a delay in diagnosis and treatment may be catastrophic.

Symptoms greater than 36 h. All patients who present after 36 h of pain should undergo CT scanning. If there is a perforation and it is contained to the RLQ, conservative treatment is an option. An abscess can be drained percutaneously by interventional radiology. A large phlegmon on CT scan should be treated with antibiotics and bowel rest. Patients who fail to improve are treated with surgical exploration.
Appendicitis

A. History, Physical Exam

B. Diagnosis
- labs: CBC, beta-HCG, U/A
- Alvarado’s grading system
- imaging: ultrasound, CT scan

C. Treatment

< 36 hours of illness
- children with typical presentation
  - surgery
- women of child-bearing age
  - imaging studies
    - diagnostic: surgery
    - non-diagnostic: observe
- men
  - surgery
- elderly
  - early CT
  - early surgery

> 36 hours of illness
- CT scan
- abscess
  - percutaneous drainage
- phlegmon
  - antibiotics
  - bowel rest
- interval appendectomy
The acute abdomen is considered by many to be the sudden onset of abdominal pain requiring surgical intervention. In reality, not all causes of acute abdominal pain require surgery, and likewise not all “surgical” abdomens are necessarily of acute onset. The assessment of the acute abdomen pain is an art form that all surgeons must master. Understanding the nature of the abdominal pain, as well as the ability to obtain a thorough history and physical, may be the only tools necessary to determine the diagnosis and whether urgent surgical intervention is required. If a patient is stable, carefully chosen diagnostic studies may help to pinpoint or confirm the diagnosis and guide intervention.

Abdominal pain can be divided into two components that correlate with the innervation of the visceral and parietal peritoneum. The visceral peritoneum is innervated by C fibers that course with the autonomic ganglia. Visceral pain is a response to injury of the organ and its adherent visceral peritoneum. Distension, stretch, traction, compression, torsion, ischemia, and inflammation of the visceral peritoneum trigger visceral pain fibers. Contrarily, abdominal organs are insensate to heat, cutting, or electrical stimulation. C fibers are unmyelinated, polymodal nociceptors that conduct slowly (0.5–5 m/s) producing a dull, crampy pain. C fibers travel bilaterally with the sympathetic and parasympathetic chains, and will often be interpreted as vague, central abdominal pain. Organs proximal to the ligament of Treitz, embryologically derived from the foregut (stomach, duodenum, pancreas), will refer to the celiac chain. This is perceived as epigastric pain. Likewise, the midgut (small bowel and right colon) presents as periumbilical pain and the hindgut (transverse, left, sigmoid colon, and rectum) as hypogastric pain.

Somatic pain travels via Aδ somatic fibers coursing with the spinal nerves T7 through L2. Aδ fibers are thinly myelinated, fast conductors that, when fired, are perceived as sharp, pricking pain. When an abdominal process inflames the parietal peritoneum, or peritonitis, the pain becomes severe and localizes to the source of inflammation. Movement or aggravation of the parietal peritoneum will exacerbate the pain.

Clinicians often describe pain as referred or associated with rebound or rigidity. The perception of pain removed from the location of its source, or referred pain, can be predicted by the nerves with which the pain fibers travel. For example, appendiceal obstruction leading to its distension will present as vague, dull, referred visceral pain to the umbilicus. An inflamed gallbladder causing parietal irritation of the diaphragm will refer via the phrenic nerve as shoulder pain. The retroperitoneal genitourinary organs and pancreas share innervation with the abdominal viscera and can also present as abdominal pain. The pain may be vague from referral via the autonomic ganglia of the abdomen and pelvis, or more severe from direct irritation of the abutting parietal peritoneum.

While pain is described by the patient, this should be distinguished from the tenderness invoked on exam. Rebound tenderness occurs after deep palpation of the abdomen is released and indicates peritoneal inflammation. Similarly, muscular rigidity is an involuntary spasm of the abdominal muscles in response to parietal inflammation. When the rigidity is voluntarily overcome by the patient, this is referred to as voluntary guarding. Involuntary guarding cannot be overcome and suggests a more ominous, diffuse peritonitis.

A. History and Physical Exam. Most causes of acute abdomen can be determined by a thorough history. Onset of symptoms and associated activities, changes in bowel habits, color and consistency of vomitus and stool, as well as associated constitutional symptoms (weight loss, fever, anorexia) should be elicited. Determine prior medical problems or surgery, as well as recent or current medications that may predispose a patient to certain entities (i.e., NSAIDS in ulcer disease) or mask symptoms (i.e., oral steroids that may dampen the inflammatory response). Age, family history, alcohol and drug use, and menstrual history are also key elements of the history. The nature of the pain should be explored, including its onset, acuity, radiation, exacerbating or alleviating factors, and its progression over time. For example, appendicitis may present initially as anorexia and dull periumbilical pain (visceral pain), later localizing as severe right lower quadrant pain from
transmural inflammation (localized peritonitis). After rupture and spillage into the abdominal cavity, diffuse peritonitis with rigidity may develop. Bowel obstruction may be described as colicky, crampy abdominal pain associated with bilious vomiting. The emesis may become feculant, and the patient may pass stool early in the process, but later develop obstipation.

Initial exam should assess overall appearance, vital signs, and evidence of shock. A toxic appearing patient with tachycardia and hypotension may be in hypovolemic or septic shock. The respiratory status should be assessed and protected, intravenous access established, and resuscitative fluids initiated. The most urgent and devastating cause of acute abdomen is a ruptured aortic aneurysm and should be considered in any unstable patient. For the more stable patient the general appearance should be evaluated for hydration status (dry mucous membranes, poor skin turgor, slow capillary refill, pale skin, or conjunctiva), as well as the patient’s comfort level (wringing suggests colicky pain, whereas a patient with diffuse peritonitis will lie very still). Exam of the abdomen should be done systematically, avoiding the area of pain until necessary. Inspect the abdomen for distension, surgical scars, organomegaly, asymmetry from mass effect, varices, and respiratory variation. Ask the patients to indicate the area of pain, noting whether they can precisely localize the pain or not. Auscultate, noting hyper-, hypo- or absent bowel sounds, and any abdominal bruit. Percussion can be a very sensitive tool for determining peritoneal inflammation, as well as to determine tympany from distended bowel or a fluid shift from ascites. Gentle palpation should note organomegaly, muscular tone, fullness or masses, hernias, or pulsatile masses as well as tenderness. The testicles should be evaluated for torsion, tenderness, or hernia. Females should have a speculum exam with cultures and a bimanual exam to determine potential pelvic inflammatory or adnexal sources of abdominal pain. The retroperitoneum can be indirectly examined via the obturator (flexed external rotation of the thigh) and psoas (extension of the leg) maneuvers. Pain associated with these maneuvers suggests inflammation in the pelvis or of the muscles of the retroperitoneum and pelvic floor. Rectal exam should include inspection for fissures and fistulas that may be associated with inflammatory bowel disease. Pelvic masses or tenderness can be appreciated, and will augment the difficult exam confounded by abdominal wall rigidity or guarding. Presence of stool and fecal impaction, as well as gross or occult blood, should be noted.

B. Diagnostic Testing. The history and physical should help to focus the diagnostic workup. “Routine” labs, which are often nonspecific, can be very helpful in determining the presence of infection (leukocytosis) or anemia. Electrolyte and hydration status in the patient with vomiting, diarrhea, or prolonged anorexia will help tailor the aggressiveness of fluid resuscitation. Urinalysis may diagnose a urinary tract infection or isolated pyuria, which may be associated with an inflammatory process in the pelvis (i.e., appendicitis or diverticulitis). Transaminases, alkaline phosphatase, bilirubin, amylase, and lipase will help delineate hepatitis, biliary inflammation or obstruction, and pancreatitis. Chest roentgenogram can evaluate for pneumonia or pleurisy that may present as abdominal pain. An obstructive series can evaluate for free air suggesting viscus perforation, air-fluid levels, bowel gas pattern, and dilation suggesting obstruction or volvulus. Bowel wall edema, fecoliths, and occasionally biliary and renal calculi can also be seen on abdominal films. Ultrasound can be used to evaluate for cholecystitis, biliary dilation or stones, hepatic masses, hydropspherosis, or renal calculi. In the female with abdominal pain, pelvic and transvaginal ultrasound will help delineate adnexal pathology from other sources of pelvic pain. Urine or serum HCG level should be determined for every woman of childbearing age, and ectopic pregnancy considered high in the differential diagnosis until proven otherwise. Computed tomography, upper and lower contrast studies should be reserved unless specific disease processes are possible. Pancreatitis, diverticulitis, and abscess are evaluated well by CT scan. Diverticular disease, inflammatory bowel disease, ulcer disease, and malignancies can be evaluated by contrast studies.

Evaluation of the acute abdomen requires carefully exploring the history of the patient. An understanding of the disease processes that can cause abdominal pain, as well as the spectrum and time course of physiologic responses that occur, is essential to assist the investigator in narrowing the differential diagnosis. Of key importance is recognition of the need for early surgical intervention. A diagnosis is not necessary before deciding to operate and a lengthy work-up that can potentially delay treatment should be avoided. A diagnostic laparoscopy or laparotomy should always be considered if catastrophic processes (i.e., ischemic bowel, appendicitis) are likely. A negative laparotomy is never a morbidity. Mastering the sleuth of the history and physical, the efficiency of choosing an appropriate and focused work-up, and the expeditious resuscitation and ultimate treatment is the art of acute management of abdominal pain.
The Acute Abdomen

A. History
   Physical Examination

   suspect ruptured aneurysm?
     yes → resuscitation and urgent surgery
     no
       no → hemodynamically stable?
         yes → rigid abdomen?
           yes → chest X-ray abdominal X-rays
           no → treat
           non-specific investigation, operate promptly if fails to improve
         no → treat
       yes → treat

B. Diagnostic Tests
   choice determined by location of pain and most likely diagnosis at that site
     RUQ epigastric
     LUQ
     RLQ
     LLQ
     perforation obstruction → resuscitation, surgery

A. Pain. Diverticular disease of the colon is an acquired condition affecting societies that consume a refined, fiber-deficient diet. Consequently, the degree of colonic muscle contraction needed to propel hard, inspissated stools caudally is increased and this, in turn, causes herniation of mucosa through weakened points. These false diverticula are found between the taenia (where there is only the circular muscle layer of the muscularis propria) at the point where the vessels penetrate the bowel wall from the subserosa to the submucosa. Most diverticula are located in the sigmoid colon and do not produce symptoms. Occasionally, a diverticulum will become infected with normal colonic flora as a result of stasis or mucosal abrasion from a fecalith; this sets in motion a cascade of inflammation which may spread either longitudinally within the wall or transmurally into adjacent structures. A patient will then experience left lower quadrant pain. Other conditions which initial produce a similar clinical picture include cancer, ischemic colitis, inflammatory bowel disease, and irritable bowel syndrome.

B. Initial Evaluation: Initial evaluation should include a query into how long the symptoms have been present, whether the patient has been hospitalized for this in the past, and whether there are cancer-related symptoms such as weight loss or blood in the stool. Physical examination should include auscultation and palpation of the abdomen. One should obtain a complete blood count (leukocytosis) and an upright chest x-ray to rule out pneumoperitoneum. The patient should receive antibiotics effective against gram-negative and anaerobic bacteria; the route of administration is determined by the patient’s clinical condition.

C. Mild Diverticulitis: Outpatient treatment is possible for the afebrile patient who lacks a profound leukocytosis, pneumoperitoneum, or abnormal physical exam. Oral antibiotics, a liquid diet, and bed rest are prescribed. When the symptoms have resolved, a barium enema or colonoscopy should be obtained to rule out cancer or other causes of the symptoms. Approximately 70% of patients so treated will never have a recurrence of diverticulitis.

D. Peritonitis: Patients with peritonitis, sepsis, or pneumoperitoneum are usually gravely ill and require aggressive support and treatment. Invasive studies such as fiberoptic endoscopy or contrast studies of the colon are contraindicated and would delay surgery needlessly. Intravenous (IV) antibiotics are promptly administered and immediate laparotomy is undertaken. Rupture of a pericolonic or pelvic abscess can produce a purulent peritonitis; free rupture of the colon will cause a feculent peritonitis. The best surgical option includes resection of the diseased colon with construction of an end colostomy and closed rectal stump (Hartmann’s resection). To simply drain the abdomen and construct a colostomy is not considered optimal management; the diseased segment of colon should be removed. This should be used only for the most unstable patients who are not tolerating the operation.

E. Moderate to severe diverticulitis: Fortunately, most hospitalized patients do not require urgent surgery. Although they may have fever, localized left lower quadrant pain, and leukocytosis, most can be initially managed nonoperatively with IV antibiotics and bowel rest. Occasionally, a nasogastric tube is needed if the patient has been vomiting. If improvement is not seen within 24–48 h, an intra-abdominal abscess should be suspected and a computed tomographic (CT) scan obtained. CT findings suggestive of diverticulitis include streaking or inflammation of the pericolonic fat, colonic diverticula, and thickening of the bowel wall. If an abscess is seen, consideration should be given to percutaneous drainage under radiographic guidance (see Sect. H.).

F. Phlegmon: Uncomplicated diverticulitis (i.e., no evidence of abscess, fistula, or perforation) is treated nonoperatively as outlined above for several days. Many clinicians advocate surgery after one hospitalization for young patients (less than 40 years). This stems from the fact that these patients are more likely to be hospitalized again and suffer severe infectious complications than their older counterparts. Indications for surgery include failure of medical therapy within a reasonable period of time (usually 5–7 days), worsening clinical signs despite aggressive medical therapy, recurrent attacks requiring hospitalization for treatment, and inability to rule out cancer. Surgery for these latter two indications is performed once the inflammation has been controlled. If nonoperative therapy is successful, a colonoscopy
or barium enema should be obtained when the patient is pain-free to rule out colon cancer, which may present in a similar fashion. Recurrence of diverticulitis requiring hospitalization occurs in ~25% of patients.

G. Fistula: The inflammatory process that accompanies diverticulitis may erode into adjacent organs such as the bladder or the vagina. The former is manifested by pneumaturia, fecaluria, and recurrent urinary tract infections. The latter causes distressing passage of stool and gas per vagina and usually occurs in women who have undergone a prior hysterectomy. Once the inflammatory process has resolved, colovesical fistulas are treated with a one-stage colectomy and primary anastomosis (no colostomy). The bladder opening is simply debrided and closed, so partial cystectomy is not generally necessary. Omentum should be placed between the colon and the bladder, and dependent bladder drainage instituted for 7–10 days. Colovaginal fistulas are treated in a similar fashion, that is, a one-stage resection of the sigmoid colon and immediate anastomosis without a colostomy.

H. Percutaneous abscess drainage: If a pericolonic or pelvic abscess is identified on CT scan, it should be drained percutaneously provided the abscess is accessible and there is a safe “window,” that is, the only route available is not through other bowel segments or viscera. If successful, the advantage of percutaneous drainage is that the patient may then undergo a one-stage operation (resection and anastomosis); however, this operation should be undertaken only when the patient is stable, is relatively pain free, and the inflammatory process given every chance to subside.

Surgery for diverticulitis should follow these guidelines:

I. Surgery:

1. The proximal and distal lines of resection are chosen so that the likelihood of recurrent diverticulitis is minimized. This requires removal of the high pressure areas, namely, the sigmoid colon and hypertrophic descending colon. The distal line of resection should always be in the rectum to ensure the high-pressure sigmoid colon is entirely removed.

2. One does not need to resect all diverticula-bearing portions of the bowel. The proximal margin of resection should be soft, compliant, nonhypertrophied or thickened portions of colon.

3. If the inflammatory process has been severe, consideration should be given to inserting ureteral catheters preoperatively. This may facilitate their identification at surgery and potentially spare them injury.

4. If a colostomy is even a remote possibility, the stoma site should be marked preoperatively by an enterostomal therapist. This is performed in consideration of the patient’s skin creases, waist line, body habitus, and bony prominences.
Diverticulitis

A. Left lower quadrant pain differential diagnosis:
   - cancer
   - ischemic colitis
   - inflammatory bowel disease
   - irritable bowel syndrome

B. Initial evaluation:
   - history, physical exam
   - lab tests
   - chest X-ray
   - start antibiotics

C. Mild diverticulitis (abdominal, normal WBC):
   - outpatient treatment
   - oral antibiotics

D. Peritonitis, sepsis, pneumoperitoneum
   - prompt laparotomy

E. Moderate to severe diverticulitis:
   - IV antibiotics
   - bowel rest
   - NG tube?

F. Inflamed phlegmon

G. Cloacal or colovesical fistula

H. Pericolonic or pelvic abscess
   - Percutaneous drainage

I. Surgery see text for indications and description of technique
As the mean age of our population rises, there will be an expected rise in the incidence of large bowel obstruction (LBO). Prompt diagnosis and treatment of LBO can result in a decrease in morbidity and mortality from this disease process. Signs, symptoms, physical exam, and x-ray findings must be evaluated and interpreted together to accurately diagnose and manage the problem.

A. Differential Diagnosis. The three most common causes of LBO include colorectal carcinoma, colonic volvulus, and complicated colonic diverticular disease. These three disorders make up over 85% of the LBO cases. The relative frequency of these entities varies geographically. In the United States, cancer is the most common cause. In certain areas of the Middle East, volvulus is more common. Other causes of LBO include hernia, fecal impaction, adhesions, radiation injury, endometriosis, ischemic strictures, inflammatory bowel disease, anastomotic stricture, intussusception, and colonic pseudoobstruction.

B. History and Physical Exam. Patients with LBO present with abdominal distention, cramps, and obstipation. Patients with cancer may complain of a progressive decrease in the caliber of the stool, tenesmus, hematochezia, and weight loss. A history of recurrent episodes of left lower quadrant pain, fever, and leukocytosis may suggest previous sigmoid diverticulitis, which may lead to fibrosis and a LBO. Patients with a chronic history of constipation and prior episodes of abdominal distention, colic, and obstipation may have sigmoid volvulus. Ischemic strictures may follow a bout of ischemic colitis that did not lead to perforation and peritonitis. A personal or family history of colon and rectal cancer may suggest malignancy as the underlying cause of obstruction.

The physical exam begins with the overall assessment of the patient’s condition. Vital signs are taken with particular attention to the heart rate, blood pressure, and temperature. Signs of sepsis and shock are reflected by tachycardia and hypotension. Together with fever, these findings suggest the possibility of bowel ischemia, necrosis, or perforation.

Inspection of the abdomen reveals distention from gas and stool accumulation in the bowel. Hyperresonance to percussion and high-pitched bowel sounds may be found. Palpation may identify an abdominal mass such as a colonic tumor, a hepatic metastasis, or a stool-filled cecum. Localized tenderness may also be found. Generalized tenderness with diffuse guarding and rebound tenderness suggests severe ischemia or perforation, findings which mandate emergency laparotomy.

Of utmost importance in the initial stages of evaluation is prompt resuscitation. Intravenous (IV) access must be established and volume replacement given. Significant volume depletion and dehydration may occur because of vomiting and sequestration of fluids in the bowel lumen. Early placement of a nasogastric tube prevents worsening of bowel distension. A Foley catheter is inserted for monitoring urine output. Central venous catheterization and/or Swan-Ganz catheter insertion may be necessary to guide fluid administration for the elderly patient with cardiopulmonary disease.

C. Initial Evaluation. During the physical examination and resuscitation phase, routine laboratory tests should be obtained, including a complete blood count. Iron-deficient anemia is suggestive of chronic gastrointestinal blood loss, which can be seen in colon carcinoma. An elevated white blood cell count is seen with diverticulitis as well as intestinal ischemia. A complete metabolic profile should be obtained as well. Abdominal radiographs, including an upright chest x-ray, should be obtained. The degree and location of gaseous bowel distention is assessed. Free air within the abdominal cavity is an indication for emergent laparotomy. Cecal distension of 10–12 cm should alert one to the possibility of pending cecal perforation. The cecum is the point of greatest luminal diameter, which is the site of highest mural tension (pressure), and hence is the most frequent site of perforation.

In cases of long-standing small bowel obstruction, gaseous distention of the small intestine and air-fluid levels are seen with a lack of colonic gas. A proximal colonic obstruction such as a cecal carcinoma may give this same radiographic appearance. An incompetent ileocecal valve may lead to small bowel distention in the setting of LBO at any level. Radiographs may suggest sigmoid volvulus by the appearance of a “bent inner tube” to the distended sigmoid colon. Imagine a
distended segment of intestine folded over itself with the two ends located in close proximity to each other in the left lower quadrant.

D. Contrast Studies. If physical examination, laboratory examination, and radiographic assessment do not establish a diagnosis, a contrast study is warranted using water-soluble contrast. The use of barium is contraindicated since a perforation would facilitate spillage of barium into the abdominal cavity compounding the morbidity. The water-soluble enema can identify the site of obstruction and help plan further treatment. Any evidence of perforation warrants emergent laparotomy.

In a more subacute setting, CT scan of the abdomen and pelvis with oral, IV, and rectal contrast can be helpful in delineating the cause of LBO. The administration of oral contrast can be hazardous in the setting of intestinal obstruction because of the risk of emesis and aspiration.

E. Volvulus. Volvulus is most frequently seen in the sigmoid colon. It can also occur in the cecum and transverse colon. Up to 40–60% of patients have a history of chronic obstruction and abdominal distention. Spontaneous decompression with resolution of symptoms can occur. The level at which the volvulus occurs will occasionally require contrast study conformation.

When colonic volvulus is suspected on plain radiographs (and confirmed by contrast) and there are signs of peritonitis, emergent surgery is warranted because of the possible presence of ischemic or necrotic bowel. If the patient has no evidence of peritonitis, attempted endoscopic decompression is warranted. This can be done with a rigid proctoscope in the emergency department. If decompression is successful, a rectal tube may be left in place. Decompressing a cecal volvulus may require a flexible colonoscope to reach the level of obstruction. Successful endoscopic decompression is less likely to occur with cecal volvulus compared to sigmoid volvulus but, nevertheless, an attempt at doing so is still worthwhile. This permits bowel cleansing, an elective operation, and avoidance of a stoma. Failed endoscopic decompression warrants prompt laparotomy. Even if endoscopic decompression is successful, recurrence of volvulus is high and, for this reason, resection is indicated once the patient has been stabilized and prepared.

F. Emergency Surgery. Emergent laparotomy is indicated for peritonitis or intestinal perforation. The latter is diagnosed by the presence of free air on plain radiographs or contrast extravasation on a water-soluble x-ray study.

At the time of laparotomy, the etiology of LBO can be easily assessed. In order to remove the septic focus, bowel resection is undertaken provided the patient’s condition permits. It is generally ill-advised to attempt a primary anastomosis in the face of peritonitis or perforation as the risk of anastomotic leak is prohibitive. Consequently, a colostomy is required.

G. Right-Sided Obstructions. A contrast study may identify the area of obstruction in the right colon. A cancer is easily distinguished from a cecal volvulus on contrast radiographs; the latter is seen as a distended viscus shaped like a coffee bean. A cancer will have shelf-like edges and an intraluminal mass. Laparotomy is warranted and an anastomosis is usually possible for right-sided lesions. Small bowel to colonic anastomoses are generally safe even in unprepped bowel. Resection with an ileostomy is needed in the setting of bowel perforation and severe intraperitoneal contamination with stool. Bypass of an obstructed segment of the right colon is infrequently used, but may be indicated for Crohn’s disease where the inflammatory phlegmon may be adherent to the duodenum, kidney, and ureter. Resection in this instance may be extremely hazardous.

H. Left-Sided Obstruction. Contrast studies may identify the level of obstruction to be within the left colon. A determination must be made as to the degree of obstruction. If the obstruction is incomplete, a gentle bowel preparation may be feasible in preparation for laparotomy, resection, and primary anastomosis. With a higher grade obstruction, standard bowel preparation may not be feasible. The use of endoscopically inserted colonic stents has been shown to be very helpful. The stent is placed with endoscopic and radiographic guidance across the obstruction. The stent is self-expanding and can lead to decompression of the obstruction, which then permits subsequent bowel preparation and an elective resection.

If bowel preparation is accomplished, a single-stage procedure with resection of the area of pathology and primary anastomosis can be accomplished. If bowel preparation is not possible, resection with end colostomy and Hartmann’s pouch is an option. A second operation can then be undertaken 2–6 months later for reestablishment of intestinal continuity.

Obstructions higher up in the sigmoid and descending colon can be treated with subtotal colectomy and anastomosis of the terminal ileum to the decompressed distal colonic. Resection with intraoperative bowel lavage has been described and can be useful depending on the degree of bowel distention. Bowel lavage is accomplished by irrigating the colon with several liters of saline infused through a tube placed in the appendix. Following resection, the open end of the distal colon is connected to sterilized corrugated plastic tubing to allow drainage of the saline irrigant. When the fecal load has been evacuated, an anastomosis is then performed.

Under most circumstances, it is favorable to resect the area of pathology and reanastomose if one of the bowel preparation techniques described above can be implemented. Diversions only with a colostomy or ileostomy can be done if the patient is unstable and cannot tolerate a resection.

In conclusion, treatment of LBO requires that the physician have a good understanding of the causes as well as the different diagnostic modalities. Prompt resuscitation and evaluation of patients with LBO is extremely important. Delay in diagnosis and treatment may lead to ischemia and perforation. There are multiple surgical options that may be used.
Large Bowel Obstruction

A. Differential Diagnosis
   - cancer, volvulus, diverticular disease, stricture, IBD, etc.

B. History and Physical Exam
   - inspect for peritonitis
   - begin resuscitation

C. Initial Evaluation
   - lab tests
   - abdominal radiographs

D. Contrast Studies
   - water soluble contrast

E. Volvulus
   - peritonitis
   - normal exam
   - operate
   - endoscopic decompression
   - elective surgery

F. Emergency Surgery
   - peritonitis, perforation
   - resection, stoma

G. Right Sided
   - resection/anastomosis
   - resection/stoma
   - bypass

H. Left Sided
   - Hartmann's resection
   - colonic stent
   - surgical lavage
   - subtotal colectomy
   - diversion alone
Colon cancer is consistently one of the most common malignancies diagnosed in the United States and is a significant source of morbidity and mortality. An estimated 130,000 cases are diagnosed annually and roughly 57,000 patients die in the United States. Although roughly 7% of cases occur in patients younger than 50 years of age, the incidence of colon cancer in the general population increases exponentially after the fifth decade of life.

Data from prospective colon cancer screening and endoscopic polypectomy studies indicate that most colon cancers originate within adenomatous polyps. Normal colonic mucosa, via a series of genetic mutations, slowly transforms into an adenoma and then into a cancer in what has been coined “the adenoma-carcinoma sequence.” This malignant transformation is estimated to occur over 10 years and is the rationale for screening and prevention protocols. Colon cancer is a preventable malignancy because this lengthy premalignant period allows for the detection and eradication of adenomas provided that patients undergo regular colonic screening.

Risk factors for developing colon cancer include a personal history of colorectal adenoma or cancer, inflammatory bowel disease, pelvic irradiation, and breast or genital tract cancers. Inherited risk factors include familial adenomatous polyposis, a family history of sporadic or hereditary nonpolyposis (Lynch syndrome) colorectal cancer, and other less common neoplastic syndromes. Nonetheless, 75% of colon cancer cases are sporadic and occur in patients at “average risk” of developing colon cancer. These patients have no identifiable risk factors other than age over 50.

A. Diagnosis. Despite the implementation of screening protocols, most patients are diagnosed with colonic neoplasms during large bowel evaluations prompted by symptoms. Presenting signs and symptoms include abdominal pain, hematochezia, melena, bowel obstruction, change in stool caliber, diarrhea, and anemia. It is important to evaluate the entire large bowel for synchronous cancers and adenomas with colonoscopy or air-contrast barium enema plus flexible sigmoidoscopy. Up to 5% of patients will be found to have a second malignancy and in these patients the operative plan will need to be altered in order to address both tumors.

B. Preoperative Assessment. The carcinoembryonic antigen (CEA) level should be checked and plain films of the chest should be obtained in addition to routine medical and laboratory evaluations. Computerized tomographic (CT) scans may be performed although this is controversial. The findings may influence the treatment plan; for example, leaving a nonobstructing, nonbleeding tumor in place in the small subset of patients with diffuse metastatic disease is an option to be considered. Furthermore, the finding of one or even several well-positioned liver metastases preoperatively may, on occasion, allow for concomitant colonic and hepatic resections. Patients may inquire about laparoscopic colectomy for cancer. Multi-institutional prospective studies comparing open colectomy to laparoscopic colectomy have shown comparable oncologic results provided the operation was performed by properly trained and experienced laparoscopists. Most surgeons recommend a mechanical and oral antibiotic bowel preparation prior to surgery to minimize luminal fecal content and bacteria in anticipation of a colonic anastomosis. Perioperative antibiotics are also routinely administered.

Informed consent must be obtained. The procedure-specific complications that should be discussed include anastomotic leak and intra-abdominal abscess. The remote possibility that a temporary colostomy or ileostomy may be required should also be discussed. Deep venous thrombosis prophylaxis should be reviewed with the patient. Acceptable methods of prophylaxis include early ambulation, sequential compression devices, subcutaneous heparin, and low molecular weight heparin. Operations for colon cancer are usually lengthy, are performed in the aged, and may incur higher risk of deep venous thrombosis. Consequently, prophylaxis is highly recommended.

C. Obstructing Lesions. Colon cancer patients who present with colonic obstruction cannot tolerate a preoperative bowel preparation and, thus, pose challenging management issues.
After fluid resuscitation and nasogastric tube decompression (for those with small bowel dilatation and vomiting), the patient is taken to surgery. The surgeon has four options: (1) resection with anastomosis following on-table colonic lavage, (2) resection with Hartmann pouch formation and diversion (colostomy or ileostomy), (3) subtotal colectomy, or (4) diversion alone. The first three options remove the pathology while the fourth choice permits bowel preparation and full evaluation of the large bowel prior to resection.

D. Nonobstructing Lesion. The tumor along with a reasonable length of uninvolved proximal and distal bowel and the mesentery should be removed. The mesenteric resection permits lymph node sampling for accurate staging and reduces the incidence of loco-regional recurrence. Care is taken to minimize manipulation of the cancer (no-touch technique). Some believe that early ligation of the vessels, early occlusion of the bowel proximal and distal to the lesion, and the use of tumoricidal colonic irrigation reduce the rate of recurrence. In the event that a small colon cancer or polypectomy site cannot be identified at the time of operation, intraoperative endoscopy is used for localization. Not uncommonly, a colon cancer may be found invading neighboring structures (e.g., the kidney); in this situation, radical *en bloc* resection is advised. Rarely, an unresectable colon cancer is found at operation requiring either a palliative bypass or a proximal stoma formation.

The finding of sessile adenomatous polyps, dysplastic polyps, carcinoma in situ, or polypoid cancers merits special consideration. Large sessile adenomatous polyps not amenable to endoscopic removal will require a segmental colonic resection. Some large sessile benign polyps can be managed via piecemeal excision in multiple colonoscopic sessions carried out at 3–6-month intervals. *Fully* removed sessile or pedunculated polyps that demonstrate dysplasia or carcinoma in situ (cancer confined to the mucosa) do not require resection and can be followed via interval colonoscopy and close surveillance. Fully removed pedunculated polyps, containing well or moderately differentiated adenocarcinoma confined to the head of the polyp without demonstrable vascular or lymphatic invasion and with negative margins, are at minimal risk for lymphatic spread. In this instance, segmental colon resection is not necessary. Sessile polypoid cancers or pedunculated polypoid cancers with less favorable pathology usually require segmental resection. In these cases, preoperative endoscopic tattooing of the polyp or polypectomy site is recommended in order to facilitate identification of the segment in question at the time of surgery.

The abdomen should be thoroughly examined for metastases. Hepatic metastases that are amenable to wedge resection are removed, but more involved hepatic disease is usually only biopsied for histologic confirmation. An extensive hepatic resection (i.e., lobectomy) is considered several months after colon surgery, usually after several cycles of systemic chemotherapy have been given. Unfortunately, during this time the majority of patients develop additional or enlarged metastases that prohibit curative hepatic resection. In the subgroup of patients with liver metastases that remain candidates for resection, metastasectomy may then be carried out.

Patients with Stage 3 disease (regional lymph node involvement without distant metastases) are offered adjuvant 5-fluorouracil-based combination chemotherapy as this regimen has been shown to lower recurrence rates and improve survival. Chemotherapy for selected patients with Stage 2 disease (large lesions without lymph node involvement) has not shown a convincing survival benefit and remains controversial. Adjuvant radiotherapy is not given to colon cancer patients because of small intestine toxicity, among other reasons. Other postoperative adjuvant or palliative treatment approaches, most of which remain investigational, include portal vein or hepatic artery chemotherapy infusion and immunotherapy.

E. Follow-up. Patients with a personal history of colon cancer or adenoma are at increased risk of developing metachronous colorectal neoplasms compared to the general population. Patients are counseled to undergo interval surveillance colonoscopy 1 year post-resection and then at 3–5 year intervals thereafter or as dictated by exam findings. In addition to endoscopy, most recommend serial abdominal and pelvic CT scans at 12-month intervals to evaluate for metastatic disease. Although controversial, we advise serum CEA levels every 3–4 months for patients in whom the CEA level was elevated preoperatively and then normalized post-resection. Aggressive follow up is warranted after colon cancer resection as the majority of recurrences occur within the first 2 years. We counsel first-degree relatives of patients with colon cancer to undergo colorectal cancer screening as their risk for colorectal cancer is increased.
A. **Diagnosis**
- screening programs (reduce mortality)
- symptoms (pain, bleeding, anemia)

B. **Preoperative Assessment**
- serum CEA
- evaluate entire colon and rectum
- CT scanning (optional)

**C. Obstructing Lesions**
- resection with anastomosis
- Hartmann’s resection
- subtotal colectomy
- stoma only

**D. Non-obstructing Lesions**
- mesenteric dissection
- inspect for metastases
- en bloc resection of adjacent organs
- chemotherapy indicated if there are lymph node metastases

**E. Follow-Up**
- serial CEA’s
- colonoscopy yrs 1,3,5

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*large benign sessile polyps: segmental resection or endoscopic piecemeal excision in multiple sessions
carcinoma-in-situ: follow with colonoscopy if completely removed
polyps with cancer: surgery if positive margin, lymphatic invasion, etc.*
Approximately 50,000 new rectal cancers are diagnosed annually in the United States. The most common histologic form is adenocarcinoma; other variants include squamous cell carcinoma (for the distal rectum), neuroendocrine cancers, sarcomas, and lymphoma. This discussion focuses on adenocarcinoma. Most tumors are found on digital rectal exams or colonoscopy performed for screening purposes, or surveillance of high-risk groups (genetic predisposition, inflammatory bowel disease, family history, prior personal history of colorectal polyps, or cancer).

A. Tumor Location. Once the diagnosis is made, the most critical factor in determining treatment is location of the tumor within the rectum. The rectum has been arbitrarily divided into thirds. The distal third extends from the upper aspect of the anal canal up to the first mucosal valve of Houston, a distance of ~5 cm. The mid-third extends from 5 to 10 cm, the upper third extends from 10 cm up to the rectosigmoid junction, which is located at ~15 cm. When radical surgical resection is performed, it is paramount that the surgeon obtain a 2 cm tumor-free distal mural margin; otherwise local recurrence may be increased. Many argue that a 5 cm mesenteric clearance below the tumor is essential in order to adequately remove potential metastases in the mesorectum. This practice is called mesorectal excision (total for distal tumors, partial for proximal cancers). While it may not be problematic for lesions in the mid and upper third, getting a 2 cm distal margin for low-lying tumors may be difficult. It may even be impossible if the lesion is in immediate proximity to the sphincter muscle in which case surgical removal of the entire rectum, anus, and sphincter muscle may be required. The patient then lives with a permanent colostomy. Tumor location is best measured with a rigid proctoscope.

B. Rectal Ultrasound. Once the exact distance from the anal canal has been determined, the treating physician must determine the extent of disease, both locally and at distant sites. The latter can be assessed with computerized tomographic (CT) scans of the chest, abdomen, and pelvis. One is primarily concerned with lung and liver metastases. With respect to the former, the depth of penetration (into the rectal wall) and the presence of lymph node metastases within the mesenteric fat can be determined with imaging studies such as magnetic resonance (MR) scans or endorectal ultrasounds. These two factors (depth, lymph nodes) have been identified in multiple studies as high-risk predictors for local recurrence.

Ultrasound is the preferred method for locally staging rectal cancers. Compared to CT and MR scans, it is less expensive, is mobile (can be used in the office, operating room, or patient’s bedside), and more accurate. If imaging studies show either that the tumor has penetrated into the extra-rectal fat or there are suspicious lymph nodes, most experts recommend initial preoperative treatment with pelvic external beam radiotherapy and chemotherapy. Radiation is delivered to the tumor bed and the pelvic sidewalls to a total dose of ~5000 cGy over 6 weeks. An additional 4–6 weeks is advised before surgery during which time radiation-related edema and tissue fragility should regress. Chemotherapy regimens are 5-fluorouracil-based and are given either with a bolus technique or as a constant infusion during radiation.

Preoperative chemoradiation (also known as neoadjuvant therapy) induces a complete histologic response in 20–30% of patients. The remainder respond to varying degrees. Significant downstaging can be expected and large bulky tumors may be rendered respectable with a higher likelihood of having negative margins. In a small percentage of cases, patients with very distally located cancers may avoid a permanent colostomy if enough shrinkage occurs to allow an anastomosis.

In the absence of transmural penetration or nodal metastases, surgery may be done promptly without delay. The issue at this point is whether the patient is a candidate for conservative surgery or needs a radical operation. Before proceeding with surgery, the remainder of the colon should be checked. Approximately 5–7% will have a metachronous cancer and 30% a benign polyp elsewhere in the colon.

C. Local Excision. Local excision either via a transanal or transsacral approach is an acceptable operation for properly selected cancers. Since any form of local excision merely...
removes the tumor itself and not the regional lymphatics, only those lesions with a low statistical chance of nodal metastases are selected for this option. Such tumors are limited to the mucosa and submucosa (uT1), are well-differentiated, are accessible transanally, and lack lymphovascular invasion. When compared to radical surgery, local excision should provide comparable cure rates for these early superficial lesions. Patients should be followed closely for recurrence. Local excision should not be offered to patients whose tumors have penetrated into the muscularis propria or beyond.

D. Radical Surgery. Transabdominal surgery is advised for rectal tumors that have either penetrated partially into the muscularis propria by ultrasound (uT2) or following neoadjuvant therapy (uT3, 4N0, or any TN1). Again, if preoperative imaging studies show that the lesion has penetrated partially into the muscularis, there is no need for preoperative chemoradiation. If possible, sphincter preservation should be practiced; however, this is dependent on whether the lesion is located in the upper, mid, or distal rectum. This is also determined by the skill of the operating surgeon, the patient’s gender (pelvic surgery is easier in women), and the body habitus of the patient. Resection of the rectum with primary anastomosis is termed low anterior resection (LAR) and is generally feasible the higher the tumor is located in the rectum. When the entire rectum, anal canal, and sphincter are removed, the operation is referred to as an abdominoperineal resection (APR); this is usually reserved for tumors located at or very close to the anal canal or sphincter muscles.

Whenever radical surgery and an anastomosis is done for rectal cancer, the surgeon must decide whether a temporary ileostomy or colostomy is necessary and whether functional outcome can be improved by construction of a colonic reservoir. Generally, temporary fecal diversion is performed when the anastomosis is located low in the pelvis or if neoadjuvant therapy was administered preoperatively. Stoma takedown is performed at a second surgery after postoperative chemotherapy has finished. Functional outcome may be improved by the addition of a colonic reservoir, using either a J-shaped pouch or a coloplasty. Either technique increases the storage capability of the rectum; this can lead to fewer daily bowel movements, less tenesmus, and less urgency. Fecal incontinence may also be enhanced.

E. Upper Rectal Cancers. Lesions above 10cm more closely resemble sigmoid cancers in their natural history and behavior, especially those whose lower edge is at 12cm or higher from the anus. For these lesions, the following practices are generally applicable:

1. Temporary or permanent colostomies or ileostomies are usually unnecessary. There is enough tissue below the lesion to get a negative distal margin, perform an anastomosis, and spare the anus and sphincter muscle.
2. Neoadjuvant therapy is not given. Instead, surgery is performed promptly once the diagnosis is made. Chemotherapy (postoperative) is given if there are histologically confirmed lymph node metastases.
3. Local excision, even for T1 cancers, is usually not possible because these lesions may not be accessible transanally. Advanced surgical techniques using transanal endoscopic microsurgery may provide access if this technology is available.

Follow-up of any patient who as been treated for rectal cancer should include office visits and digital rectal exams every 3 months for 2 years. This is usually combined with rigid proctoscopy to assess for possible anastomotic recurrence. Transrectal ultrasound is added for patients who have undergone local excision. Serum carcinoembryonic antigen (CEA) is checked every 3 months for 2 years. A colonoscopy is done at 1 year.
Rectal Cancer

A. Determine Exact Location
   Rigid Proctoscopy

B. Rectal Ultrasound
   - Depth of penetration
   - Metastatic lymph node

Distal Rectum (0-5cm)

- Ultrasound uT<sub>1</sub>
- uT<sub>2-4</sub> or (+)N<sub>1</sub>

C. Local Excision
   Confirm superficial nature of the lesion
   • temporary stoma
   • Colonic reservoir

D. Radical Surgery
   LAR, APR
   APR less likely to be needed

Mid Rectum (5-10cm)

- uT<sub>1</sub>
- uT<sub>2-4</sub> or (+)N<sub>1</sub>

C. Local Excision
D. Radical Surgery

E. Upper Rectal Cancer

- uT<sub>1</sub>
- uT<sub>2-4</sub> or (+)N<sub>1</sub>

C. Local Excision
D. Radical Surgery

No adjuvant therapy should proceed surgery for uT<sub>3-4</sub> or (+)UN<sub>1</sub> cancer.

Neoadjuvant therapy not given

Postoperative chemotherapy for lymph node metastasis
A. General Considerations. Cancer of the perianal skin (anal margin) and anal canal are uncommon malignancies, constituting less than 5% of cancers of the lower gastrointestinal tract. Most cancers in this region arise cephalad to the dentate line of the anal canal by a ratio of 3 to 1. Anal canal cancers are more frequently found in women, and anal margin tumors are more frequently found in men. Regardless of location, the patients are usually in their mid-50s when the diagnosis is established.

Presenting signs and symptoms include pain, bleeding, itching, mucous discharge, and the sensation of a mass. Advanced lesions may impede the passage of stool, erode into the vagina, cause tenesmus, invade the sphincter, or metastasize to inguinal lymph nodes. Frequently, an unsuspected cancer will be found within a surgical hemorrhoidectomy specimen.

B. High-Risk Conditions. Certain conditions may place an individual at higher risk for anal cancer. Immunosuppression from either HIV-infection or renal transplant medications raises the relative risk significantly. In a manner analogous to cervical cancer, infection with the human papilloma virus (HPV) types 16 and 18 also raises the risk. It appears that the practice of anoreceptive intercourse raises risk independently of either HPV or HIV infection. Risk may be additive. For example, individuals who engage in anal intercourse and are infected with HPV 16 have a relative risk 33% higher than the general population. Other factors implicated include cigarette smoking and chronic untreated perianal disease, such as fistulas and Crohn’s disease. Infection with herpes simplex virus 2 has also been implicated.

C. Diagnosis. It is imperative that any unusual or abnormal perianal or anal lesion be biopsied to establish a diagnosis. This is especially so for lesions that are persistent, nonhealing, and refractory to conservative measures. Tissue may be obtained by simple office-based biopsy under local anesthesia in the majority of instances. Occasionally, an examination and biopsy, under general anesthesia, in the operating room is required if the patient is uncomfortable and experiencing pain.

Most invasive lesions are epidermoid in origin, namely, they are stratified squamous cell carcinomas or their variants—transition cell, basaloid, or cloacogenic. These histologic variants are treated the same. Adenocarcinomas occur within the anal canal, albeit rarely. Bowen’s disease is an intra-epithelial squamous cell cancer that by definition is not invasive; however, it may be widely present in this region and at high risk for recurrence following treatment. Controversial data suggests that Bowen’s disease is associated with an internal malignancy and that it may progress to invasive cancer. Most agree that Bowen’s disease should be treated with wide local excision. Paget’s disease is an intra-epithelial adenocarcinoma.

D. Anal Margin. Lesions that arise distal to the dentate line generally have a more favorable prognosis. Superficial, well-differentiated, small lesions may be treated with wide excision, taking care to obtain negative margins. The defect resulting from local excision may be closed primarily, left open to heal by secondary intention, or covered with tissue rotated internally via anoplasty techniques. More locally advanced lesions are treated with radiation to the primary site and possibly the inguinal nodes.

E. Anal Canal. Local excision is probably an adequate operation for cancers which are limited to the mucosa and submucosa. Some even advocate local excision for lesions which have invaded into the internal sphincter; however, this is controversial. Endoanal ultrasound may assist in determining depth of invasion preoperatively. If the lesion penetrates deeply, local excision should not be considered.

Combined modality therapy for invasive anal epidermoid cancer (radiation therapy and chemotherapy) was initially reported in 1974. The results revolutionized the treatment of this cancer and it has become the treatment of choice, although variations of the protocol are widespread. Preoperative radiation is administered to the primary tumor, the pelvis, and the inguinal nodal basins. Initial work involved a dose of 30 Gy over a 3-week period (15 treatments); most treatment protocols currently use doses between 45 and 50 Gy. Intravenous
5-fluorouracil is given during the first and last 4 days of radiation. A single dose of mitomycin-C is given on day 1. Complete response rates of 85–90% have been reported. After treatment has been completed, biopsies of the primary tumor may be done to check for persistent disease.

F. Persistent or Recurrent Disease. Persistent cancer following combined multimodal therapy is treated with abdominoperineal resection. This operation involves radical removal of the rectum, anus, their mesentery, and the sphincter muscles. A permanent colostomy is created. Recurrent cancer detected during follow-up is treated in a similar fashion. Prophylactic groin dissection is not performed at any time, either at initial diagnosis or when persistent or recurrent is detected. However, if inguinal node metastases are found, groin dissection is performed for local control.
A. General Considerations
   Symptom:
   Pain, Bleeding, Mass

C. Diagnosis
   Tissue needed

B. High Risk Conditions
   HPV infection
   Immunosuppression
   Anoreceptive intercourse
   HIV Infection

Treatment

E. Anal Canal
   Local Excision
   Combined modality therapy

E. Anal Margin
   Wide excision if superficial.
   Radiation, chemotherapy if locally extensive.

F. Persistent or Recurrent Disease
A. History and Physical Exam. Patients often seek medical advice for anorectal complaints. Symptoms are typically attributed to hemorrhoids, but in reality, there are numerous conditions which could cause pain or discomfort. Moreover, many patients tend to delay evaluation because of embarrassment or fear of cancer. Most anorectal conditions are benign and easily treated. However, because of this inherent delay, many of these conditions often present as advanced disease necessitating more extensive treatment. The key historical points are whether anorectal pain is constant or intermittent and whether it is related to bowel movements.

If the patient has obstructive symptoms or changes in bowel habits, the entire colon should be evaluated by colonoscopy or barium enema. Anorectal assessment consists of inspection and palpation. The physician should be vigilant for anorectal cancer, and a proctoscopic evaluation should be considered in all patients. The patient can be positioned in the left lateral decubitus position for the examination or in the prone jack-knife position.

B. Intermittent Anal Pain. Anal fissure is a linear tear in the lining of the anal canal below the dentate line. It typically presents as anal pain only during or after defecation accompanied by the passage of bright red blood. The pain is often severe and may last from a few minutes to several hours post-defecation. Examination reveals spasm of the anal sphincter, which may preclude digital insertion or endoscopic assessment. Acute fissures tend to have distinct mucosal edges and granulation tissue at their base. Chronic fissures have indurated edges, a lack of granulation tissue, a sentinel skin tag, a hypertrophied anal papilla, and some degree of anal stenosis. Fissures are usually single and occur in the posterior midline; however, up to 10% of fissures may occur in the anterior midline, especially in women. Multiple or lateral fissures should raise suspicion for underlying inflammatory bowel disease, tuberculosis, syphilis, or immunosuppression.

More than 90% of acute anal fissures will heal spontaneously or with a high-fiber diet, increased oral fluid intake, sitz baths, and stool softeners. Patients with chronic fissures tend to have elevated resting anal pressures (with manometry) and treatment is directed at reducing the spasm of the internal anal sphincter. First line of therapy includes chemical sphincterotomy with topical nitroglycerin, calcium channel blockers, or botulinum toxin. Patients with recurrent fissures and those who failed medical treatment should undergo lateral internal sphincterotomy where the lower third of the internal anal sphincter is incised. Gross incontinence is rare.

Levator ani syndrome is characterized by intermittent deep-seated rectal pain. It may occur at night, awaken patients from their sleep, and resolve without treatment. Its cause is unknown but symptoms are due to spasm of the pelvic floor muscles. Levator ani syndrome is a diagnosis of exclusion; if common conditions are ruled out using clinical, endoscopic, or radiographic means, then the diagnosis is more secure. Digital rectal examination should be performed and if palpation of the levator muscle reproduces the pain, the patient likely has this condition. No specific treatment has proven itself to be universally successful but commonly used modalities include transrectal or transvaginal pelvic floor massage, nonsteroidal anti-inflammatory agents, and electrical stimulation.

C. Constant Rectal Pain. Anal abscesses and fistulas occur because of infection in the anal canal crypts and glands. These glands are located at the dentate line and can become impacted, leading to a retrograde infection. Patients with abscesses typically present with constant perianal pain and possibly fever along with localized erythema, swelling, tenderness, and fluctuance. Abscesses occur in the perianal, ischiorectal, intersphincteric, and supralelevator spaces, and the patient’s symptoms will vary depending upon location. Perianal and ischiorectal abscess produce localized pain and are usually visible to the examiner. Intersphincteric and supralelevator abscess cause deeper and more poorly localized pain; there are frequently no visible signs of an abscess with these entities. Supralelevator abscesses may be due to pelvic pathology such as diverticulitis and pelvic inflammatory disease.

Treatment for anorectal abscesses is incision and drainage, which can usually be done in the office or emergency room.
Large abscesses or those associated with fever or leukocytosis should be drained in the operating room. The incision should be placed over the area of fluctuance as close to the anal verge as possible. This is done to decrease the potential distance of a fistula. Antibiotics should be used only if there is significant cellulitis, valvular heart disease, prosthetic devices, or if the patient is immunosuppressed because of diabetes mellitus or HIV infection. If an abscess recurs at the same location following an initial incision and drainage, or if the wound doesn’t fully heal, a fistula should be suspected. Fistulae do not cause pain but may cause chronic drainage.

**Hemorrhoids** are cushions of vascular tissue in the anal canal which contribute to continence. These submucosal vessels are sinusoids, not veins, and can exhibit arterial bleeding. The main cushions are located in the left lateral, right anterolateral, and right posterolateral locations. Hemorrhoids are classified as internal or external depending on their relation to the dentate line. They may present with mucosal protrusion, bleeding, mucus discharge, and pain. Pain occurs with thrombosis, strangulation, and gangrene and is usually constant in nature. During physical exam, the patient should be asked to Valsalva to reproduce the conditions which cause prolapse.

External hemorrhoids occur distal to the dentate line and are covered by squamous epithelium. These vessels drain into the internal iliac veins. These hemorrhoids have somatic innervation, which allows for pain sensation. Treatment consists of topical agents to relieve pain, decrease swelling, and improve hygiene. Patients may often present with acute thrombosis. This can usually be treated conservatively with analgesics, stool softeners, and Sitz baths. However, an extremely painful thrombosed hemorrhoid should be excised.

Internal hemorrhoids occur proximal to the dentate line and are covered by columnar or transitional epithelium. These vessels drain into the portal circulation through the superior hemorrhoidal or rectal veins. These hemorrhoids are not typically painful because they lack somatic innervation.
Anorectal Pain

A. History and Physical Exam
   is pain constant or intermittent?

B. Intermittent Pain
   only with BM's
   - anal fissure
     - stool softeners
     - bulking agents
     - topical nitroglycerin
     - calcium channel blockers
     - botox injection
     - surgery (internal sphincterotomy)
   - levator ani syndrome
     - massage therapy
     - NSAIDS
     - electrical stimulation

C. Constant Pain
   - abscess
     - perianal ischiorectal intersphincteric suprarelevator
     - incision and drainage
   - hemorrhoids
     - thrombosed
     - incarcerated strangulated necrotic
     - conservative vs. enucleate
     - operate
   - follow for possible fistula
Upper gastrointestinal (GI) hemorrhage has a variety of causes and may present with subtle or massive blood loss. Potential sources may be located anywhere from the mouth to the ligament of Treitz. The history and physical examination are cornerstones for initial assessment; if the patient’s vital signs are unstable, resuscitation and evaluation are promptly instituted simultaneously with a quick physical assessment and interview of the patient or his/her family.

Intravenous crystalloid resuscitation is begun through two large-bore peripheral catheters (14- or 16-gauge). Laboratory studies such as hemoglobin, platelet count, PT, PTT, and type and cross are obtained. Simultaneously, the patient’s airway is assessed and an 18-gauge nasogastric (NG) tube and indwelling bladder catheter are placed.

Significant hematemesis, tachycardia, or hypotension should prompt early intubation to secure the airway and prevent aspiration. Room temperature saline gastric lavage is performed and the response to initial management is evaluated.

A. Unstable Patient: Patients with massive bleeding and hemodynamic instability should be intubated and transferred to the operating room. Further resuscitation and esophagogastroduodenoscopy (EGD) can be rapidly performed there before exploratory laparotomy. If endoscopy reveals esophageal varices as the source of hemorrhage, laparotomy may be withheld in favor of an attempt at tamponade with a Sengstaken-Blakemore tube. Critical points in the use of this device include endotracheal intubation to protect the airway, adequate inflation of the gastric balloon to avoid tube migration, and avoidance of esophageal compromise by monitoring pressure within the esophageal balloon.

If the decision is made to proceed with surgery for the unstable patient, an upper midline incision is used to enter the abdomen and explore for clues as to the etiology of bleeding. If none are discovered, the stomach is opened longitudinally and the abdomen, GE junction, pylorus, and duodenum are inspected for ulcers, Mallory-Weiss tears, neoplasms, arteriovenous malformations, or other pathology.

B. Stable Patient: Most patients with upper GI bleeding are stable enough to undergo elective endoscopy in either the emergency room or endoscopy suite. An Ewald tube placed into the stomach just before the endoscopy will efficiently remove blood clots with irrigation and suction, thereby improving visualization. Many sources of hemorrhage can be successfully diagnosed and treated with endoscopy. Subsequent admission to an intensive care unit is prudent for monitoring.

C. Esophageal Varices: Endoscopy with either sclerosis using 5% sodium morrhuate or rubber band ligation may control acute hemorrhage from esophageal varices. Other therapeutic options include vasopressin, octreotide, beta blockade, nitrates, and a Sengstaken-Blakemore tube (see Sect. A).

D. Esophagitis, Gastritis, and Duodenitis: These conditions are treated with intravenous and subsequently oral proton pump inhibitors together with antacid therapy either by mouth or through the NG tube. Tissue biopsy or cloe test for *Helicobacter pylori* should be performed and antibiotics added if positive. Additionally, any offending medications are discontinued. Diffuse uncontrolled bleeding from stress gastritis requiring operation is now rare. If necessary, the stomach is opened and bleeding points controlled with suture ligature and Gelfoam with thrombin and/or Surgicel. Truncal vagotomy will decrease mucosal blood flow and should be considered if these maneuvers fail. Partial or total gastrectomy may be required depending upon the predominant sites of bleeding, although this is rarely required.

E. Neoplasms: A variety of benign and malignant neoplasms of the esophagus, stomach, and duodenum may bleed. Bleeding is usually not torrential at presentation; lesions can be identified, biopsied, and electively resected after evaluation and staging is completed. Gastric leiomyomas may bleed massively and require emergent resection.

F. Gastric Ulcer: The endoscopic diagnosis of gastric ulcer is usually straightforward. It is important to biopsy to rule out malignancy and to diagnose *H. pylori* infection. Endoscopic therapeutic options to control bleeding include heater probe,
unipolar or bipolar cautery, laser therapy, epinephrine injection (1:10,000), and endoscopic clip placement. A gastroduodenal ulcer that has a viable vessel at its base has a 40–50% rebleeding rate. Rebleeding during the same hospitalization or a transfusion requirement of more than 6 units of red blood cells are considered indications for surgery. Surgical options for bleeding gastric ulcers include oversew, ulcer excision, and hemigastrectomy. Vagotomy is not required, but is essential in the treatment of duodenal and prepyloric gastric ulcers.

G. Duodenal Ulcer: The medical treatment of duodenal ulcer is similar to gastric ulcer management. Intravenous proton pump inhibitors are utilized and Helicobacter is treated if detected. Inability to endoscopically control bleeding or evidence of persistent or recurrent hemorrhage indicates the need for operation. Surgical treatment for bleeding duodenal ulcers includes dividing the pylorus longitudinally with prompt suture control of the bleeding vessel and/or the gastroduodenal artery. Vagotomy and pyloroplasty complete the procedure. A large pyloroduodenal ulcer may require antrectomy.

H. Mallory-Weiss Tear: Mallory-Weiss tears occur at the distal esophagus and extend to the gastric cardia. Often a history of intense vomiting precedes brisk arterial bleeding. Surgery is necessary if endoscopic control fails and consists of high gastrotomy and oversew of the bleeding point.

I. Vascular Malformations: A variety of vascular malformations may occur in the gastric mucosa. These include arteriovenous malformations, radiation-induced telangiectasia, congestive gastropathy (portal hypertension), antral ectasia (watermelon stomach), and Dieulafoy’s arterial malformation. All may present with upper tract bleeding, are relatively uncommon, and can be controlled with endoscopic hemostatic techniques. Surgery is reserved for treatment failure.

J. Duodenal Diverticula: Bleeding duodenal diverticulae present a special challenge in both diagnosis and therapy. They may be difficult to visualize on routine upper endoscopy and can be found in each portion of the duodenum. If endoscopic techniques fail to control hemorrhage, surgery with diverticulectomy may be required.

K. Hemobilia and Hemosuccus Pancreaticus: Unlocalized upper GI bleeding, blood in the duodenum without apparent source, or blood from the Ampulla of Vater may be due to hemobilia or hemosuccus pancreaticus. Liver trauma and indwelling stents may cause the former while inflammatory pseudocysts with erosion and pseudoaneurysm formation cause the latter. Angiography with embolization is diagnostic and therapeutic for both conditions.

L. Aortoduodenal Fistula: Patients with prior aortic graft surgery may present with upper or lower GI hemorrhage usually as a herald bleed followed by a more significant bleed. This history should prompt immediate evaluation for aortoduodenal fistula. Unstable patients should be taken promptly to the operating room. Stable patients can be evaluated with endoscopy, CT scanning, and aortic angiography. Pseudoaneurysm formation at the proximal anastomosis or visible graft in the third to fourth portion of the duodenum requires emergency surgery and extra anatomic arterial reconstruction.
Hematemesis

Assess airway
Start fluid resuscitation
Type and cross match
Labs-Hgb, platelet count, coagulation status
Nasogastric tube
Bladder catheter

A. Unstable patient

Go to surgery

1. Continue resuscitation
2. Upper endoscopy to r/o varices
3. Laparotomy if no varices
   gastrotomy, inspect stomach, duodenum, GE junction, oversee bleeding lesion

B. Stable patient

Upper endoscopy

I. Arterial malformations
J. Duodenal diverticulae
K. Hemobilia, hemosuccus pancreaticus
L. Aortoduodenal fistula

H. Mallory-Weiss tear
   -endoscopic control
   -surgery, oversew

G. Duodenal ulcer
   -treat for H. pylori
   -proton pump inhibitors

C. Varices
   -sclerosis
   -rubber banding

D. Esophagitis, gastroduodenitis
   -proton pump inhibitors
   -H. pylori
   -vagotomy if needed

E. Neoplasms
   -biopsy
   -consider for surgery

F. Gastric ulcers
   -biopsy,
   -treat for H. pylori
   -proton pump inhibitors

if endoscopy treatment fails, consider laparotomy with oversee or excision of ulcer, possible hemigastrectomy
A. Initial Assessment. The lower gastrointestinal (LGI) tract includes the small bowel distal to the ligament of Treitz and the colon, rectum, and anus. In children and adolescents, the differential diagnosis of LGI bleeding includes juvenile polyps, inflammatory bowel disease, intussusception, and Meckel’s diverticulum. In adults, the common causes include angiodysplasia, diverticulosis, neoplasm, inflammatory bowel disease, and anorectal sources such as hemorrhoids and fissures. “Hemorrhage” implies rapid bleeding often accompanied by hemodynamic instability.

The history in these patients should be targeted, yet thorough. Important points to assess are a history of prior bleeding episodes, coagulopathies, the use of nonsteroidal anti-inflammatory agents, alcohol abuse, cancer, liver or renal failure, and prior aortic operations. Patterns of symptoms are helpful to know, including duration of bleeding, frequency and degree of blood loss, recent bowel habit changes, and whether the patient has been experiencing emesis, dyspepsia, or hematemesis.

Examination needs to be quick and thorough. Attention should first be directed to the vital signs. Tachycardia, hypotension, and mental status changes that do not respond to initial resuscitation are worrisome. Previous scars, jaundice, and stigmata of chronic liver disease may direct the differential. Digital rectal exam and anoscopy and/or proctoscopy are mandatory to rule out an anorectal source. Placement of a nasogastric tube followed by gastric lavage is essential; bilious return without blood excludes an upper source with reasonable certainty.

Simultaneous with this initial assessment, aggressive fluid resuscitation should be instituted. Two large-bore peripheral intravenous catheters are placed, isotonic crystalloid (initially normal saline) fluid is administered, and a Foley catheter is placed to monitor urine output.

B. Assessing Bleeding Severity. There are three patterns of bleeding.

1. Occult blood loss often presents with iron-deficiency anemia or guaiac-positive stools. Work-up is usually nonemergent and consists of colonoscopy. If the patient has upper gastrointestinal symptoms, upper endoscopy is indicated as well.

2. Moderate/Intermittent blood loss presents with bloody stools and is often self-limited. These patients are usually stable enough to allow elective work-up as well, albeit during the same hospitalization. Colonoscopy is the preferred diagnostic test and will often reveal colonic, rectal, or anal pathology. Although blood acts as an excellent cathartic, a good mechanical prep will allow visualization of small angiodysplastic lesions.

3. Massive blood loss is active, profuse, and usually accompanied by hemodynamic instability. Security of airway, breathing, and circulation are a priority. Central venous monitoring is indicated for elderly patients, for those with preexisting cardiopulmonary disease, and for patients who remain unstable. Blood count, electrolyte levels, hepatic function, and coagulation profiles are all checked. Blood is typed and crossed. Vitamin K and/or FFP may be given for severe coagulopathies. Desmopressin may be indicated for patients with renal failure or for those with known platelet dysfunction.

C. The Unstable Patient. If the patient is massively hemorrhaging and is unstable despite infusion of large quantities of crystalloid solution and blood, the best course of action may be to perform urgent laparotomy. Since there was insufficient time to attempt localization, the surgeon should perform a subtotal colectomy rather than incorrectly assuming that bleeding has originated in a particular segment. After the induction of general anesthesia, upper endoscopy can be performed to rule out an upper source. The small bowel should be thoroughly inspected and palpated over its entire course to rule out a tumor or a Meckel’s diverticulum.

D. The Stable Patient. The patient who is actively bleeding but remains stable should undergo localization studies. Which test is performed first is controversial and will be determined by test availability and variation in institutional practices. Most clinicians would proceed with either a technetium-tagged RBC scan or angiography. Even when performed by
an experienced endoscopist, colonoscopy may be difficult, as active bleeding may impair visibility. If a bleeding source is found, the decision as to whether surgery is needed must be individualized. In general, if bleeding does not stop and the transfusion requirement is 6 units of packed red blood cells over 24 h or 10 units over 48 h, surgery is indicated. Rebleeding in the hospital after initially stopping is another indication.

E. Localizing the Site of Bleeding. The goal of the various diagnostic tests is to localize the bleeding site and potentially treat it in a nonsurgical way. If surgery is necessary, accurate localization of the bleeding site preoperatively will permit a segmental resection, sparing the patient the morbidity of a total colectomy. The severity and rate of bleeding will determine the sequence of tests ordered. Active, rapid, profuse bleeding may be manifested by tachycardia, hypotension, oliguria, and persistent hemodynamic instability despite aggressive fluid resuscitation. The frequency with which blood is passed cannot be used as a means of assessing the rate of bleeding.

1. $^{99}$Tc-tagged RBC scans are very sensitive and can detect bleeding at rates as slow as 0.1 ml/min. It can be repeated over 24–48 h if at first it is nondiagnostic. The scan is mostly used before angiography to determine if bleeding is still ongoing. Interpretation of the scan can be problematic, even if a blush or hot spot is seen. Identifying the exact location of the blush is difficult; for example, a blush noted in the right lower quadrant could be secondary to a cecal angiodysplastic lesion, a Meckel’s diverticulum, or a bleeding diverticulum in a redundant (floppy) sigmoid colon. Furthermore, if the scan is not done exactly at the time of bleeding, the tracer can be propelled aborally and an incorrect segment can be blamed.

2. Angiography is very specific and detects bleeding at rates of 0.5–1.0 ml/min. Since the bleeding is most likely from the right colon regardless of the cause, the superior mesenteric artery is injected first, followed by the inferior mesenteric and celiac arteries. A diverticular bleed is arterial and shows dye extravasation easily. Angiodysplasia will show a vascular tuft and an early filling and slowly emptying mesenteric vein. Extravasation of dye secondary to angiodysplasia does not occur as often as with diverticulosis. Angiography will help localize and may be used to treat the source of bleeding. Bleeding will stop in 80–90% of patients following intra-arterial selective vasopressin infusion (0.2 units/min), but often recurs once it is discontinued. Embolization of the bleeding vessel with absorbable gelatin strips or coils is useful in poor surgical candidates but can cause bowel wall necrosis.

3. Colonoscopy is technically difficult, if not impossible, in cases of massive hemorrhage. It is best utilized when the bleeding has slowed down or stopped. Colonoscopy can be therapeutic by injecting vasoconstricting agents or thermally ablating lesions.

4. Barium enema examinations should be avoided altogether since the contrast will interfere with angiography and colonoscopy.

F. Surgery. In the majority of instances, the bleeding will stop spontaneously. Patients may experience recurrent bleeding requiring readmission to the hospital; if so, the algorithm should be repeated with each episode. The small bowel should not be overlooked as a potential source of recurrent bleeding; unfortunately, this area of the gastrointestinal tract is difficult to examine. Options include an enteroclysis, or contrast study, or small bowel endoscopy with a long fiberoptic endoscope inserted orally. The small bowel may be examined intraoperatively using this latter technique as well.

If localization studies were successful in identifying the site of bleeding, segmented colectomy can be done with reasonable expectation that bleeding will not recur. Blind segmental resection of the right colon following unsuccessful localization incorrectly assumes the right colon is the source. While most bleeding does originate from the right side, blind right hemicolecction is associated with unacceptable rebleeding rates. Total abdominal colectomy is preferred in those instances where localization studies were nondiagnostic.
Lower Gastrointestinal Hemorrhage

A. Initial Assessment
   - History
   - Physical Exam
   - Proctoscopy
   - NG tube
   - Resuscitation

B. Assess severity

C. The Unstable Patient
   - If unstable despite resuscitation, perform urgent subtotal colectomy.

D. The Stable Patient

E. Localize Site
   - Angiography
   - Selective vasopressin embolization
   - Colonoscopy

F. Surgery
   - Do not overlook small bowel
   - Avoid segmental resection unless localization successful
   - Repeat algorithm for recurrent bleeding
A. Asymptomatic Cholelithiasis. A majority of individuals with cholelithiasis have few or no related symptoms. Approximately 2% of the asymptomatic population will develop biliary symptoms each year; only a small proportion of these will initially present with complications of cholelithiasis, such as acute cholecystitis, choledocholithiasis, or pancreatitis. Prophylactic or anticipatory cholecystectomy is not necessary for most gallstone patients provided they are truly asymptomatic. The clinician should evaluate symptoms carefully, however, since not all patients present with typical complaints.

There are circumstances that may be exceptions to the above and for which cholecystectomy should be considered on an individual basis: (1) Patients undergoing an unrelated major open abdominal operation. Cholecystectomy can usually be performed efficiently and safely in this setting and will prevent postoperative cholecystitis that can occur. This especially applies to patients undergoing bariatric surgery, to those requiring resection of large lengths of intestine that may result in short gut syndrome, and to diabetic patients. (2) Patients with anticipated long-term parenteral nutrition because they develop gallstones and sludge. Morbidity, mortality, and the need for emergency biliary operations are more frequent in these patients. (3) Women anticipating pregnancy. Biliary stasis associated with pregnancy may increase the risk of developing symptoms. Symptomatic and especially complicated cholelithiasis during pregnancy jeopardizes both the fetus and the mother. (4) Immunosuppressed transplant patients may develop serious septic biliary complications. Some centers routinely screen all potential kidney recipients for gallstones and recommend cholecystectomy prior to transplantation if stones are found. (5) Children, because of their higher lifetime risk of becoming symptomatic. (6) Individuals with known gallstones who will be without access to surgical care for prolonged periods of time. (7) Individuals (especially younger) who belong to populations at high risk for gallbladder cancer.

Current evidence does not support diabetes mellitus alone as an indication for cholecystectomy for patients with asymptomatic gallstones. However, substantial experience indicates that diabetic patients who develop complications of cholelithiasis or require emergency biliary operations have a higher associated morbidity and mortality than do nondiabetic individuals.

B. Symptomatic Cholelithiasis. Symptomatic cholelithiasis is one of the most common conditions that a general surgeon will encounter. Most patients who develop symptoms will continue to experience them and all are at risk of complications unless the gallbladder is removed. Symptomatic cholelithiasis includes a spectrum of clinical manifestations of varying severity. Some presentations can be managed medically with elective operation later; others are life-threatening and require emergent intervention. This algorithm provides a general structure for decision-making that will be modified according to specifics of the individual patient and the available local expertise.

C. Acute Cholecystitis. Acute cholecystitis occurs in 10–20% of individuals with symptomatic gallstones. From a purists’ standpoint, acute cholecystitis is a histologic diagnosis. From a practical perspective, it is strictly a clinical diagnosis. The typical presentation includes right upper quadrant abdominal pain and tenderness, nausea, vomiting, fever, and leukocytosis. Elevations of liver enzymes and amylase are not unusual even without common duct stones or pancreatitis. Ultrasound confirms gallstones and may demonstrate other characteristic (although not diagnostic) features, such as gallbladder distension, wall thickening, pericholecystic fluid, and a “sonographic Murphy’s sign.” A HIDA scan will fail to visualize the gallbladder but is not usually necessary. Initial medical management includes intravenous fluid and electrolyte resuscitation, bowel rest (NPO ± NG tube), intravenous antibiotics, and analgesics.

Ten percent of patients with acute cholecystitis warrant urgent cholecystectomy because of toxic manifestations. This applies particularly to diabetic or elderly patients who may progress rapidly to gangrene and who do not tolerate sepsis well. Operation can usually be performed after several hours of intravenous hydration and correction of metabolic abnormalities. Early cholecystectomy means that the patient is admitted to the hospital, treated medically (antibiotics, hydration), and operated upon
during the next available OR schedule. This is preferred for most patients with acute cholecystitis because it can be performed safely (and usually laparoscopically) and leads to the quickest resolution of the problem. Delayed cholecystectomy is performed a few weeks after signs and symptoms of acute cholecystitis have resolved and the patient has been discharged from the hospital. This may occasionally be indicated for patients with serious medical conditions that can be improved during the interim or for patients who refuse initial operation. Delayed operation risks recurrent biliary symptoms. Moreover, one-third of medically treated patients do not improve or worsen during their initial hospitalization. Cholecystectomy is often technically easier during the first few days of acute cholecystitis than during the ensuing week. The strategy of treating patients nonoperatively with the hypothesis that a delayed operation will more likely allow laparoscopic cholecystectomy to be performed has not been predictably successful.

D. Common Duct Stones. Approximately 10% of patients operated on for gallstone disease will have stones in the common bile duct at some time during their course. Choledocholithiasis is as frequent in patients with acute cholecystitis as in patients with chronic cholecystitis. A high probability (2–50%) of choledocholithiasis exists in patients with (1) obstructive jaundice, (2) a common bile duct stone seen on transabdominal ultrasound (note, however, that transabdominal ultrasound is not a sensitive test for identifying common bile duct stones), and possibly (3) elevated alkaline phosphatase and liver function tests (LFTs) greater than 2–3 times normal. Under these circumstances, preoperative evaluation of the bile duct by endoscopic retrograde cholangiography (ERC) is appropriate. If stones are found, endoscopic sphincterotomy (ES) is performed and stone removal is attempted.

Minor indicators of choledocholithiasis include pancreatitis, mild elevations of LFTs, a dilated common bile duct on abdominal ultrasound, or a history of prior jaundice. Patients in this group can usually proceed directly to laparoscopic cholecystectomy. Intraoperative imaging of the common bile duct by intraoperative cholangiography and/or intraoperative ultrasonography should be performed.

Patients with major or minor indicators of choledocholithiasis require intraoperative imaging of the bile duct when cholecystectomy is performed. This includes patients who have had preoperative ERC ± ES performed because of the possibility of missed or retained bile duct stones. Patients selected for preoperative ERC are at higher risk for common bile duct stones at the outset and the incidence of stones found during intraoperative evaluation is typically higher than it is for patients without preoperative ERC.

Intraoperative imaging is usually achieved with intraoperative cholangiography, which is most accurate when performed dynamically with fluoroscopy rather than with static x-ray images alone. Intraoperative (usually laparoscopic) ultrasonography is also highly accurate for the detection of common bile duct stones and is a complementary exam to intraoperative cholangiography.

If intraoperative imaging does not reveal common bile duct stones, then cholecystectomy is simply completed. If common bile duct stones are identified, they should be removed. Often this can be accomplished laparoscopically. If the laparoscopic approach is not successful or possible, then traditional open common bile duct exploration is recommended. Intraoperative ERC and ES can also successfully remove common bile duct stones but is a more logistically complex and less frequently employed alternative.

Patients who are septic merit special consideration. If the clinical picture suggests acute cholecystitis, prompt operation is indicated even if choledocholithiasis is also suspected. If the clinical picture suggests cholangitis as the primary problem, initial treatment is medical. Urgent bile duct decompression is indicated for patients with toxic cholangitis (hypotension, obtundation) who do not respond promptly to fluid resuscitation and antibiotics. This represents 5% of patients with cholangitis. Urgent decompression is usually accomplished endoscopically but can also be achieved by operation or by placement of percutaneous transhepatic catheters. A distinction between acute cholecystitis with choledocholithiasis and acute cholangitis without acute cholecystitis cannot always be made with certainty on clinical grounds.

E. Chronic Cholecystitis. Chronic cholecystitis can be diagnosed on the basis of symptoms of episodic postprandial right upper quadrant and epigastric pain with gallstones identified by ultrasonography. If major indicators of common bile duct stones are present (as described above), ERC is usually performed prior to cholecystectomy. If the surgeon is skilled in laparoscopic common bile duct exploration, then preoperative ERC is not necessary. Preoperative ERC is also unnecessary if the patient has indications for initial open cholecystectomy (i.e., extensive prior surgery) since an open common bile duct operation can be performed if choledocholithiasis is identified during the operation.

Patients with chronic cholecystitis and no indicators of choledocholithiasis are treated by elective (laparoscopic) cholecystectomy. In these patients, intraoperative imaging of the bile duct is selective according to the preference of the surgeon.

F. Biliary Pancreatitis. The diagnosis of biliary pancreatitis is usually made in patients with abdominal pain, gallstones, and hyperamylasemia. All patients with gallstones and elevated serum amylase or lipase do not truly, however, have pancreatitis. Pancreatic enzymes can be elevated with acute cholecystitis or choledocholithiasis even in the absence of pancreatic inflammation. Nonetheless, when biliary pancreatitis is suspected, initial management is medical. The overwhelming majority of patients with biliary pancreatitis have mild pancreatitis. Cholecystectomy is performed when the patient’s symptoms have improved (usually 24–72 h). Preoperative ERC is not necessary unless other more reliable indicators of choledocholithiasis, such as jaundice or cholangitis, exist. In patients with severe biliary pancreatitis, delayed cholecystectomy is carried out after medical management and resolution of the pancreatitis.
G. *Gallstone Ileus*. Gallstone ileus is intestinal obstruction caused by a sizeable gallstone that has entered the intestines through a fistula. The most common location of the fistula is between the gallbladder and the duodenum (cholecystoduodenal) and the most common site of obstruction is in the small intestine (ileum). A preoperative diagnosis can be made on the basis of clinical suspicion (elderly female patient, intestinal obstruction, no prior abdominal surgery) and radiologic detection of air in the biliary tract (pneumobilia). The primary goal of treatment is to relieve the intestinal obstruction. This is accomplished by laparotomy and removal of the intestinal stone(s) after the patient has been hydrated and properly resuscitated. Cholecystectomy, fistula closure, and evaluation of the common bile duct are reserved for patients who are stable and more fit and when the right upper quadrant dissection is not deemed unduly hazardous.
Cholelithiasis: Incidental and Symptomatic

A. Asymptomatic

Gall stones discovered

B. Symptomatic

Cholelithiasis ultrasound shows wall thickening, pericholecystic fluid, distended gall bladder

C. Acute Cholecystitis

Delayed surgery-controversial

D. Common Duct Stones?

High probability jaundice; common duct stone on ultrasound

E. Cholecystitis elective surgery (laparoscopic)

F. Biliary Pancreatitis delay surgery until pancreatitis has resolved

G. Gallstones ileus cholecystoduodenal fistula is most common

- Relieve obstruction with procedure of choice for the stable patient

Minor indicators: pancreatitis, dilated common bile duct, prior jaundice

Pre-operative ERC +/- sphincterotomy

Ultrasound

Elevated LFT's 2 - 3" times normal

Intra-operative cholangiogram or ultrasound

Surgery (see text)
A. Differential Diagnosis. A patient with jaundice usually presents with scleral icterus or yellow discoloration of the mucous membranes or skin. The jaundice is a clinical indication of hyperbilirubinemia. Hyperbilirubinemia can be the result of elevated levels of conjugated (measured as direct bilirubin) or unconjugated bilirubin. The initial laboratory tests obtained should include a bilirubin with measurement of the direct bilirubin fraction. If fractionating is not available, an elevated urine bilirubin level will indicate conjugated hyperbilirubinemia, as unconjugated bilirubin cannot be filtered by the kidney. If the direct fraction is low then unconjugated hyperbilirubinemia is diagnosed; causes include hemolysis, hematoma reabsorption, and Gilbert’s disease. An elevated direct fraction indicates conjugated hyperbilirubinemia. Pain, previous biliary operations, and ingestion of hepatotoxic drugs are important elements in the history of patients with conjugated hyperbilirubinemia. Conjugated hyperbilirubinemia can be caused by inability of the hepatocytes to excrete conjugated bilirubin or obstruction in the biliary system. Serum transaminases will be elevated in patients with hepatocellular dysfunction; causes include viral and alcoholic hepatitis, sepsis, parenteral nutrition, etc. Elevations in the coagulation profile can also indicate hepatic parenchymal disease. Alkaline phosphatase is elevated in a malignant segmental ductal obstruction (rather than common duct) in which case the bilirubin is usually normal. Low levels of urinary urobilogen are also indicative of obstructive jaundice.

B. Evaluation. If the jaundice appears to be due to an obstructive process, right upper quadrant ultrasound is the first diagnostic test obtained. If the ultrasound indicates gallstones or common bile duct stones then an endoscopic retrograde cholangiopancreatography (ERCP) is indicated and will evaluate the distal common bile duct and the pancreatic duct. If common duct stones are found then sphincterotomy should be performed to facilitate internal drainage and passage of the stones. Since the gallbladder is usually the source of the stones, a laparoscopic or open cholecystectomy should be performed if the patient is an acceptable surgical candidate. If the endoscopist could not extract the stones, a laparoscopic or open common bile duct exploration is performed to remove the stones and the gall bladder.

If no stones are found and a stricture is present, brushings for cytology should be performed to rule out malignancy. Benign cytology does not completely rule out cancer; however, options include stenting or a surgical bypass such as a choledochoduodenostomy or choledochojejunostomy. A stricture can be further evaluated with computerized tomographic (CT) scans, looking for soft tissue fullness in the vicinity of the stricture, enlarged lymph nodes, or space-occupying lesions in the liver parenchyma. These findings suggest cancer and possible metastases.

When the common duct is dilated for its entire length and ERCP does not show stones or clear evidence of a stricture, one should suspect a periampullary or pancreatic cancer. A CT scan may help determine resectability; if there are no metastases and the mesenteric vessels are not encased, the tumor is considered resectable. Further evaluation can be performed with endoscopic ultrasound. Portal vein invasion is not necessarily a contraindication for surgical resection. A resectable mass should be removed by pancreaticoduodenectomy (Whipple procedure). If the lesion is not resected, the jaundice may be treated with stenting or surgical bypass.

If only intrahepatic biliary dilatation is found by ultrasound, again a CT scan should be performed to rule out porta hepatitis lymphadenopathy or liver metastases. A cholangiocarcinoma at the bifurcation should be suspected in these instances. In the absence of metastases, a percutaneous transhepatic cholangiogram (PTC) should be performed to determine the level of biliary obstruction, the degree of intrahepatic involvement, and evidence of an intraductal lesion. An angiogram to rule out vascular invasion can be helpful in determining resectability. Operative resection of a cholangiocarcinoma may include partial hepatectomy and Roux-en-Y hepaticojejunostomy. If hepatic resection is not possible, PTC can be performed to relieve biliary obstruction.
A. Differential Diagnosis
hyperbilirubinemia, fractionation essential

conjugated hyperbilirubinemia
- hepatocyte dysfunction
- hepatitis
- sepsis
- parenteral nutrition
- familial

duct obstruction
- cholelithiasis +/- biliary dilatation
  - ERCP
  - stones
  - stricture
  - sphincterotomy followed by cholecystectomy
- choledocholithiasis

unconjugated hyperbilirubinemia
- hemolysis
- hematoma
- Gilbert's disease

B. Evaluation
RUQ ultrasound

cholelithiasis
- biliary dilatation
- ERCP
- brushings
- CT scans
- internal drainage, bypass or resection if cancer suspected

no cholelithiasis
- choledocholithiasis
- CT scan
- ERCP
- evaluate for possible resection

hemolytic jaundice
- hematoma
- hemolysis

Gilbert's disease
- familial hyperbilirubinemia

Parenteral nutrition
- hepatocyte dysfunction
- hepatitis
- sepsis
A. Introduction:
Risk Factors: Most liver masses are asymptomatic and found incidentally during clinical examination, imaging studies, or surgery. When symptomatic they may present with pain, fever, jaundice, weight loss, or rupture. Risk factors for the various liver tumors have been identified and studied. Oral contraceptive use is associated with hepatocellular adenoma. Cirrhosis and chronic hepatitis predispose to hepatocellular carcinoma. Carcinogens associated with liver neoplasms include thorotrast, aflatoxin, vinyl chloride, and arsenic. Physical examination is often negative. Evidence of cirrhosis or primary tumors in other organs (large bowel, breast, and lung) should be sought for. Abnormalities of liver function tests are inconsistent and nonspecific. If serum transaminases are markedly abnormal, a hepatitis panel should be obtained. Tumor markers, such as alfa-fetoprotein (AFP) and carcinoembryonic antigen, have a role in the diagnosis and postoperative follow-up of hepatocellular carcinoma, cholangiocarcinoma, and metastatic colon carcinoma. Imaging studies are complementary, rather than competitive, and provide useful information in diagnosis and in evaluating resectability.

B. Types of Masses:
1. Cavernous hemangioma is the most common benign tumor of the liver. Most patients are asymptomatic. Symptoms seen with large masses include pain, early satiety, nausea, vomiting, and fever. Although bleeding is rare, thrombosis of a hemangioma can lead to consumption coagulopathy. Hapatomegaly or a bruit may be elicited. Needle biopsy is contraindicated because of the risk of hemorrhage. Characteristic computerized tomographic (CT) scan features are a precontrast hypodense mass followed by postcontrast peripheral enhancement, centripetal filling, and delayed emptying. A tagged red blood cell scan confirms the diagnosis with a “hot spot” and obviates the need for an angiogram. Symptomatic cavernous hemangiomas are enucleated and the feeding artery is ligated.
2. Hepatocellular adenoma is the most common benign liver tumor in young women. Long-term oral contraceptive use is the most important predisposing factor. Up to 25% of males with hepatocellular adenoma give a history of androgen use. Abdominal pain occurs in half the patients. Patients may present emergently with rupture, bleeding, and shock. Liver function tests are often abnormal but AFP is not elevated. CT scan shows areas of hemorrhage and necrosis. Magnetic resonance imaging (MRI) shows hyperintense areas of hemorrhage or necrosis on T2-weighted images and gadolinium contrast enhances the tumor. MRI with mangafodipir or radionuclide liver scan confirms presence of functional hepatocytes. The risk of seeding from a malignant lesion discourages use of needle biopsy. Asymptomatic hepatocellular adenomas less than 6 cm in size may be observed. Women on birth control pills should practice alternative methods of contraception. Resection is indicated if the mass is symptomatic, greater than 6 cm in size, shows progression in size with serial CT scans, or is associated with elevated serum AFP levels.
3. Focal nodular hyperplasia occurs in both sexes and all age groups. The incidence is highest in young women but there is no association with oral contraceptive use. Focal nodular hyperplasia is thought to arise from a hyperplastic response around a vascular malformation. The lesions are usually asymptomatic and are detected incidentally by ultrasound or CT scan. Bleeding complications and malignant transformation are rare. Symptomatic patients complain of abdominal discomfort. Gross characteristics include lobulation and a central stellate scar. Elevations in liver function tests are seen in less than 20% of patients. The hypervascular central scar is seen in delayed contrast-enhanced CT images as a hyperdense center. Increased Kupffer cell uptake in radionucleotide liver scans appears as a “hot spot” and is the diagnostic procedure of choice. Asymptomatic focal nodular hyperplasia should be observed, while lesions with substantial symptoms are resected.
4. Cystic liver masses: Solitary congenital hepatic cysts are usually asymptomatic and do not need surgery. The presence of internal echoes on ultrasound raises the suspicion of infection or malignancy. Symptomatic cysts are treated...
with laparoscopic unroofing. *Polycystic liver disease* occurs with polycystic kidney and may be associated with intracranial berry aneurysms. When symptomatic, they are treated surgically with excision or unroofing. Combined liver and kidney transplantation may be required in some individuals. *Pyogenic liver abscess* arises as a consequence of other infections such as cholangitis, gastrointestinal sepsis, surgery, or bacterial endocarditis. Cirrhosis, diabetes, HIV, and metastases are predisposing factors. The typical presentation includes right upper quadrant pain, fever, chills, and weight loss. Ultrasound-guided external drainage and intravenous antibiotics for 2 weeks followed by oral antibiotics for 4 weeks is adequate treatment in most cases. Laparotomy and drainage may be required in selected cases. *Amebic liver abscess* resembles pyogenic liver abscess but is caused by *Entamoeba histolytica*. A single, large abscess, in the dome of the right lobe of the liver, is the typical presentation. A history of travel to an endemic area and a history of alcoholism are important predisposing factors. Serologic tests, such as ELISA and immunofluorescence tests, are available to confirm the diagnosis. Most amebic liver abscesses resolve without drainage when treated with metronidazole 750 mg three times daily. Large left liver lobe amebic abscesses are an indication for therapeutic drainage due to the imminent danger of fatal intra-pericardial rupture. *Hydatid cysts of the liver* (echinococcal cysts) arise from infestation by the tapeworm *Echinococcus granulosus*. The dog is the primary host that spreads the disease by the fecal-oral route to intermediate hosts such as sheep, cattle, and humans. Less often the organ involved is the brain, lung, spleen, or bone. Symptomatic patients experience abdominal pain or jaundice. A plain radiograph of the abdomen may show "egg-shell calcification" of the hydatid cyst. Serologic tests are positive in ~80% of cases. Ultrasound and CT scan are the best diagnostic imaging tests. Surgical treatment is required to excise the cysts under controlled circumstances, avoiding spillage. Mebendazole given before and after surgery reduces recurrence.

5. **Primary hepatic malignancies.** Predisposing factors for *hepatocellular carcinoma* include hepatitis B and C, cirrhosis, and exposure to hepatotoxins (aflatoxin, thorotrast). When symptomatic, painful hepatomegaly may be associated with anorexia and weight loss. Hemorrhage causes sudden, severe pain or produces shock. AFP levels are elevated (also elevated in hepatitis, cirrhosis, massive hepatic necrosis, pregnancy, and yolk sac tumors). The triad of liver mass, positive hepatitis serology, and high AFP levels is diagnostic of hepatocellular carcinoma. MRI evaluates vascular involvement and extrahepatic disease. Angiography detects variations in vascular anatomy. Intraoperative ultrasound is the most sensitive method of evaluating vascular involvement to decide extent of surgical resection. As cirrhotics do not tolerate major hepatic resections, limited peripheral wedge resections, radiofrequency ablation, or cryotherapy for central lesions are options. Chemotherapy is indicated for lesions greater than 5 cm and for multicentric lesions. For noncirrhotic patients, curative liver resections should be performed whenever possible. Surgical resection is associated with an operative mortality of 5–10% and a 5-year survival rate of about 25%. Intrahepatic *cholangiocarcinomas* are rare and are classified into three groups: (1) peripheral cholangiocarcinomas, (2) cholangiocarcinomas that arise from the right or left hepatic duct, and (3) hilar cholangiocarcinomas that arise at the junction of the common, left and right hepatic ducts (Klatskin’s tumor). Predisposing factors include ulcerative colitis, choledochal cysts, Caroli’s disease, sclerosing cholangitis, and clonorchis infestations. Klatskin’s tumors present early because of the biliary tract obstruction. Serum AFP levels are normal while serum carcinoembryonic antigen levels are elevated. Angiogram is required to ascertain portal vein involvement and invasion of the contralateral hepatic artery. Further evaluation and treatment of intrahepatic cholangiocarcinoma is similar to that of hepatocellular carcinoma. *Angiosarcoma* is the most common primary hepatic sarcoma. Exposure to chemical carcinogens such as vinyl chloride, arsenic, and thorotrast is strongly implicated in its pathogenesis, while therapeutic radiation and hemochromatosis have been identified as predisposing factors. Angiosarcoma is a nonencapsulated multinodular lesion that is prone to intralesional hemorrhage or fatal hemoperitoneum. On ultrasound examination, intralobal hemorrhage gives it a heterogenous echogenicity. Contrast-enhanced CT scans show a very vascular tumor and the centripetal enhancement pattern mimics that of hemangioma. Percutaneous needle biopsy for a tissue diagnosis followed by chemotherapy is the treatment for nonresectable cases. Angiosarcomas have a high degree of local invasion and metastases leading to early death.

6. **Secondary hepatic malignancies.** Metastatic cancers of the liver are more common than primary cancers in North America. The main primary malignancies associated with liver metastases are colorectal cancer, breast cancer, lung cancer, melanomas, lymphomas, renal cell carcinoma, pancreatic islet cell tumors, and carcinoid. Resection, when possible, is indicated for primary colonic carcinoma, symptomatic carcinoid, and renal cell carcinoma.
A. Risk Factors for Cancer
Oral contraceptive vs. cirrhosis, chronic hepatitis carcinogens

Physical Exam
Rule out primary tumor elsewhere

Laboratory Tests
Liver Chemistries
Tumor markers (alpha fetoprotein, CEA)

Imaging Studies
Stigmata of chronic liver disease

Cystic by ultrasound

Solitary congenital hepatic cyst
Polycystic liver disease
Pyogenic liver abscess
Amebic liver abscess
Hydatid liver cyst

Solid by ultrasound, CT

Peripheral enhancement

Centripetal filling
Cavernous hemangioma

Heterogenous

MRI Liver Scan

(+)
Focal nodular hyperplasia

(-)

Adenoma
Carcinoma
cholangiocarcinoma
Peptic ulcer disease (PUD) affects 10% of the population in the United States. Ulceration of the lining of the gastrointestinal (GI) tract develops when the natural balance between gastric acid production and mucosal defense mechanisms is altered. Recent association of Helicobacter pylori (H. pylori) infection with PUD and the availability of endoscopy have revolutionized our ability to diagnose and treat the disease in the majority of patients. Other contributing factors include nonsteroidal anti-inflammatory drug (NSAID) use, cancer, steroid use, tobacco use, severe physiologic stress from burns (Curling’s ulcers), head injury (Cushing’s ulcers), surgery or critical illness, and hypersecretory conditions such as a gastrinoma or antral G-cell hyperplasia.

A. Symptoms in patients with PUD can, but do not necessarily, differ based on the location of the ulcer. Both gastric and duodenal ulcers usually present with epigastric abdominal pain that may be relieved by food or antacids while pain from a duodenal ulcer can radiate to the back. Gastric ulcers may be more symptomatic in the morning or fasting state when there is a low intragastric pH and relieved by food because of an increased pH by dilution. Duodenal ulcers are said to be aggravated by eating as the pH drops in the duodenum when the stomach empties. Hematemesis, melena, or bright red blood per rectum can be manifestations of bleeding from either type of ulcer. Those with a gastric ulcer near the pylorus may present with nausea and vomiting from a gastric outlet obstruction secondary to edema or scarring. Special attention should be paid to signs and symptoms such as weight loss, melena, hematemesis, anemia, vomiting, dysphagia, or a palpable abdominal mass, which may be indicative of a malignant process.

B. Physical examination should focus on vital signs and a thorough abdominal exam. Patients with bleeding may show signs of anemia, shock, or may simply have blood detectable in their stool. Abdominal findings can range from a normal exam to an acute abdomen.

C. Diagnostic testing for those with suspected PUD differs depending on the manner in which the patient presents. Typically, much of the workup is done as an outpatient. In those less than 45 years old without NSAID use or gastroesophageal reflux disease (GERD), or signs or symptoms listed above, noninvasive testing for H. pylori, such as a urea breath test or serologic examination for anti-H. pylori IgG, should be performed. If positive, proceed directly to first-line therapy for H. pylori. In the presence of symptoms or persistence despite first-line treatment, some form of imaging is needed. Upper GI studies have given way to endoscopy with biopsy as the diagnostic study of choice. Those with refractory or recurrent PUD should be evaluated for a gastrinoma by obtaining a fasting gastrin level while off antisecretory medication for at least 2 weeks.

In the acute setting, initial workup should include routine laboratory tests, particularly a complete blood count (CBC) to evaluate for anemia or leukocytosis and electrolyte abnormalities. An upright chest X-ray may demonstrate free air in the case of perforated PUD and the need for emergent surgical intervention. If there is evidence of bleeding (anemia, hematemesis, melena, or bright red blood per rectum (BRBPR), urgent upper endoscopy should be performed.

D. Triple therapy for H. pylori was originally described to consist of bismuth, metronidazole, and tetracycline (BMT). This resulted in eradication rates of greater than 95%. Newer regimen of proton pump inhibitors (PPIs) and antibiotic combinations result in similar eradication rates with easier dosing schedules. Patients should all be consulted on lifestyle modifications to eliminate offending substances, such as alcohol, tobacco use, caffeine, as well as healthy eating habits. Patients with NSAID-induced PUD should avoid the offending agents.

Prior to the introduction of antisecretory medications and H. pylori treatment, operations for PUD were performed nearly as frequently as those for gallbladder disease. Contemporary surgical intervention for PUD is reserved for the complications of the disease, namely, bleeding, obstruction, perforation, or the inability to rule out cancer. Acid reduction procedures are only rarely performed for intractable disease.
E. Operations for bleeding typically involve duodenal ulcers and consist of duodenotomy with oversewing of the bleeding ulcer. Gastric outlet obstruction from chronic PUD can be dealt with by resection or drainage procedures. Perforation of duodenal ulcers is commonly addressed with omental patch (Graham patch). Addition of an antisecretory procedure in the form of a highly selective vagotomy (HSV) is advocated by many in the surgical management of complications of PUD.

Chronic nonhealing gastric ulcers always raise suspicion for malignancy. Management strategies should include resection, either local wedge or formal oncologic procedure as indicated by the level of suspicion and patient condition. An antisecretory neurotomy, HSV or vagotomy with drainage procedure, is recommended in this setting.

Most, if not all, of the described surgeries for PUD have been performed laparoscopically, which offers patient advantages in the form of fewer wound complications and less postoperative pain. The laparoscopic approach should be encouraged where levels of expertise and comfort exist.
Peptic Ulcer Disease

A. Signs and Symptoms
1.) Pain
2.) Weight loss
3.) Melena
4.) Hematemesis
5.) Anemia
6.) Vomiting
7.) Dysphagia
8.) Mass
9.) Early Satiety

B. Physical Exam
1.) Vital Signs
2.) Abdominal Exam
3.) Stool Guaiac

C. Diagnostic Studies
1.) H. Pylori Testing
   - Urea breath test
   - Serum studies
2.) CBC
3.) CXR
4.) Endoscopy

D. Treatment
1.) (+) H. Pylori
   Triple Therapy
   PPI + 2 Antibiotics

E. Surgical / Indications
1.) Bleeding not amenable to EGD
2.) Perforation
3.) Obstruction
4.) Unable to rule out malignancy
5.) Intractable

Duodenal
1.) Bleeding → oversew with or without HSV
2.) Perforation → omental patch with or without HSV
3.) Obstruction → resection or diversion with or without HSV or TV with Drainage
4.) Intractable → HSV or TV with drainage

Gastric
1.) local wedge resection with HSV or TV with drainage
2.) Oncologic resection with HSV or TV with drainage

Fails
Serum Gastrin levels
A. Dysphagia, or difficulty swallowing, can be very distressful to a patient. A thorough history and physical examination may help identify the cause, which may include anatomic, physiologic, or neurologic problems. A differential diagnosis is provided in Table 52.1.

B. A barium swallow should be the first diagnostic test. Many physicians do not proceed initially with an upper endoscopy until the esophageal anatomy has been delineated with radiographic means.

C. Esophageal diverticula are categorized as either pulsion or traction diverticula. Traction diverticula occur in the middle third of the esophagus and are the result of an inflammatory process, such as tuberculosis, histoplasmosis, or toxoplasmosis, within the mediastinum. Treatment is medical, rather than surgical. Pulsion diverticula that occur in the upper third of the esophagus are called “Zenker’s diverticula.” These are false diverticula caused by uncoordinated swallowing and they form at a weak point just distal to the cricopharyngeus muscle. The diverticulum is approached through a left neck incision and will be found posterior to the esophagus. The carotid sheath and recurrent laryngeal nerve are retracted away from the field. A myotomy is performed and extended distally onto the esophagus for several centimeters. Small diverticula may be left alone; larger ones are generally resected usually with the help of stapling devices. Diverticulopexy is an alternative. If symptomatic, epiphrenic diverticula are excised through a left thoracotomy and an esophageal myotomy is performed distal to the excision site.

D. Achalasia is caused by a failure of the lower esophageal sphincter to relax with swallowing and deficient progressive peristalsis within the esophagus which is present with normal swallowing. Forceful balloon dilatation may be used to treat the symptoms of early disease; however, only 65% of patients will experience long-term relief. If dilatation fails, the treatment of choice is distal esophageal myotomy combined with an antireflux procedure. The myotomy can be performed through a left thoracotomy (Heller operation), transabdominally or laparoscopically. A successful myotomy will divide both longitudinal and circular muscles of the esophagus and is extended through the lower esophageal sphincter onto the stomach. The incidence of gastroesophageal reflux disease (GERD) after myotomy can be as high as 50%; therefore, the myotomy should be accompanied by an antireflux procedure (usually a partial fundoplication, i.e., Toupet or Dor).

E. If the barium swallow reveals an obstructing lesion or stenosis, endoscopy with biopsy is indicated. If the lesion is benign and the obstruction is thought to be a peptic stricture from reflux or a stricture from previous ingestion of a caustic solution, dilatation should be performed. Resection is needed only when the disease is refractory to dilatation; the colon and the stomach have both been used as replacements for the esophagus. If the biopsy reveals a benign cyst or leiomyoma, excision should be carried out through a thoracic approach. Esophageal webs and rings are treated with endoscopic dilatation or ablative therapy; excision is reserved for those instances when conservative measures have failed.

F. If cancer is diagnosed, a thorough metastatic survey including computerized tomography (CT) of the neck, chest, and abdomen should be performed. Endoscopic ultrasound (EUS) may assess for depth of tumor invasion and for periesophageal or celiac axis lymph node metastases. In the absence of metastases to distant sites or invasion into adjacent structures, preoperative multimodal therapy with radiation and chemotherapy followed by resection is the preferred approach. Most patients present with advanced disease and the goal then becomes palliation; this can be achieved with an endoscopically placed esophageal stent as well as with laser ablative therapy. Bypass
procedures, that is, substernal colon interposition, can be done if the esophagus is completely obstructed.

G. When GERD is detected by barium swallow, endoscopy should follow to determine the extent of esophagitis. Manometry is done to confirm a laxity of the LES (normal resting pressure is 25 mmHg) and to rule out spasm or motility disorders of the proximal esophagus. A pH study is reserved for those patients with symptoms suggestive of reflux but have normal endoscopic and manometric findings and is the most precise measure of the presence of acid in the esophageal lumen. Normal acid exposure in the esophagus is about 5%, but in patients with erosive esophagitis, the exposure can be as high as 12% of the recording duration. If these three tests confirm gastroesophageal reflux, the initial medical treatment should incorporate lifestyle modifications (weight loss and avoidance of caffeine, alcohol, and nicotine) and the use of a proton pump inhibitor. If this fails, then a surgical fundoplication should be performed. The laparoscopic fundoplication is now commonly performed and provides results comparable to the open technique.

H. When the barium swallow is normal, endoscopy should be performed to rule out a false-negative study. If endoscopy is also normal, manometry should be performed to rule out diffuse spasm, which is treated with calcium channel blockers, and if this fails, a total esophageal myotomy should be performed. Scleroderma can also be detected by endoscopy and manometry and is treated medically with steroids and proton pump inhibitors. If all studies are normal, it is unlikely that a surgical disease is present and one should then consider pharyngeal dysphagia from a neuromuscular or vascular process.

<table>
<thead>
<tr>
<th>Differential diagnosis of dysphagia</th>
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<tr>
<td>I. Anatomic</td>
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<tr>
<td>Leiomyoma</td>
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<td>Esophageal web or ring</td>
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<tr>
<td>Peptic stricture</td>
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<td>Carcinoma</td>
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<td>II. Physiologic</td>
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<tr>
<td>Zenker’s diverticulum</td>
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<td>Esophageal traction diverticulum</td>
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<td>Epiphrenic diverticula</td>
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<td>Achalasia</td>
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<td>Diffuse esophageal spasm</td>
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<td>Scleroderma</td>
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<tr>
<td>III. Neurologic</td>
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<tr>
<td>Chagas disease</td>
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<td>Central deficit/Pharyngeal dysphagia</td>
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Table 52.1. Differential diagnosis of dysphagia.
A. Amount of Bleeding. Assessing a complaint of rectal bleeding requires taking a thorough history and concentrating on the character, amount, and frequency of the bleeding. The reader is encouraged to review the chapter on lower gastrointestinal hemorrhage. This chapter will focus on anorectal causes of bleeding that is mild and doesn’t compromise hemodynamic stability. Physical examination with particular attention to the presence of hemorrhoids, fissures, rectal masses, or signs of trauma must be performed at the initial visit. Rigid proctoscopy is used to assess the distal 25 cm of the colon, the rectum, and the anus. It is capable of detecting the benign conditions mentioned.

If a patient describes seeing blood only on the toilet paper or blood dripping into the toilet bowl, then it is most likely coming from the anal outlet. If, however, a patient is complaining of bright red blood either mixed with or on the outside of the stool, the presentation is more consistent with a proximal source of bleeding. While in either case a flexible sigmoidoscopy can be used as an initial diagnostic modality, the latter patient will require a full colonoscopy regardless of sigmoidoscopic findings to rule out proximal pathology.

B. Anorectal Source. For a patient with suspected anorectal source of bleeding and confirmed hemorrhoids by flexible sigmoidoscopy, appropriate therapy must be initiated. However, if the symptoms of bleeding persist despite apparent resolution of hemorrhoids, then a colonoscopy should be performed. Colonoscopy should be performed anyway in patients more than age 50 for cancer screening. If the initial sigmoidoscopy fails to reveal the source of bleeding or if bleeding is accompanied by change in bowel habits, a colonoscopy or a barium enema is the next diagnostic test. If the sigmoidoscopy reveals a polyp or cancer, the entire colon must be examined to reliably rule out a synchronous lesion in the proximal bowel.

C. Colonoscopy. The decision to perform colonoscopy versus double contrast barium enema depends on the physician’s preference. Colonoscopy is favored by most because, despite being more costly and invasive than the barium enema, it can detect lesions less than 1 cm. It can also be used as a therapeutic modality for lesions amenable to endoscopic removal—those with an adequate stalk and complete exposure. Sessile polyps may require polypectomy in piecemeal fashion or just a biopsy to obtain tissue diagnosis and direct further therapy if polypectomy is not feasible. Complications of endoscopic polypectomy are perforation and hemorrhage from the polypectomy site.

Villous adenomas have the highest chance of containing occult cancer followed by tubulovillous adenomas and finally tubular adenomas. Risk of harboring cancer is also related to the size of the polyp—half of all polyps greater than 2 cm will contain carcinoma, while only 1% of polyps less than 1 cm will do so.

No further treatment is needed for a pedunculated polyp with cancer if the margin is clear, there is no venous or lymphatic invasion, and there is well differentiation. Sessile polyps removed in a piecemeal fashion and found to contain cancer are treated with colectomy because the margin of resection is not reliable and the probability of hematogenous or lymphatic spread is high due to the absence of a stalk.

D. Fecal Occult Blood Test. If blood is detected during a fecal occult blood test (FOBT), one must make sure that the test was performed correctly. The patient must avoid NSAIDs, red meat, and all foods containing peroxidase-like activity for 3 days prior to FOBT. If any of the above conditions have been violated, the test must be repeated to ensure maximum sensitivity and specificity. If the FOBT is still positive, the entire colon must be evaluated via colonoscopy or flexible sigmoidoscopy and barium enema. If the workup is negative and the patient remains asymptomatic, FOBT should be repeated in 1 year. Repeat positive FOBT mandates repeat colonoscopy. If the colonoscopy remains normal, then a UGI source of bleeding must be sought via EGD or barium swallow.

E. CT Colography. A newer technique of evaluating the colon is CT colonography or “virtual colonoscopy” which entails 3D computer-generated reconstruction of images of the colon obtained by a contrast-enhanced helical CT scan. At the present time, it is not a replacement for conventional colonoscopy; however, it is suited for patients who did not tolerate colonoscopy because of the tortuous colonic anatomy, excessive pain, or both. For CT colonography, a bowel cleansing is still required.
Rectal Bleeding

A. Amount
- massive
  - See Chapter on LGI Hemorrhage

B. Anorectal Source
- Patient >50 yrs
  - Treat anorectal source
  - Bleeding persists

C. Complete colonic evaluation
- Colonoscopy
- Flex Sig with barium enema

D. Positive Fecal Occult Blood
- Test correctly performed?
  - Yes
    - Is endoscopic removal adequate treatment?
      - Yes
        - Cancer Present
          - Surgery
      - No
        - No
    - No
      - Advanced cancer
        - Surgery
A. History: Approximately 5 million people in the United States have an inguinal hernia. Seven hundred fifty thousand inguinal hernia repairs are performed annually in this country; 75,000 of these repairs are done for recurrent hernias. The history is usually that of a groin bulge or pain occurring during straining or exercise. Approximately 10% of men during their lifetime will be diagnosed with an inguinal hernia. Patients are typically referred to a surgeon by a primary care physician who has noted an asymptomatic hernia on physical examination. Alternatively, patients may seek care because of persistent pain or discomfort which limit physical activities. Inguinal hernias are more common in men than in women (20:1 ratio).

B. Physical Examination: The physical exam is of paramount importance. The most common physical finding is a palpable soft bulge produced by coughing or Valsalva which is diagnostic. Over 50% of patients will have a visible asymmetry or bulge noted on inspection of both groins with the patient standing. Anatomically, inguinal hernias are classified as direct, indirect, or femoral (Table 54.1). Indirect and direct hernias cannot be differentiated on physical exam, as both present as bulges at the external ring. Femoral hernias are palpable in the upper medial thigh at the outlet of the femoral canal.

C. Unilateral Hernia: The vast majority of inguinal hernias are reducible; the risk of incarceration or strangulation is 1–2% over a lifetime. Although uncommon, incarceration or strangulation is a serious event which can lead to significant morbidity. Over time, inguinal hernias will increase in size, cause pain, become cosmetically unappealing, and be more difficult to repair. For these reasons, most surgeons agree that inguinal hernias should be repaired unless comorbid conditions preclude surgery. Almost all unilateral hernia repairs can be performed using local anesthesia and intravenous sedation. Mesh repairs have a recurrence rate of 1%, are associated with less postoperative pain, and have faster recovery rates because of the tension-free nature of the repair.

During the 1990s, laparoscopy emerged as a safe technique for inguinal hernia repair but is associated with increased instrumentation cost and for the most part requires a general anesthetic. In contrast, the mesh-plug hernioplasty, the Lichtenstein flat mesh, and the Kugel preperitoneal patch can be performed under local anesthesia (Table 54.2). The mesh-plug hernioplasty has the quickest recovery, the least amount of postoperative pain, and is the most simple technique to master. For these reasons, the mesh-plug hernioplasty is rapidly becoming the gold standard for unilateral inguinal hernias.

Incarcerated unilateral hernias require emergent repair; this is usually performed under general or spinal anesthesia. The contents of the hernia sac must be examined for possible ischemic intestine and a laparotomy or laparoscopy may be necessary if the bowel has retracted internally and viability is still in question. Because of the risk of infection, mesh should not be inserted in this situation.

D. Recurrent Hernia: For a recurrent hernia, it is important to determine whether a mesh or non-mesh repair was previously performed. For the latter, we explore the groin through the previous incision and insert a mesh plug through the recurrent defect. This repair can be done using local anesthesia and intravenous sedation with minimal trauma to the spermatic cord, vessels, and local nerves. Another possibility would be to perform a preperitoneal Kugel patch. This repair can also be performed using local anesthesia and intravenous sedation but has a steeper learning curve. For previously repaired mesh recurrences and multiple recurrent hernias, the preferred approach is a laparoscopic preperitoneal mesh repair.

E. Bilateral Hernias: Ten percent of men with inguinal hernias also have contralateral hernias at presentation. Before mesh repairs became the gold standard, staged repairs were favored. However, since mesh insertion provides a tension-free repair, recurrence rates and speed of recovery are not compromised by a simultaneous approach. Bilateral repairs
can be performed by either an open mesh plug or a laparoscopic preperitoneal repair; both approaches require general anesthesia. In the obese population a laparoscopic approach would be preferred for bilateral hernias to limit the size of incisions and amount of tissue dissection.

**F. Persistent Pain.** If a patient complains of groin pain, yet a hernia is not palpable, one should reexamine the patient in 3 months to make sure that a hernia is not missed. The physical examination can be supplemented with an ultrasound examination while the patient is standing and coughing or straining. Another possible diagnostic consideration includes irritation of the ilioinguinal, iliohypogastric, or genital-femoral nerves. A steroid injection of Kenalog in 10 cc of 1% Xylocaine can be used to see if the pain is relieved. If pain is relieved with the injection, then the injection can be repeated every 2 weeks for three sessions and most likely the patient’s nerve irritation will resolve. If it does not resolve, consultation with a pain center for phenol injections or cryoprobe treatments is warranted. *The most common reason for groin pain with no palpable hernia is chronic muscle strain.* These can take up to 4–6 months to heal. They require local heat, the avoidance of exercise, and nonsteroidal anti-inflammatory agents. In most instances, time will heal these injuries. Groin exploration for patients with chronic injuries or nerve irritations will likely make the situation worse.

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**Table 54.1. Anatomy of inguinal hernias.**

<table>
<thead>
<tr>
<th>Type</th>
<th>Anatomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct</td>
<td>Medial to inferior epigastric vessels</td>
</tr>
<tr>
<td></td>
<td>Through Hesselbach’s triangle</td>
</tr>
<tr>
<td>Indirect</td>
<td>Lateral to inferior epigastric vessels</td>
</tr>
<tr>
<td></td>
<td>Through internal ring along spermatic cord</td>
</tr>
<tr>
<td>Femoral</td>
<td>Below inguinal ligament</td>
</tr>
<tr>
<td></td>
<td>Through femoral canal, medial to femoral vein</td>
</tr>
</tbody>
</table>

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**Table 54.2. Types of repairs.**

<table>
<thead>
<tr>
<th>Type</th>
<th>Anesthetic</th>
<th>Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesh-Plug</td>
<td>Local with sedation</td>
<td>Plug inserted into defect, only mesh covers inguinal floor</td>
</tr>
<tr>
<td>Lichtenstein</td>
<td>Local with sedation</td>
<td>Flat mesh sutured to conjoined tendon, pubic tubercle, inguinal ligament. Mesh split laterally to create new internal ring.</td>
</tr>
<tr>
<td>Kugel preperitoneal</td>
<td>Local with sedation</td>
<td>Large piece of flat mesh used to cover entire inguinal floor</td>
</tr>
<tr>
<td>Femoral (McVay)</td>
<td>Local with sedation</td>
<td>Mesh sutured to conjoined tendon, and below to Cooper’s ligament medial to femoral vein and to inguinal ligament lateral to vein.</td>
</tr>
</tbody>
</table>
Inguinal Hernia

A. History
   bulge
   pain

B. Physical Exam

C. Unilateral Hernia
   reducible
   → open repair
     - mesh plug
     - Lichtenstein
     - preperitoneal
   incarcerated
   → possible laparotomy or laparoscopy

D. Recurrent Hernia
   no prior mesh
   → mesh-plug repair
   → preperitoneal repair
   prior mesh
   → laparoscopy

E. Bilateral Hernias
   → bilateral open mesh-plug repair
   → laparoscopic mesh repair

F. Persistent Pain
   No Hernia
   re-exam in 1-3 months
   ultrasound of groin
   nerve irritation
   → steroid injection or nerve block
   muscle strain
   → local heat, rest, anti-inflammatory agents
Portal Hypertension

Demetrios J. Louis

Portal hypertension is caused by distortion of the hepatic architecture which leads to an increased resistance to portal blood flow and eventually an increase in portal venous pressure. Less commonly, portal hypertension may result from increased portal blood flow. Normal portal venous pressure ranges from 6 to 8 mmHg. In portal hypertension, portal vein pressure rises to greater than 12 mmHg (averaging 20 mmHg), and sometimes exceeds 50–60 mmHg. Portal hypertension is classified into prehepatic, intrahepatic, and posthepatic causes. (Table 55.1)

A. Clinical Manifestations. Substantial complications may arise because of hepatocellular dysfunction from portal hypertension. Major sequelae include ascites, hepatic encephalopathy (asterixis), hepatorenal syndrome, variceal bleeding (esophageal, splenic, and gastric), spontaneous bacterial peritonitis, and hepatopulmonary syndrome. Manifestations can be classified into three major categories: hepatic, vascular, and hematologic. Hepatic manifestations include spider angiomas, palmar erythema, Dupuytren’s contracture, hypoalbuminemia, gynecomastia, testicular atrophy, fetor hepaticus, and loss of body hair. Vascular manifestations include internal hemorrhoids (controversial), caput medusae, and clubbing of fingers. The hematologic signs include pancytopenia, resulting in anemia, leukopenia, and/or thrombocytopenia.

B. Diagnosis. Functional hepatic reserve must be assessed, then the portal venous anatomy and hepatic hemodynamics should be defined and evaluated. The risks associated with any therapeutic intervention for portal hypertension are best determined after properly assessing hepatic functional reserve; the conventional method for doing so is the Child-Pugh classification (Table 55.2). In determining the cause of cirrhosis and the activity of the liver disease, a percutaneous liver biopsy is beneficial; however, in the presence of coagulopathy or moderate ascites, biopsy should be obtained via a transjugular vein approach or laparoscopically. Measuring the hepatic venous wedge pressure indirectly estimates the portal pressure. However, in patients with presinusoidal portal hypertension the measurement of portal pressure can only be measured directly by transhepatic or umbilical venous cannulation of the portal venous system, or by percutaneous puncture of the spleen. Visceral angiography is a useful method for direct visualization of the portal venous anatomy, and is beneficial before performing portosystemic shunts. A noninvasive alternative to angiography is duplex ultrasound, which is helpful in assessing portal venous patency, portal blood flow, and portal shunt patency. Endoscopy is the test of choice for diagnosing varices and should be performed only if the patient is hemodynamically stable and blood clots have been evacuated from the stomach with a large-bore lavage tube.

C. Treatment of Acute Bleeding. Treatment of portal hypertension is directed at the specific complication of the disease process (e.g., bleeding esophageal varices, encephalopathy, and ascites) and can be considered prophylactic or therapeutic. Prophylactic therapy (e.g., sclerotherapy or propanolol) is reserved for those patients who have never had variceal bleeding. Therapeutic procedures (e.g., shunts) are performed in those patients who have had variceal bleeding or other manifestations of portal hypertension.

For acute bleeding, initial management should focus primarily on the ABCs of resuscitation and stabilization. Resuscitation should begin with isotonic crystalloid solutions, usually D5W to prevent salt retention and ascites. A nasogastric tube is placed to prevent aspiration. Six units of PRBCs should be typed and crossed. The volume status can be monitored by urinary output, central venous pressure measurements, and if necessary, a Swan-Ganz pulmonary artery catheter can be placed. Prothrombin time (PT) should be assessed and fresh frozen plasma is given if the PT is longer than 3 sec. Platelet transfusion may be started if the platelet count falls to less than 50,000/mm³. Once the patient is stable, endoscopy is performed to determine the cause of the bleeding.

Endoscopic treatment involves variceal sclerosis or ligation. Ligation is accomplished with rubber banding with acceptable results. For the former, each varix is injected with 1–2 ml of sclerosant (sodium morrhuate or sodium tetradecyl sulfate), which can control bleeding in more than 85% of
patients. After the acute bleed has been controlled, repetitive sclerotherapy should be performed until all of the varices have been eliminated. Rebleeding rates and mortality are reduced. Patients who fail should be considered for elective shunting or transplantation depending on reserve.

Intravenous vasopressin may control hemorrhage in up to 50% of patients; it is a potent splanchic vasconstrictor and also constricts systemic arterioles, causing hypertension, bradycardia, coronary vasoconstriction, and decreased cardiac output. Nitroglycerin is usually administered simultaneously to control blood pressure. Somatostatin and its longer acting analogue octreotide can be used in place of vasopressin since it has fewer side effects. Octreotide is now commonly used as an adjunct to endoscopy.

For those patients who do not respond to pharmacotherapy or have failed sclerotherapy, variceal tamponade may be accomplished with a Sengstaken-Blakemore tube. Variations include the Minnesota tube and the Linton tube. The patient’s airway must be protected when these tubes are placed, therefore endotracheal intubation is necessary. Proper position of the gastric balloon below the gastroesophageal junction must be verified radiographically before inflation. The esophageal balloon pressure should be monitored closely. Overinflation may cause esophageal necrosis or perforation. Recurrent hemorrhage frequently occurs after balloon deflation; therefore definitive treatment [endoscopic therapy, transjugular intrahepatic portacaval shunt (TIPS), or an operation] should follow.

TIPS is a nonsurgical treatment modality performed by an interventional radiologist. A needle is advanced from the IVC through a hepatic vein to a major portal vein branch. Once the portal vein is reached, a guide wire is placed and a tract is created with a balloon catheter. A metal stent is placed, creating the shunt. Cessation of bleeding is noted in 80% of patients with acute variceal bleeding. TIPS serves as a temporary bridge to liver transplantation, by controlling bleeding while a patient awaits a transplant. The TIPS shunt allows for easier transplantation since the portal circulation is decompressed. Patients with Child’s class C benefit more from the TIPS procedure than with an emergency operation.

Portosystemic shunts are indicated when the acutely bleeding patient does not respond to the above procedures. Shunts decompress the portal venous system by either partially or completely diverting the portal blood flow into the low pressure inferior vena cava. In the acute setting, end-to-side or side-to-side shunts are preferable because they are technically easier to perform and require less operative time to complete.

**D. Chronic Treatment.** For the nonbleeding, stable patient, reduction of portal hypertension can be achieved by shunting or transplantation. The former is most suitable for Child’s A or B, whereas the latter is chosen for patients with end-stage liver disease. Nonselective shunts completely divert portal blood away from the liver; these include the end-to-side, side-to-side, mesovagal, and central splenorenal shunts. In the first instance, the portal vein is transected and the end of the intestinal side is sutured to the vena cava. In the second type, a side-to-side anastomosis is created. When a mesovagal shunt is performed, graft or vein is used to connect the superior mesenteric vein to the cava. In a central splenorenal shunt, the spleen is removed and the proximal splenic vein is anastomosed to the renal vein. The main complication of these operations is hepatic encephalopathy. Progressive hepatic failure is treated with transplantation.

**Selective** shunts are less likely to cause encephalopathy but are contraindicated in patients with ascites. The Warren operation or distal splenorenal shunt consists of transecting the splenic vein and anastomosing the splenic end of the vein to the left renal vein. Also ligated are the left gastric vein, right gastroepiploic vein, and veins in the splenocolic ligament. This selectively decompresses gastroesophageal varices.

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**Table 55.1. Causes of portal hypertension.**

| A. Prehepatic (portal vein obstruction) | 1. Portal vein thrombosis, accounts for half the cases in children |
| B. Intrahepatic | 1. Cirrhosis, accounts for 85% of cases in the United States |
| | (a) Alcoholic, most common cause |
| | (b) Postnecrotic cirrhosis, hepatitis |
| | (c) Biliary cirrhosis, primary is secondary to autoimmune processes; secondary occurs in long-standing biliary obstruction |
| | (d) Other causes (Hemochromatosis, Wilson’s disease, α1-antitrypsin deficiency) |
| C. Posthepatic | 1. Budd-Chiari syndrome |
| | 2. Veno-occlusive disease, associated with graft versus host, chemotherapy, and radiation |
| | 3. Constrictive pericarditis |

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**Table 55.2. Child-Pugh classification of hepatic functional reserve.**

<table>
<thead>
<tr>
<th></th>
<th>Class A Risk: Low</th>
<th>Class B Risk: Moderate</th>
<th>Class C Risk: High</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum bilirubin</td>
<td>&lt;2</td>
<td>2–3</td>
<td>&gt;3</td>
</tr>
<tr>
<td>Serum albumin</td>
<td>&gt;3.5</td>
<td>2.8–3.5</td>
<td>&lt;2.8</td>
</tr>
<tr>
<td>Prothrombin time (second above control)</td>
<td>1–3</td>
<td>4–6</td>
<td>&gt;6</td>
</tr>
<tr>
<td>Neurologic disorder encephalopathy</td>
<td>None</td>
<td>Slight Minimal grade I-II</td>
<td>Moderate Advanced grade III-IV, “coma”</td>
</tr>
<tr>
<td>Operative mortality rate</td>
<td>0–5%</td>
<td>10–15%</td>
<td>&gt;25%</td>
</tr>
</tbody>
</table>
For patients with persistent variceal bleeding despite previous shunting or for those who are not candidates for shunting, the Sugiura procedure may be considered. This operation consists of splenectomy, devascularization of the stomach and lower esophagus, and esophageal transection followed by anastomosis.

Hepatic transplantation is reserved for patients in whom pharmacologic, endoscopic, and TIPS procedures have failed. Candidates include nonalcoholic cirrhotics with bleeding esophageal varices or alcoholic cirrhotic patients who are abstinent and have either limited hepatic reserve or poor quality of life due to encephalopathy or bone pain. For patients who fit this criterion, TIPS is performed as a short-term bridge to transplantation.

Hepatic encephalopathy is treated with dietary measures and medications. Protein intake should be restricted; initially the restriction is significant but with improvement, intake can be more liberal. An osmotic diarrhea should be induced by the administration of lactulose, this may help reduce serum ammonia levels. Oral neomycin and flagyl are also used to treat encephalopathy.

Ascites is treated with salt restriction (1000mg/day), diuretic therapy with spironolactone (25 mg bid) and possibly furosemide (20 mg/day), paracentesis, and possibly a peritoneal venous shunt.
Portal Hypertension

A. Clinical Manifestations
- Ascites
- Hepatic Encephalopathy
- Hepatorenal Syndrome
- Variceal bleeding

B. Diagnosis
- Acute bleeding
  - Yes
    - Endoscopy
  - No
    - Liver Biopsy (percutaneous or laparoscopic)
      - Visceral Angiography or Duplex Ultrasound

D. Hepatic Function Reserve
- Child’s A
- Child’s B
- Child’s C
  - Distal Splenorenal Shunt
  - Side-to-Side Shunt
  - End-to-Side Portocanal Shunt
    - Fails
      - No
        - Hepatic Transplantation
      - Yes
        - 6mo – 1yr assessments
          - Mesovacal shunt
          - Nonshunt (Sugiura)

C. Bleeding Esophageal varices
- IVF Resuscitation
  - Vasopression or Octreotide with NG tube
    - Bleeding stops
    - Bleeding continues
      - Sengstaken-Blakemore balloon tamponade
      - TIPS
A. **Introduction.** Gastroesophageal reflux disease (GERD) is a common medical problem that affects over 30% of adults; however, very few patients need surgical intervention. Typical GERD symptoms include heartburn, regurgitation, and dysphagia.

B. **Patient evaluation.** Patients with only heartburn and regurgitation are commonly treated with H2-blockers and/or proton pump inhibitors; the latter is given twice a day for 6 weeks. Relief of symptoms is diagnostic for GERD since other gastroesophageal conditions (e.g., cancer) are not as likely to resolve with medical therapy alone. These medications are prescribed frequently without the aid of any diagnostic tests; this practice commonly occurs and is generally acceptable. However, persistent or refractory symptoms require further evaluation. In addition, if medical therapy is not tolerated by the patient, continued assessment is indicated. Upper endoscopy and a barium esophagogram are essential steps in the evaluation of patients with GERD symptoms. Esophagogram helps determine the esophageal anatomy and is the initial step in the workup of dysphagia. Other disease processes (masses, diverticulum, etc.) can be ruled out with an esophagogram. Endoscopy is used to diagnose and document the extent of esophageal mucosal injury. If other disease processes (masses, tumors, and diverticulum) are found, they are assessed and treated accordingly.

C. **Structures.** Esophageal strictures should be biopsied to rule out cancer although they are most likely due to long-standing GERD. Esophageal dilation and further workup for GERD is appropriate when cancer is not seen. Cancer requires esophagectomy if the patient is an appropriate candidate.

D. **Esophagitis.** Esophagitis and any suspicious mucosal areas should be biopsied to check for Barrett’s dysplasia.

E. **High-grade dysplasia.** Patients with high-grade Barrett’s dysplasia are offered esophagectomy due to the high prevalence of current and future esophageal adenocarcinoma.

F. **Low-grade dysplasia.** Patients with non-high-grade Barrett’s dysplasia should undergo manometry and pH monitoring to confirm the diagnosis of GERD, to lower esophageal sphincter insufficiency, and to rule out other disease processes. If these tests are confirmatory, patients should be offered fundoplication or continued medical therapy and should all undergo continued endoscopic surveillance for possible progression of the dysplastic changes.

G. **Normal findings.** Symptoms of heartburn, regurgitation, and dysphagia may be due to conditions other than GERD. Conversely, GERD may occur without endoscopic signs of inflammation or histologic evidence of dysplasia. Twenty-four-hour pH monitoring and manometry are helpful diagnostic tests; the former is essential for quantifying acid reflux. Persistent GERD symptoms while on maximal medical therapy may warrant surgery even without inflammation or dysplasia.

Esophageal manometry documents the function of the esophageal body as well as the lower esophageal sphincter pressure (normally ranges between 12 and 30 mm Hg). Achalasia, diffuse esophageal spasm, and other esophageal dysmotility disorders may be diagnosed and appropriately treated. Degree of esophageal body dysmotility will help determine the extent of the fundoplication (partial or full). Patients with severe esophageal body dysmotility should probably not undergo a 360° fundoplication because of possible postoperative dysphagia. Choices for these patients include observation, partial fundoplication, or, as a last resort, esophagectomy. Patients with moderate dysmotility may undergo a partial (180–270°) fundoplication. All other patients are offered a full (360°) fundoplication.
Gastroesophageal Reflux

A. Symptoms
Heartburn
Regurgitation
Dysphagia

B. Evaluation
H₂ Blockers
PPI’s

C. Stricture
Biopsy

D. Esophagitis
Biopsy

E. High-Grade Barretts
Esophagectomy

F. High-Grade Barretts
pH monitoring

G. Normal
pH monitoring, manometry

Cancer → Esophagectomy
Benign → Dilation

GERD, achalasia, esophageal dysmotility

Confirm diagnosis of lower Esoph sphincter dysfunction - fundoplication
Carcinoid Tumors

Edward F. Hollinger

The term “karzenoide” initially was used to describe a class of intestinal tumors that appear more homogenous and have a slower growth rate than intestinal adenocarcinomas. Carcinoid tumors now are known to comprise a spectrum of neoplasms that originate from neuroendocrine cells. They are considered “APUDomas” because they have the capacity for amine precursor uptake and decarboxylation, and may produce biologically active amines or peptides. The incidence of carcinoids in the United States is ~7–15 per million per year. Nearly half of patients have metastatic disease evident at the time of diagnosis, and the overall 5-year survival rate is ~50%.

A. The most common location for carcinoid tumors is in the gastrointestinal (GI) tract (74–85%), but tumors also may develop in the lungs (10–25%) or in other organs such as skin, ovary, prostate, or kidney (1–5%). Within the GI tract, the most frequent locations are the appendix (45%), jejunum (28%), and rectum (16%). Hormones produced by carcinoids vary according to their site of origin in the embryonic foregut, midgut, or hindgut. Carcinoid tumors of the respiratory tract, stomach, duodenum, and pancreas arise from foregut derivatives and typically produce serotonin and its immediate precursor, 5-hydroxytryptophan (5-HTP). Bronchial carcinoids may also secrete pituitary hormones such as adrenocorticotropic hormone (ACTH) or neuropeptides, while those of the proximal GI tract (stomach and duodenum) may also secrete GI peptides and histamine. Carcinoid tumors of the midgut (jejunum, ileum, and rectum) may produce high levels of serotonin and peptides of the tachykinin group, but rarely produce 5-HTP. Hindgut carcinoids (left colon and rectum) may produce gut peptides, but rarely produce significant amounts of serotonin or 5-HTP.

Local effects of carcinoid tumors include mucosal ulceration, pain, and bleeding. Bioactive carcinoids can induce local desmoplastic tissue growth, resulting in bronchial or bowel obstruction. Bioactive GI carcinoids can induce symptoms from intra-abdominal and retroperitoneal fibrosis.

B. Carcinoid syndrome is the most important of the systemic effects of carcinoid tumors, occurring in 5–10% of patients. Classical symptoms include cutaneous flushing (94%), diarrhea (78%), bronchoconstriction (19%), and peripheral edema (19%). Heart disease can result from subendocardial fibrosis of the pulmonic and tricuspid valves in up to 40% of patients with long-standing disease. The syndrome results when bioactive tumors produce serotonin and other hormones in anatomic areas outside those served by the portal system, that is, bronchial or ovarian primary tumors or hepatic metastases of GI carcinoids. Tumors drained by portal tributaries are unlikely to cause carcinoid syndrome because the liver metabolizes the secretory products.

Although carcinoid syndrome classically was associated with tumors producing serotonin, recent evidence suggests that histamine, dopamine, substance P, and other tachykinins may also play a significant role. Elevated levels (> 10 mg/24 h) of the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA) in the urine confirm the diagnosis of carcinoid syndrome. Diarrhea associated with carcinoid syndrome can be treated with serotonin antagonists such as methysergide, cyproheptadine, and odansetron. However, these drugs have largely been replaced by long-acting somatostatin analogs such as octreotide and lanreotide, which reduce both flushing and diarrhea. Somatostatin analogs have few significant side effects, the one exception being the formation of gallstones with prolonged administration. If extended therapy is planned, cholecystectomy should be performed when the primary tumor is resected.

C. Pulmonary carcinoids are a subset of neuroendocrine bronchopulmonary tumors that comprise a spectrum of tumors from low-grade malignant typical carcinoids to atypical carcinoids to high-grade malignant small cell carcinomas. They account for 1–2% of all lung neoplasms. Clinical symptoms include bronchial obstruction, hemoptysis, pneumonia, pleural effusions, and cough, although many patients are asymptomatic. Tumors may be detected on chest x-ray or computed tomography (CT) imaging, but definitive diagnosis usually requires biopsy. Pulmonary resection is the treatment of choice. With treatment, the overall 5- and 10-year survival rates for patients with pulmonary carcinoids are ~93 and 82%, respectively,
with 10-year survival rates significantly worse for patients with atypical carcinoids (18–60%) than for those with typical carcinoids (85–100%).

D. Gastric carcinoids account for less than 0.5% of all gastric malignancies, but may represent 10–30% of GI carcinoids. They can mimic ulcers, polyps, or carcinoma, presenting with symptoms of abdominal pain, hematemesis, diarrhea, or gastric outlet obstruction. Alternatively, they may be discovered incidentally during gastroscopy for conditions such as atrophic gastritis. Although endocrine syndromes are unusual with gastric carcinoids, they may precipitate an atypical carcinoid syndrome of prolonged, patchy, bright red flushing, facial edema, lacrimation, headache, and bronchoconstriction. This syndrome can be treated with \( \text{H}_1 \) and \( \text{H}_2 \) histamine antagonists, and excessive gastric acid secretion can be controlled by \( \text{H}_2 \) receptor and proton pump antagonists. Gastroscopy with biopsy is generally the most useful test for evaluating gastric carcinoids. Fewer than 3–5 small (<1–1.5 cm) carcinoids without evidence of metastasis may be removed by endoscopic polypectomy with follow-up by endoscopic surveillance. Lesions 1–2 cm in diameter should be surgically excised with consideration given to local lymph node dissection, while tumors larger than 2 cm should be treated with aggressive local resection, antrectomy, and lymph node dissection. Gastric carcinoids occurring without hypergastrinemia usually are solitary and show rapid, aggressive progression with local invasion and metastasis to regional lymph nodes and the liver. These tumors should be treated by gastric resection (including antrectomy) and lymph node dissection.

E. Small-bowel carcinoids represent up to one third of all malignant small-bowel tumors. GI carcinoids grow slowly, so small (<1 cm) localized small-bowel carcinoids are usually found incidentally or at autopsy. Larger tumors can cause GI bleeding or precipitate small-bowel obstruction. The lack of overt symptoms from small primary tumors means many patients have large tumors or disseminated disease at the time of diagnosis, and up to 40% of patients have multiple tumors. These carcinoids most commonly metastasize to regional lymph nodes and then to the liver, but may rarely metastasize to bone. In 17% of cases, they are associated with synchronous or metachronous tumors, most commonly adenocarcinomas of the colon.

Both anatomical techniques (upper and lower bowel contrast x-rays, enteroclysis, CT, and endoscopy) and functional imaging (somatostatin scintigraphy and 5-HT positron emission tomography) can assist in localizing tumors. Quantitative 5-HIAA measurement or provocation testing with pentagastrin can be used where a clinical suspicion of carcinoid tumor remains in the face of negative imaging studies. Primary tumors that have not metastasized should be resected together with the corresponding regional mesenteric lymph nodes, with careful examination for multicentric carcinoid disease or other simultaneous malignancy. Invasive disease with absent or limited hepatic metastasis should be treated by aggressive local resection of the primary tumor with the associated mesentery or, less optimally, local resection and debulking of involved lymph nodes. Superficial solitary hepatic lesions can be treated by wedge resection. For hormone-producing tumors, preoperative prophylaxis with octreotide may prevent carcinoid crisis, especially if hepatic metastases are present. If more advanced disease is present, the initial surgery should focus on removing the primary tumor and associated local metastasis; this reduces tumor burden and decreases the incidence of local symptoms such as bowel obstruction. Surgical and/or medical therapy for hepatic metastasis can follow. Follow-up for midgut carcinoids should include assessment of symptoms and urinary 5-HIAA measurements approximately every 3 months, with CT at longer intervals or if the patient becomes symptomatic. The 5-year survival for localized small-bowel carcinoids is about 65%, decreasing to 35% when distant disease is present.

F. Appendiceal carcinoids are the most common tumors of the appendix. They are most commonly found incidentally, often during appendectomy. Compared with other carcinoids, they occur at an earlier age (42 vs. 62 years) but have a more favorable prognosis. However, concurrent adenocarcinoma in another site within the GI tract occurs with 10–15% of appendiceal carcinoids.

Most carcinoids (70%) occur at the tip of the appendix, with fewer at the body (20%) or base (10%). More than 70% of tumors are <1 cm at the time of discovery; tumors of this size are not associated with metastasis. For these small carcinoids, simple appendectomy is curative. Tumors 1–2 cm in size metastasize in fewer than 1% of cases, so resection more extensive than simple appendectomy should be reserved for cases where evidence of aggressive tumor behavior (invasion of the mesoappendix or subserosal lymphatics) is seen. However, metastases are common for appendiceal carcinoids >2 cm in size, so right hemicolectomy should be performed. Therapy for carcinoids at the base of the appendix remains controversial. Patients without metastases or evidence of tumor invasion are best treated by appendectomy alone. Right hemicolectomy is reserved for tumors that show extensive local invasion or lymph node involvement. The prognosis for patients with appendiceal carcinoids is quite favorable, with 5-year survivals of 94% for localized lesions, 85% for regional disease, and 34% for patients with distant metastases.

G. Colon carcinoids comprise about 8% of all carcinoids, occurring most frequently in the cecum and right colon. More than 60% have metastases at the time of diagnosis, contributing to a relatively poor 42% 5-year survival despite a 71% survival for patients with only localized disease. They are also associated with synchronous neoplasms, most commonly adenocarcinomas, in 25–40% of cases.

Most patients with colon carcinoids are diagnosed after complaining of pain, anorexia, and weight loss or as an incidental finding on routine colonoscopy or barium enema. Rectal bleeding or bowel obstruction is unusual. Because
many patients will have metastatic disease at presentation, preoperative evaluation should include assessment for distant disease. Resection of the colon and associated lymphatic drainage should be standard therapy for all colonic carcinoids regardless of size because of the high rate of lymphatic involvement. Usually this consists of a right colectomy. If local invasion into contiguous organs has occurred, colectomy can be supplemented with en bloc resection of tissues showing local invasion. Unresectable tumors should be debulked, and resection or debulking of liver metastases is especially important in reducing symptoms of carcinoid syndrome.

H. Rectal carcinoids are the most common large-bowel carcinoid tumors, and the third most common carcinoid within the GI tract. Rectal tumors are frequently discovered early and have a low propensity for metastasis. They may express hormones, but are not usually associated with carcinoid syndrome. On digital exam, rectal carcinoids are firm, discrete, and mobile submucosal lesions. Rigid proctoscopy can be used for direct visualization of the tumor. Staging for rectal carcinoids consists of evaluating the primary tumor, normally by colonoscopy, and looking for evidence of metastatic disease or invasion. Endorectal ultrasound and conventional CT imaging may be useful in evaluating the extent of disease when regional metastases are suspected. Distal metastases may be evaluated similarly to other GI carcinoids.

Rectal carcinoids < 1 cm in diameter are rarely associated with metastasis and can be treated by endoscopic excision. Tumors 1–2 cm in diameter have been associated with non-localized disease in up to 45% of patients. For these tumors, treatment should consist of transmural resection with adequate margins to evaluate invasion of the muscularis. Appropriate additional therapy can be determined based on intraoperative pathology, including the histology of the lesion (typical or atypical carcinoid) and the presence of invasion. Pathology showing invasion of the muscularis or an atypical carcinoid should be treated further by aggressively re-excising the tumor site to ensure disease-free margins. The patient should be carefully evaluated for the presence of metastatic disease, and more-frequent follow-up may be indicated. Rectal carcinoids > 2 cm in diameter are associated with metastases in more than 60% of cases. Because these tumors have a poor response to chemotherapy or radiation, surgery remains the mainstay of therapy. However, several recent studies suggest that extensive surgical resection does not significantly improve disease-free survival in patients with large rectal carcinoids. A compromise may be that tumors that can be resected without compromising sphincter function should be treated with low anterior resection. However, if complete resection cannot be undertaken without compromising sphincter function, the decreased quality of life incurred by patients undergoing abdominoperitoneal resection may not be justified given the high risk of disease recurrence.
Carcinoid Tumors

A. Characteristics
   a.) Location
      1) GI tract (75-85%)
      2) Lung (10-15%)
      3) Other (1-5%)
   b.)
      1) Serotonin
      2) 5-HTP
      3) ACTH
      4) Neuropeptides
      5) GI peptides
      6) Histamine

B. Carcinoid Syndrome (5-10%)
   1.) Flushing (94%)
   2.) Diarrhea (78%)
   3.) Bronchoconstriction (19%)
   4.) Peripheral edema (19%)
   5.) Heart disease in long standing patients

C. Pulmonary Carcinoids
   1.) 1-2% of lung neoplasms
   2.) Suspcion by CT or chest X-ray
   3.) Diagnosis requires biopsy
   4.) Pulmonary resection therapy of choice
   5.) 82% 10 year survival

D. Gastric Carcinoid
   1.) 10-30 % GI Carcinoid
   2.) Endocrine symptoms unusual
   3.) EGD with biopsy diagnostic
   4.) Lesion <2cm excision
   5.) Lesion >2cm excision, antrectomy, lymph node dissection

E. Small Bowel Carcinoids
   1.) Commonly metastasize to lymph nodes then liver
   2.) CT, endoscopy, somatostatin scintigraphy
   3.) Resection with lymph node dissection for localized tumors
   4.) Advanced disease: remove primary and associated local disease to reduce tumor burden
   5.) Surgical or medical therapy for hepatic metastases

F. Appendiceal Carcinoids
   1.) Most common tumor of appendix
   2.) Lesions <1cm appendectomy
   3.) Lesions 1-2cm appendectomy unless invasion of mesoappendix on subserosal lymphatics then right hemicolecotomy
   4.) Lesions >2cm right hemicolecotomy

G. Colon Carcinoids
   1.) 60% metastasize at time of diagnosis
   2.) Resection of colon and associated lymphatics regardless of extent of disease
   3.) Debulking required with treatment of liver mets for advanced disease

H. Rectal Carcinoids
   1.) Most common large bowel carcinoid
   2.) Low propensity to metastasize
   3.) Endoscopy, endorectal ultrasound and CT diagnostic evaluation
   4.) Lesion <1cm endoscopic excision
   5.) Lesion 1-2cm low anterior resection
   6.) Lesion >2cm low anterior resection if possible to preserve sphincter
Indications for Renal Transplantation

Deepak Mital

A. Indications. Most patients with end-stage renal disease (ESRD) are candidates for renal transplantation. ESRD is defined as a creatinine clearance < 20 ml/min. Patients are maintained on either hemodialysis or peritoneal dialysis. If a living donor is available, the patient may receive a “preemptive” transplant without ever starting dialysis.

Metabolic causes of ESRD are as follows: diabetes mellitus (the most common cause of ESRD in North America) is the only group where a distinct survival advantage has been shown with a transplant (vs. dialysis); hyperoxaluria carries a risk of recurrence of renal failure. In some cases, a liver–kidney transplant may be indicated; Fabry’s disease (angiokeratoma corporis diffusum universale) rarely recurs after renal transplantation, with graft survival at 3 years as high as 80%; hypertension is a common cause of ESRD, especially in the African-American population; glomerulonephritis may be due to IgA nephropathy or focal segmental glomerulosclerosis (FSGS). The former carries a > 50% risk of recurrence in the transplant kidney. This can be prevented to a large extent by posttransplant administration of fish oil (omega 3 fatty acids) capsules long term. FSGS is expected to recur in 20–50% of renal transplants. It may be treated with plasmapheresis and high-dose calcineurin inhibitors (CNIs; cyclosporine or Prograf). Other causes include hereditary, for example, polycystic kidney disease, Alport’s syndrome, and obstructive uropathy, and toxic, for example, analgesic nephropathy is caused by long-term administration of nonsteroidal anti-inflammatory drugs (NSAIDs) like Ibuprofen. After a transplant, these drugs should be avoided and hemolytic uremic syndrome.

Patients with SLE (Systemic Lupus Erythematosus) may have high levels of autoantibodies, which may lead to a false-positive cross-match. The disease usually stays quiescent after a transplant, due to the immunosuppression. It is important to look for anticoagulants and other lupus antibodies which may cause vascular thrombosis due to a hypercoagulable syndrome.

B. Contraindications are as follows:

1. AIDS: Some patients with HIV who are doing well on antiretroviral therapy are now being considered as candidates. The CD4 counts are carefully monitored.

2. Sepsis: These patients will do worse with immunosuppression, for example, TB, hepatitis BsAg positive, and viremic hepatitis C.

3. Advanced cardiovascular disease: For example, ejection fraction < 30%, bilateral severe iliac/femoral artery atherosclerosis, and pulmonary hypertension may be contraindications for a transplant.

4. Poor respiratory status: Needing oxygen therapy at rest for advanced interstitial lung disease indicates poor respiratory reserve.

5. Cancer: Unresolved/active malignancy will worsen with immunosuppression (locally invasive skin cancers are exceptions).

6. Psychosocial: Active drug abuse/psychosis with lack of family support predicts noncompliance with medications. These groups are at high risk for graft loss due to rejection.

C. Evaluation. Recipients should undergo a thorough history and physical examination: advanced chronological age (≥ 70) is not a contraindication, if the patient is in good physiological condition. CBC (anemia, leukopenia), chemistry panel (PTH levels, amylase/lipase), and liver enzymes (hepatitis) are markers for conditions needing more detailed pretransplant evaluation and management. Serologic tests include CMV, EBV (prophylaxis with ganciclovir), hepatitis B and C (interferon for hepatitis C after liver biopsy, if indicated), and HIV (CD4 status).

A living donor should be at least 18 years (age of consent legally). If older than 60, the donor should be free of long-standing diabetes, hypertension, and viral hepatitis or HIV. An unrelated living donor should have a strong emotional bond (e.g., spouse). A detailed psychological assessment is indicated in some cases where the relationship is not easily verifiable. The donor should also undergo evaluation by a nephrologist not involved in the care of the recipient. This includes measurement of creatinine clearance, HbA1C, etc. and as a final step, a spiral CT scan to look for the renovascular anatomy.

Cadaveric donors must have confirmed brain death; this is usually a result of trauma or a cerebrovascular accident. The
donor is maintained on a ventilator and the heart is beating until the organs are procured. Donors must be in excellent physiologic shape, lack hypotension, be free of high-dose pressor agents, and lack elevated serum creatinine, oligoanuria, and trauma to other organs. The harvested organs are preserved by flushing with the University of Wisconsin solution, which resembles intracellular fluid. If the “cold ischemia time” (CIT) prior to renal transplantation exceeds 24 h, the incidence of acute tubular necrosis (ATN) rises exponentially. With pulsatile-perfusion machine preservation, ATN rates are significantly lower with CIT > 24 h.

Non-heart-beating donors are a small subset of patients where cardiac arrest has occurred up to 30 min prior to organ perfusion and recovery. This leads to “warm ischemia,” with higher rates of ATN. Overall results are acceptable with careful selection. This may involve measuring the flow rate and resistive indices on a pulsatile-perfusion pump. High flow rates and low resistance predict a well-preserved and viable kidney.

D. HLA Matching and Cross-Match. ABO blood group compatibility is essential; otherwise, the kidney will be destroyed by preformed antibodies within hours. Rarely, exceptions are made.

Human leukocyte antigens (HLA) are located on the short arm of Chromosome 6 and indicate genotypic matching. A, B, and DR loci are considered. Parents are a one-haplotype match. Siblings may be HLA identical (25% chance), share one haplotype (50%), or have no HLA matching (25%). Long-term results are superior for HLA-identical transplants. Donor cells are mixed with recipient serum to look for preformed HLA antibodies. This may be done in the “standard” fashion by a serologic complement-dependent cytotoxic assay. In a “positive” cross-match, donor cells are destroyed, indicating a poor outcome. For the last decade, “flow” cytometry has been increasingly used because of its higher sensitivity.

E. Transplant. In the perioperative period, a chest x-ray (CXR) and electrocardiogram (EKG) are done along with a medical and laboratory assessment. Prophylactic antibiotics and immunosuppressives are administered. The kidney is transplanted retroperitoneally in the iliac fossa. The renal artery and vein are anastomosed to the external iliac vessels in an end to side fashion. The ureter is anastomosed to the bladder with an antireflux tunnel. The native kidneys are not removed and are implanted retroperitoneally in the iliac fossa. The renal artery and vein are anastomosed to the external iliac vessels in an end to side fashion. The ureter is anastomosed to the bladder with an antireflux tunnel. The native kidneys are not removed and may be treated successfully in ≥85% cases with 10–15 mg/kg bolus IV Solumedrol (methylprednisolone) given daily for 3 days. If this fails, anti-T cell antibodies are usually successful. Chronic: chronic allograft nephropathy (CAN) is a condition caused by repeated acute rejection episodes leading to chronic rejection, or CNI nephrotoxicity, or donor injury at time of procurement/transplant or preexisting donor diseases. There is no successful treatment. Eliminating or lowering the CNI dose and adding sirolimus or Cellcept are new strategies which may help.

F. Immunosuppression. “Induction” with monoclonal or polyclonal antibodies is often used. “Maintenance” is with a combination of CNIs (e.g., tacrolimus/cyclosporine), anti-proliferative drugs like mycophenolate mofetil (Cellcept) or newer agents like sirolimus—a macrolide antibiotic with immunosuppressive effects. Corticosteroids are being avoided/withdrawn early in current practice with the availability of the above potent and specific agents.

G. Complications. Surgical complications include vascular thrombosis/stenosis (<2%). This is usually due to technical error or a hypercoagulable syndrome. The graft is often lost.

Ureteric leaks or stenosis (1–5%) are usually due to technical error/ischemia. They may be treated by placing a stent (radiology) or surgical correction. Lymphoceles (5–15%) are caused by leakage of lymph from the lymphatics around the iliac vessels which were not ligated at the time of the transplant. The fluid collection compresses the ureter, leading to obstruction. Treatment consists of radiological drainage or surgical marsupialization into the peritoneum, often done laparoscopically.

Infections may occur after transplantation. Bacterial infections (central lines, wound, and urinary infection) are due to perioperative factors and occur in the first 4 weeks posttransplant. Viral infections (>1 month: CMV, EBV, and hepatitis) reflect over-immunosuppression along with transmission of viruses from the donor. Hence, antiviral prophylaxis is given for 3–6 months posttransplant. Opportunistic infections (late ≥3 months, e.g., PCP/fungal/legionella) are due to prolonged over-immunosuppression, either due to prolonged antibody “induction” therapy after transplantation or for treatment of rejection episodes, along with corticosteroids.

Malignancies occur with higher frequency in transplant patients (after 1–3 years). The incidence of lymphomas is highest with EBV transmission from the donor. Skin cancer is most common in Australia, with higher exposure to sunlight.

Rejection may follow one of three patterns: Hyperacute: leads to immediate graft loss within 6 h. It is due to preformed anti-ABO or HLA antibodies. It is rare as it can be predicted by a pretransplant cross-match. Acute: occurs in 10–30% of renal transplants. It is most common in the first 6 months to 1 year after transplantation, but may occur years later if the patient omits taking antirejection medication. It is T cell mediated and may be treated successfully in ≥85% cases with 10–15 mg/kg bolus IV Solumedrol (methylprednisolone) given daily for 3 days. If this fails, anti-T cell antibodies are usually successful.

H. Results. One-year allograft survival is now >95% for living donor renal transplant and ~90% for cadaveric transplants. Five-year allograft survival is best with HLA-identical transplants (~80%) and worst for poorly matched cadaveric transplants with ATN ≤50%. Results are improving with better immunosuppression and organ preservation. Transplantation offers restoration of an excellent quality of life and freedom from dialysis as well as the long-term effects of renal failure.
Indications for Renal Transplantation

A. Indicator
- ESRD
- Creatinine clearance <20ml/min
- Causes *

B. Contraindications
- AIDS
- Sepsis, heart disease, cancer, poor respiratory status

C. Evaluation

Recipients

Living Donor
- ≥ 18 yrs age
- No diabetes
- No hypertension
- No hepatitis

Cadaveric Donor
- Brain Death
- No hypotension
- No pressors
- No renal failure

D. HLA Matching
- Chromosome 6

E. Transplant

F. Immunosuppression

G. Complications
- Vascular, ureteric, lymphoceles, infection, rejections

* Diabetes
  Hyperoxaluria
  Fabry’s disease
  Glomerulonephritis
  Toxic
In the most simplistic view, liver transplantation is indicated for patients with irreversible liver injury. The United States statistics on death reveal that 400,000 individuals have end-stage liver disease and 26,050 die of liver disease each year. However, only 18,444 of these patients are listed for liver transplantation and 4,954 undergo liver transplantation annually. The 18,000 patients on the transplant list (1) have liver diseases in which liver transplant has been shown to significantly prolong life and (2) have been identified, through a careful evaluation process, as having acceptable comorbid disease, compliance, and social support requirements. The patients who actually undergo transplantation are determined by the allocation system for organ donors.

Although indications for liver transplant by disease criteria are fairly uniform and are listed below, they have changed over time. Additionally, different etiologies represent different quantities of risks. Each transplant program must determine the quantity of acceptable risk. These variables can result in confusion when a patient is determined to be a candidate by one program and denied by another.

A. Evaluation. When a patient is diagnosed with severe acute or chronic liver disease, referral to a liver center provides that patient with entry into the evaluation process. This process is broken into the following components:

**Etiology:** The etiology of the liver disease becomes important in estimating the probability of recurrence of the disease in the transplanted liver. While metabolic disease (absence of a specific enzyme) will not recur after transplantation, liver disease secondary to hepatitis C virus (HCV) will recur. Determination of the etiology will also help predict the rapidity of deterioration from the disease. Additionally, some etiologies are associated with the development of malignant tumors, as occurs with hepatitis B and C and with primary sclerosing cholangitis (PSC), and require frequent monitoring.

**Severity of disease:** Further evaluation will be performed to identify complications of the liver disease. If the etiology produces cirrhosis, then evaluation/management of portal hypertension is needed. Previous variceal bleeding or portal gastropathy may require medical management or shunt therapy. The presence of ascites will require the utilization of diuretic therapy, large-volume paracentesis, and possibly transjugular intrahepatic portosystemic shunt (TIPS). Intractable ascites, hydrothorax, or a history of spontaneous bacterial peritonitis are associated with decreased survival. Overall function of the liver is estimated by evaluating protein synthesis as measured by coagulation factors, albumin, bilirubin, and other visceral proteins. However, as liver function deteriorates, inability to clear neurotransmitters and/or byproducts of drug–protein metabolism results in hepatic encephalopathy, yet another predictor of decreased survival. Chronic malnutrition and muscle wasting, although unsightly, do not significantly predict survival. Renal dysfunction is evaluated to determine reversibility with liver transplant or if simultaneous transplantation of the kidney is needed. Liver disease can cause intrapulmonic shunts to occur, resulting in hypoxemia and pulmonary hypertension; the presence and reversibility of this complication must be identified prior to the determination of candidacy. Measurement of tumor markers and imaging of the liver to exclude the presence of hepatic malignancy is required.

**Risk of death:** There are multiple formulas to estimate the severity of liver disease and subsequent risk of death. The Childs-Pugh-Turcot score is used to determine whether the patient’s illness is severe enough to meet listing criteria, and the Model for End-Stage Liver Disease (MELD) or Pediatric End-Stage Liver Disease (PELD) score is used to determine severity of illness while listed. The MELD/PELD score is a formula which uses patient’s bilirubin, albumin, and INR to produce a numerical value; the higher the numerical value, the greater the risk of death in the next 3 months.

**Comorbid disease process:** Determination of comorbid diseases is utilized to evaluate the risk of the transplant procedure and to estimate life expectancy exclusive of liver disease. The presence and severity of systemic diseases, such as diabetes or coronary artery disease, are ascertained. A search for occult infection is necessary. Obesity has been associated with increased complications and decreased survival. Smoking
is addressed and attempts at cessation are made. A thorough search for occult cardiac disease requires noninvasive and/or invasive evaluation and estimation of cardiac reserve. This information will be utilized to establish the physiological age of the patient and, therefore, the risks of the transplant procedure and the probability of complications following transplantation.

**Psychosocial evaluation:** End organ diseases ravage not only the patient’s body but also his/her social and financial resources. Studies have shown that the absence of a support system negatively impacts transplant outcomes. Identification and treatment of chemical dependency (CD)/abuse is mandatory prior to candidacy.

**Financial:** Liver transplant is a recognized therapy for end-stage liver disease and is covered by Medicare, Medicaid, and most commercial payers/health maintenance organizations (HMOs). Despite these resources, patients may still present without financial means. Some groups altruistically believe that transplantation should be provided regardless. However, without coverage for life-long immunosuppression and maintenance, proceeding with transplantation would result in the waste of a rare organ. Therefore, all patients require some means of financial coverage prior to candidacy.

**Allocation:** Allocation exists because of an inadequate organ supply. Methods used to allocate donor livers have varied and changed repeatedly. Presently we utilize the MELD/PELD scoring system to stratify patients within areas of organ allocation. Those with emergent need of liver transplant make up the first level. When the needs of this group of patients have been met, the organ is offered to the next level of need. These remaining patients are listed as to their numerical MELD/PELD score. The patient with the highest score is offered the organ first, followed by the next numerical value, and so on. Organs are offered to the local center first, then within the region, and then nationally.

**B. Disease Indications. Cholestatic liver disease:** Cirrhosis of the liver occurs because of chronic inflammation from retained bile salts. Biliary atresia occurs in infancy from an unknown process which destroys the extrahepatic biliary tree. Primary biliary cirrhosis (PBC) is an autoimmune process which causes destruction of intrahepatic ducts. Primary sclerosing cholangitis, another autoimmune process, destroys the extrahepatic ducts and is associated with development of cholangiocarcinoma. Secondary biliary cirrhosis occurs from prolonged injury to the liver from chronic bile obstruction and has no autoimmune component.

**Viruses:** Hepatitis is frequently caused by viruses. Hepatitis A almost always resolves without scarring. Hepatitis B virus (HBV) can be a chronic infection causing cirrhosis. If HBV is actively replicating, as identified by a positive HBV-DNA, then recurrence in the transplant liver will occur without adjuvant therapy. HCV produces cirrhosis after 20 years of exposure. Universal recurrence in the transplant liver occurs but destruction is usually slow with acceptable survival. Hepatitis E and G do not produce acute liver failure or chronic disease.

**Autoimmune hepatitis:** Escape of autoreactive antibodies results in the destruction of tissue. Autoimmune hepatitis results from the development of antinuclear or anti-kidney-liver-microsomal antibodies. This chronic inflammation results in hepatic necrosis and progressive cirrhosis. Immunosuppression can retard the progression but has been unable to eliminate the disease process.

**Toxins/drugs:** Toxins/drugs produce hepatocyte injury either directly or indirectly. In certain cases, the degree of injury is proportional to the quantity of toxin/drug. Some substances produce an idiosyncratic reaction resulting in progressive fibrosis, while others may cause complete destruction of the hepatocytes resulting in acute fulminant liver failure.

**Metabolic diseases:** There are multiple specific enzyme deficiencies which can result in metabolic liver disease. These deficiencies can be divided into those which produce chronic liver injury and subsequent cirrhosis and those in which the affected metabolic pathway is vital, resulting in early death without adjuvant treatment. Alpha-1-antitrypsin deficiency, Wilson’s disease (absence of ceruloplasm), and hemochromatosis produce deposition within the liver, causing cirrhosis and its complications. Metabolic diseases such as defects in the urea cycle produce neurological dysfunction following birth, with resultant death if not managed by dialysis. Liver transplant from a normal donor replaces the enzyme deficiency.

**Tumors (benign or malignant) and space occupying lesions:** Malignant disease limited to the liver was originally thought to be treatable by replacing the liver. Experience, however, has confirmed the diffuse nature of metastatic disease, with subsequent recurrence of the cancer in the transplanted liver. Today, only those metastatic tumors which have a slow indolent course, such as neuroendocrine and carcinoid, are recognized indications. Primary tumors of the liver include hepatocellular carcinoma (HCC), cholangiocarcinoma, and hemangioendothelioma. Hepatocellular carcinoma has low recurrence rates for stages I–II and is therefore an approved indication for transplantation. However, stage III or IV disease has a high recurrence rate and thus transplantation is not indicated. Cholangiocarcinoma at all but the in situ stages also has an unacceptable recurrence rate. The rare vascular tumor hemangioendothelioma has an acceptable slow growth rate resulting in similar patient and graft survival to non-tumor transplant patients. Patients with benign tumors such as giant
hemangiomas or benign polycystic liver disease, which are not resectable, are acceptable candidates for transplantation.

**Alcoholic liver disease (ALD):** Transplantation for ALD still raises strong opinions regarding societal obligation. This, combined with a high recurrence rate, has made these patients less-favorable candidates. The key is to recognize ALD as two diseases: one of liver disease and the other as a CD disease. Replacement of the liver will resolve ALD but management of CD requires both pretransplantation and lifelong treatment. We have designed a novel treatment program consisting of mandatory participation in a CD program both prior to and following transplantation. We link the patient’s care so that the medical team sees the patient for management of complications of liver disease and the CD counselor sees the patient during the same clinic visit. This coupling of treatment modalities has resulted not only in the ability to maintain control of the CD behavior but also in identification of those patients in whom compliance with medical therapy is not achievable. With successful management of the patient’s CD disease, patient and graft survival approximate those of transplantation for cholestatic disease.

**Nonalcoholic steatotic hepatitis (NASH):** Pathological deposition of fat into the liver, resulting in inflammation and subsequent cirrhosis, occurs in patients who are morbidly obese with type II diabetes. Whether this disease has an additional genetic cause is unknown.

**Vascular thrombosis:** Vascular thrombosis of the liver occurs in association with congenital vena caval webs, hypercoagulable states, response to chemotherapeutic agents such as methotrexate, and following bone marrow transplant. Acute outflow obstruction produces liver enlargement, ascites, and bowel ischemia. The chronic congestion of the liver produces fibrosis, thus further impairing splanchnic/hepatic blood flow. Early management with bypass shunt techniques does not address the cause of the vascular thrombosis and has a high failure rate. Liver transplant corrects the anatomical abnormality as well as the metabolic abnormality.

**Acute fulminant hepatic failure:** Acute toxin exposure, medication, viral infection, metabolic error, or pregnancy can produce total destruction of the liver resulting in acute liver failure. The course is rapid, with death resulting within days. These patients pose a tremendous challenge because it must be determined whether the acute injury can be stopped, thereby allowing recovery, or whether emergent transplantation is needed. Once it is decided that the patient will require transplantation, it is then a further challenge to maintain the patient while waiting for a donor organ to become available. Despite our best efforts, only 30% of patients with acute fulminant hepatic failure receive transplantation; of those, 80% survive the first year.

**Cryptogenic cirrhosis:** Despite the above-described evaluation, a definite cause of liver disease cannot always be identified during the pretransplant period. Sometimes evaluation of the explanted liver identifies a cause. However, patients undergoing transplantation for cryptogenic cirrhosis have an acceptable survival rate.

**C. Contraindications to Liver Transplantation.** As with many other procedures, contraindications of one era become relative contraindications of another. Current absolute contraindications include (1) the presence of an irreversible comorbid process which will not be reversed by liver and or multiorgan transplant(s), (2) limited life expectancy despite reversal of liver disease, and (3) transplantation for a disease process such as malignancy which has a rapid recurrence with a short life expectancy. Previously, infection with human immunodeficiency virus was considered to be a contraindication; however, the University of Pittsburgh has reported its success with transplantation in the presence of this disease. Other contraindications include active infections, unless the source of the infection is the liver, such as in biliary cirrhosis. Because of the need for maintenance immunosuppression, the inability to comply with prolonged medical therapy is also considered a contraindication. Other contraindications are determined by individual program criteria but include morbid obesity, absence of identifiable social support, and excessive physiological age.

**Summary.** Liver transplantation has been the pivotal development in the care of patients with irreversible liver injury/disease. Prior to transplantation, the management of end-stage liver disease could only involve treatment of the associated complications, with no hope for complete recovery. Success rates of transplant therapy, as measured in patient survival, are in the 90% range in many centers, which exceeds many standard medical therapy success rates. The medical economics of the management of end-stage liver disease also reveals the efficacy of transplantation. Unfortunately, inadequate organ donation prevents this lifesaving procedure from being made available to the vast majority in need.
Indications, Evaluation, and Allocation for Liver Transplantation

A. Evaluation
1. Etiology
2. Severity
3. Mortality
4. Comorbidities
5. Psychosocial
6. Financial
7. Selection
8. Allocation

B. Indications
1. Cholestatic Liver Disease
2. Viral Hepatitis
3. Autoimmune Hepatitis
4. Toxins/Drugs
5. Metabolic Disease
6. Tumors
7. Alcoholic Liver Disease
8. Non-Alcoholic Steatotic Hepatitis
9. Vascular Thrombosis
10. Acute Fulminant Hepatic Failure
11. Cryptogenic Cirrhosis

C. Contraindication
1. Presence of irreversible comorbid process
2. Limited life expectancy
3. Disease process that has rapid recurrence with a short life expectancy.
4. Active infection
Melanoma

Steven D. Bines

The incidence of melanoma is rising faster than any other malignancy. With over 60,000 cases and 8,000 deaths occurring in the year 2002, melanoma causes over 90% of deaths due to skin cancer. The personal risk was estimated at 1 in 70 in the year 2000. Risk factors for melanoma include a positive family history, Type 1 and Type 2 skin, and blistering sunburn before adolescence. The most significant risk factor is the total number of moles. People with more than 50 moles have a two- to threefold increased risk. The presence of atypical nevi (macular nevi greater than 5 mm in diameter with an irregular border and reddish pigmentation) is a marker for increased risk. In patients with a strong family history, atypical nevi are considered premalignant lesions.

A. Suspicious Skin Lesion. All suspicious pigmented lesions should be biopsied. Suspicious pigmented lesions include those that are asymmetrical, those that have shown a border change, those that have changed in color, those that have increased in diameter, and those that have become elevated. These are the ABCs of skin examination. Clinically, atypical nevi (see above) should be biopsied to document the presence of atypia. Moles appearing in non-sun-exposed areas are particularly suspicious. Patients with suspicious pigmented lesions should undergo a complete skin examination and examination of the regional lymph nodes prior to biopsy.

B. Establishing the Diagnosis. The single most important prognostic indicator for melanoma is tumor thickness. For this reason, whenever possible, complete excisional biopsy should be performed to allow the pathologist to examine the entire lesion. Excisional biopsy should provide a visibly free margin and be carried into the subcutaneous fat. In lesions where this is impractical (the face, the palm, the sole), incisional or punch biopsy is acceptable. In these instances, an attempt should be made to sample the area showing elevation or findings suggesting ulceration.

The pathology report should contain information of tumor thickness as well as the presence or absence of ulceration. Tumor thickness is measured either by Clarke’s levels or by Breslow’s levels. Clarke’s levels refer to invasion of tumor relative to the anatomic structures of the skin. Level 1 lesions are above the basement membrane. Level 2 lesions are into the papillary dermis. Level 3 lesions are at the papillary reticular dermal junction. Level 4 lesions are into the reticular dermis and Level 5 lesions extend into the subcutaneous fat. Breslow’s measurements are performed by measuring tumor thickness directly from the specimen using a micrometer in the microscope eyepiece. Measurement starts from the granular layer of the epidermis and extends to the deepest point of penetration. In situ lesions above the basement membrane do not have a Breslow’s measurement. Of the two techniques, Breslow’s measurements are considered more prognostic. For each tumor thickness, the presence of ulceration is associated with a worsened prognosis.

C. Management of Melanoma. Patients with cutaneous melanoma should undergo complete physical examination with emphasis on the skin and screening of the regional lymph nodes. The exam should also include a screening neurologic examination. All patients should undergo a baseline chest x-ray and serum lactate dehydrogenase (LDH). Additional metastatic workup is not indicated unless the patient has specific symptoms or findings suggesting metastatic spread on physical examination. For asymptomatic patients with a normal physical examination and a melanoma less than 4-mm thick, additional studies beyond chest x-rays and LDH are not cost-effective. The yield of these studies is low and can be associated with a false-positive rate as high as 7%. For patients with melanomas greater than 4-mm thick or those with findings suggesting metastatic disease, workup should include an MRI of the head, CT of the chest, abdomen, and pelvis, and PET scan.

Invasive melanomas are capable of recurring locally, metastasizing to the regional lymph nodes, or developing distant metastases. In transit metastases develop in a small percentage of patients in the dermal and subcutaneous lymphatics between the primary site and the regional lymph nodes.

All malignant melanomas are treated locally with wide local excision. This excision is full-thickness carried down
to but not through the underlying fascia. In most cases, closure with elevation of local advancement flaps is possible. Occasionally, skin grafts or more complex flaps are required for closure. For in situ melanomas, the margin of excision should be 0.5 cm. For invasive melanomas up to 1 mm in thickness, a wide local excision with a 1-cm margin is indicated. For melanomas between 1 and 2 mm in thickness, a wide local excision margin of 2 cm is appropriate. For melanomas greater than 2 mm in thickness, a margin of 2–3 cm is appropriate. On extremities, these excisions should be performed along the longitudinal axis to capture the lymphatics leading to the regional lymph nodes.

With regard to the regional lymph nodes, several prospective, randomized clinical trials have shown that prophylactic lymph node dissection does not confer a survival advantage. Patients with clinically positive biopsy-proven lymph node metastases should undergo the appropriate radical lymphadenectomy. This is not a lymph node sampling, but rather an anatomically defined complete lymph node dissection. For patients with invasive melanomas 1 mm or thicker, or patients with melanomas 0.75 mm thick associated with the Clarke’s level 4 depth of invasion and/or ulceration, sentinel lymph node biopsy is indicated. This is a two-stage procedure that consists of lymphatic mapping to define the precise pattern of lymph node drainage and the location of the sentinel lymph node followed by a focused dissection to remove the sentinel node. The sentinel lymph node is the first lymph node draining the primary site and is identified by lymphatic mapping using technetium-labeled sulfur colloid. It is the node most likely to contain metastatic disease if spread to the lymph nodes has occurred. Multiple prospective trials have shown that the sentinel node is highly accurate in predicting the presence of metastatic disease. The second stage of sentinel lymph node biopsy occurs in the operating room. If technetium-labeled dye is used, the location of the node is confirmed by the use of a hand-held probe. Further confirmation can be obtained by injecting vital blue (isocyanin blue) dye, in the dermis at the primary tumor site 15 min prior to dissection in the regional lymph node basin. Sentinel lymph nodes are defined as nodes containing radioactive dye and/or blue dye (if blue dye is used). In addition, during sentinel lymph node biopsy, enlarged nodes or acanthotic nodes are removed. Patients found to contain metastatic disease in the sentinel lymph node should undergo a completion radical lymphadenectomy. The sentinel node is studied using thin-section techniques and staining by H&E as well as for immunohistochemical markers including HMB45 and S100. This process generally takes several days and regional lymph node dissection is usually performed at a second visit to the operating room.

Patients with positive sentinel nodes should undergo a metastatic workup prior to regional lymphadenectomy. Patients with positive sentinel nodes as well as patients with primary tumors greater than 4-mm thick should be considered for adjuvant treatment with high-dose interferon. High-dose interferon is the only agent approved by the Food and Drug Administration (FDA) for the adjuvant treatment of melanoma. Initial studies with high-dose interferon showed a statistically significant improvement in overall and disease-free survival. Several subsequent trials have confirmed the improvement in disease-free survival but failed to demonstrate statistically significant improvement in overall survival. For this reason, consideration should be given to enrolling these patients in ongoing clinical trials looking at the efficacy of alternative adjuvant treatments. Controversy exists as to the biologic significance of lymph nodes containing melanoma detected only by reverse transcription polymerase chain reaction RT-PCR. This question is being examined by the Sunbelt Melanoma Trial.

Patients with negative sentinel nodes are followed. The false-negative rate is 3%. Patients who develop palpable disease after a negative sentinel lymph node biopsy should undergo metastatic workup and, if negative, a radical regional lymphadenectomy.

There is no standard treatment for metastatic disease. It is worthwhile to explore multiple options and to attempt to enroll the patient in a clinical trial. Solitary metastases should be considered for resection if it is anatomically feasible, the patient is healthy, and there has been no evidence of additional metastatic disease over a short period of observation.

Postoperatively, for patients rendered disease free by surgery, follow-up should include careful physical examination every 3 months for the first 2 years, every 6 months for years 3, 4, and 5, and annually thereafter. The patient should undergo an annual chest x-ray and a serum LDH should be obtained with each office visit. Serum LDH is sometimes elevated in patients with metastatic disease and is considered a helpful but not highly sensitive or specific test.
A. Suspicious Skin Lesion
   ABCD’s
   Atypical nevis
   Moles in non sun-exposed areas

B. Establish Diagnosis

   Benign
   Atypical

   Skin exam Every 6 months

   In-Situ
   .75-1mm and Level IV or Ulcerated
   Wide local excision (WLE) 0.5cm margin

   <1mm
   WLE 1cm margin

   1-4mm
   WLE 2cm margin

   >4mm
   Metastatic Work-up

   (-) SLN
   Follow-up

   (+) SLN
   Node dissection Adjuvant therapy Systemic work-up

C. Melanoma

   Lymphatic mapping SLN biopsy

   Systemic therapy

   (+)
Hypertrophic Pyloric Stenosis
Andrew Davidoff

A. Pathophysiology. Pyloric stenosis is a condition of infants characterized by hypertrophy of the pyloric muscle. The pylorus does not relax normally, resulting in a gastric outlet obstruction. The etiology of pyloric stenosis is not known with certainty but it may be secondary to a local deficiency of nitric oxide. The differential diagnosis is quite extensive and includes many medical conditions which may result in feeding intolerance. Common conditions which may present with vomiting include poor feeding technique, formula intolerance, gastroesophageal reflux, and gastroduodenitis. Other surgical conditions causing nonbilious emesis in this age group are rare.

B. Presentation. Infants typically present between 2 weeks and 2 months of life with progressive nonbilious emesis, often described as being “projectile.” The vomiting typically occurs within 1 h after feeding and the infant appears to remain hungry. Males, often first born, are affected 4–6 times more often than females. Additionally, infants of mothers who had pyloric stenosis are affected more than 10 times as often, 3 times as often if the father had the disease. The infants may appear well or severely dehydrated at presentation, in part, depending on the length and severity of symptoms.

C. Physical Examination. On careful examination a mass (“olive”) representing the hypertrophied pylorus should be palpable in the epigastrium. Nasogastric decompression of the stomach while the infant drinks sugar water may help to calm the infant and keep the stomach decompressed to permit an optimal examination.

D. Evaluation. If the patient is of the appropriate age and has the typical presentation, the finding of a palpable epigastric mass is sufficient to confirm the diagnosis of pyloric stenosis. Nevertheless, an “olive” is not always palpable. In these cases an ultrasound examination of the upper abdomen can be used to confirm the diagnosis. The upper limits for normal pyloric measurements are 4-mm thickness and 14-mm length. A limited upper gastrointestinal (UGI) series can also be used to confirm the diagnosis of pyloric stenosis. This may demonstrate an enlarged stomach, a long and very narrow pyloric channel, and indentation at the end of the gastric antrum (“shoulder”) representing the hypertrophied pylorus.

E. Treatment. The treatment for pyloric stenosis is surgical. However, since prolonged symptoms may result in significant dehydration and electrolyte abnormalities, adequate fluid resuscitation and correction of hypokalemic, hypochloremic metabolic alkalosis are mandatory before surgery.

At operation the pylorus is delivered through a small right upper quadrant transverse incision, although some prefer a supraumbilical incision or even a laparoscopic approach. The serosa is incised longitudinally along the length of the hypertrophied pylorus and the muscle fibers underneath are divided by stretching them in a direction perpendicular to the incision. Successful completion of this maneuver should result in a bulging of the pyloric mucosa through the pyloromyotomy and the ability to move the upper and lower halves of the pylorus independently.

Postoperatively, after a brief period of dietary restriction, the patient can be fed beginning with frequent small volumes of sugar water and progressing over 24–48 h to larger volumes of formula or breast milk. Episodes of emesis often occur with the early postoperative feeds. It is important to discuss this with parents prior to the operation to avoid undue postoperative concern.

When performing the pyloromyotomy, care must be taken not to enter the mucosa, especially on the duodenal side of the pylorus. If the lumen is entered, the mucosa should be closed with fine absorbable suture; the repair is buttressed with an omental patch. An alternative is to close the seromuscular layer in addition, and perform a new pyloromyotomy on the other side of the pylorus. A longer period of dietary restriction should be maintained if the lumen is entered.

Pyloric stenosis does not recur and the long-term prognosis is excellent.
Hypertrophic Pyloric Stenosis

A. Pathophysiology

B. Presentation
- Non-bilious emesis (projectile)
- Age: 2wks to 2 mos
- +/- dehydration

C. Palpable Epigastric mass (“olive”)

D. Ultrasound Or UGI

E. Rehydration - Correction of electrolyte abnormalities

- (+)
- (-)

Surgery (pyloromyotomy)

Continue evaluation for non-surgical causes for non-bilious emesis
A. Clinically apparent icterus in a neonate generally occurs when the serum bilirubin exceeds 5 mg/dl. This level may be somewhat lower for an older child. The bilirubin can be either unconjugated (indirect) or conjugated (direct), with the differential diagnosis for each being very different.

Physiologic (unconjugated hyperbilirubinemia) jaundice of the newborn has a multifactorial etiology which includes immaturity of the enzyme glucuronyl transferase which is responsible for bilirubin conjugation. Serum bilirubin concentration should be less than 13 mg/dl and usually resolves within 1 week. Other causes of unconjugated hyperbilirubinemia include breast-milk jaundice, hemolytic disorders, hypothyroidism, and familial disorders. Surgically treatable conditions include pyloric stenosis and other forms of intestinal obstruction.

B. Direct hyperbilirubinemia is considered pathologic when the conjugated fraction is greater than 20% of the total serum bilirubin concentration or when the conjugated level is greater than 2 mg/dl. Direct hyperbilirubinemia results from either hepatocellular disease whereby conjugated bilirubin cannot be excreted out of the hepatocyte into the bile duct canaliculi or functional or mechanical (obstructive) cholestasis.

The causes of direct hyperbilirubinemia vary by age, being different for the neonate and the older child. Surgical conditions in older children include choledolithiasis/cholecystitis and choledochal cyst; however, many medical conditions may progress to jaundice and end-stage liver disease requiring transplantation. Ultrasound quickly and reliably detects stone disease or cystic abnormalities of the biliary tract. The workup of these patients must proceed in a timely fashion. While culture and serological results are pending, evaluation for possible biliary atresia can be initiated with an abdominal ultrasound. This will exclude choledochal cyst or other common duct abnormalities and determine whether a gallbladder is present. Absence of the gallbladder is suggestive of biliary atresia although its presence does not exclude the diagnosis since the gallbladder may not be in circuit with a patent biliary tree but instead be filled with mucus or “white bile.”

A radionuclide biliary study (e.g., HIDA) is useful in distinguishing between obstructive and parenchymal causes for direct hyperbilirubinemia. Prompt hepatocyte uptake without excretion into the gastrointestinal (GI) tract suggests biliary atresia, while poor uptake is typical of hepatocyte dysfunction as seen in idiopathic neonatal hepatitis. Poor hepatocyte uptake and, consequently, HIDA scan accuracy may be improved by pretreatment with phenobarbital which increases biliary flow. A percutaneous liver biopsy may help in making the distinction between atresia and hepatitis by revealing either bile duct proliferation (biliary atresia) or focal necrosis (hepatitis) although there is some overlap of these histologies. The diagnosis is firmly established by laparotomy and intraoperative cholangiogram, demonstrating either the absence of a functional gallbladder or the lack of biliary continuity between the liver and duodenum.

C. Biliary atresia is characterized by progressive obliteration of the extrahepatic biliary tree with proliferation of small intrahepatic bile ducts, plugs of inspissated bile within the canaliculi, and periportal fibrosis. Although infectious or ischemic causes have been hypothesized, an etiology for the pathogenesis of biliary atresia has not been clearly established. These patients present in the first few weeks of life with jaundice and hepatomegaly, but generally appear well, in contrast to patients with an infectious etiology for their hyperbilirubinemia. Initially, stools may have normal color but soon become acholic, consistent with the progressive nature of biliary atresia.
D. A Kasai portoenterostomy is performed for biliary atresia by carefully dissecting around the porta hepatis and anastomosing small bowel to a fibrous area around the central area of exposed liver parenchyma. Despite a technically well-performed operation, the results from this procedure are variable. About one third of the patients will have a good result and remain anicteric, while another third of the patients will never drain bile and will progress to liver failure. The remaining patients often get some relief from their jaundice but over a longer period of time ultimately suffer end-stage liver failure. One factor in the prediction of outcome for these patients is the age at operation. Those performed within the first 2 months of life have a significant survival advantage, whereas there is probably no likelihood of success after 4 month of age. The only salvage therapy for patients with a failed Kasai is liver transplantation.

E. Choledochal cyst is a congenital malformation of the biliary tree and can be categorized into five subtypes according to a modification of the Alonso-Lej classification: I—cystic dilatation of the common bile duct (most common type), II—diverticulum of the common bile duct, III—choledochocele, IV—intra- and extrahepatic cysts, and V—intrahepatic cysts only (Caroli’s disease). Infants with a choledochal cyst will present with jaundice from distal common bile duct obstruction as their only symptom, while older children may also have abdominal pain and an abdominal mass. This condition is more common in females and Asians and its pathogenesis is unknown; the theory that it is due to pancreatobiliary reflux is unproven. The cyst wall is typically fibrotic with little remaining epithelial lining.

The diagnostic modality of choice is an abdominal ultrasound. If uncertainty remains a hepatobiliary radionuclide scan or CT may provide additional information. In older patients an endoscopic retrograde cholangiopancreatography (ERCP) can be performed to confirm the diagnosis. The treatment for Type I cysts is cyst resection with internal biliary drainage established via a Roux-en-Y hepaticojunostomy. Cholecystectomy is also usually performed. If the cyst is densely adherent to the portal vein, the posterior cyst wall can be left in place after the mucosa has been excised. Simple cyst-enterostomy should be avoided because of the high incidence of stricture formation and the possibility of future malignancy. Type II cysts may be treatable with diverticulectomy while Type III require transduodenal cyst unroofing and sphincteroplasty. Types IV and V are more challenging and may require segmental liver resection or even liver transplantation.
Jaundice in the Pediatric Patient

A. Neonate with jaundice

Indirect Hyperbilirubinemia
- Direct Hyperbilirubinemia
  - e.g., cystic fibrosis, alpha-1 antitrypsin deficiency

B. Direct hyperbilirubinemia
- Zmg/dl or 20% total bilirubin

C. Biliary atresia
- Poor hepatocyte uptake
- Neonatal hepatitis

D. Liver transplant
- Failed surgery tailored to cyst type

E. Choledochal cyst (type I-V)
- Abdominal ultrasound
- No gallbladder
- No ductal excretion
- HIDA
- (-)

r/o sepsis
- e.g., metabolic, inherited disorders, hypothyroidism

r/o sepsis
- e.g., cystic fibrosis, alpha-1 antitrypsin deficiency

Jaundice in the Pediatric Patient
Pediatric Abdominal Masses

Andrew Davidoff

A. General Considerations. Abdominal masses of infants and children may be caused by congenital anomalies, tumors, trauma, infection/abscess, or organ enlargement. Patient’s age significantly influences the differential diagnosis. An ultrasound examination should be performed early during the workup; it can quickly and accurately identify the location of the mass, the organ with which it is associated, and whether it is cystic or solid. Additional imaging studies such as CT scan, barium enema, or voiding cystourethrogram may be required to confirm the diagnosis.

Many abdominal masses are found by parents while bathing a child or observing that clothes are no longer fitting, or by a physician during a routine visit. Symptoms of pain or fever imply infection; however, congenital anomalies and tumors may present with these symptoms as well. Patients with constipation or fecal impaction may have a palpable colon; the history and physical exam should be able to identify this and preempt an unnecessary and expensive workup.

B. Congenital Anomalies. Congenital anomalies of the genitourinary tract may present as an abdominal mass. Examples include enlarged polycystic kidneys, hydronephrotic kidneys secondary to ureteropelvic junction obstruction, or an enlarged bladder from obstruction of the posterior urethral valves. These entities can be diagnosed by ultrasound and voiding cystourethrogram. The bladder may be enlarged from neurogenic causes such as spinal anomalies or trauma.

The uterus and vagina may become distended if they are obstructed by an imperforate hymen. In infants, a hydrometrocolpos will develop in response to maternal estrogen stimulation. At menarche, patients may present with hematometrocolpos as the uterus and vagina become filled with blood from menses. These conditions are diagnosed during physical examination by finding a bulging hymenal membrane; treatment is with simple hymenotomy. One must also always consider the possibility of pregnancy in a young girl with an enlarged uterus.

Congenital anomalies of the gastrointestinal (GI) tract, such as duplications, may also present as abdominal masses. One third of these are discovered in the newborn period; the remainder are usually diagnosed by 2 years of age. Omental or mesenteric cysts may present in a similar manner. A meconium cyst from prenatal perforation may present as a mass in a newborn. A characteristic “sausage-shaped” mass in the right upper quadrant may be found in infants with intussusception. Hepatobiliary anomalies such as a liver cyst or choledochal cyst may present as right upper quadrant abdominal masses. In general, GI tract anomalies may be initially identified by ultrasound, but anatomy and involvement of surrounding viscera and blood vessels are better defined by contrast enema or CT scan depending on the organ of origin.

C. Tumors. The annual incidence of malignant tumors in the pediatric population is about 1–2 per 10,000 children younger than 15 years of age. Approximately one third of these are solid tumors, many of which originate in the abdomen. The most common abdominal tumors are neuroblastoma and Wilms’ tumor; less commonly encountered are liver tumors, germ cell tumors, and rhabdomyosarcoma.

Neuroblastoma is the most common extracranial solid tumor of infants and children. It arises from cells of neural crest origin, most often within the adrenal gland. One fourth of the cases are diagnosed by age 1, 50% by age 2, and 90% by age 8. An abdominal mass is palpable in the majority of patients; symptoms vary depending on tumor stage. These children are often ill appearing with systemic manifestations of advanced disease. Radiologic imaging may begin with an ultrasound or CT scan; a bone scan and/or I123-metaiodobenzylguanidine (MIBG) scan can then be performed to search for metastatic disease.

Wilms’ tumor is the most common renal neoplasm of infancy and childhood and has a mean age of about 3 years at presentation. The most common presentation is an asymptomatic abdominal mass noted either by the parents or by a physician on a routine exam. Rarely, a child may present with abdominal pain and if the mass is not appreciated, a mistaken diagnosis of appendicitis may be made. The preoperative workup generally consists of an abdominopelvic CT scan as well as a CT scan of the chest.
Liver tumors usually present as a right upper quadrant abdominal mass with or without pain. *Over two thirds of pediatric liver tumors are malignant*; hepatoblastoma occurs in children less than 4 years of age (usually less than 2) and hepatocellular carcinoma occurs in older children and adolescents. Useful serum markers for these tumors include serum α-fetoprotein and ferritin levels. An abdominal CT is important for defining the anatomy and determining resectability. An arteriogram or MRA may also be helpful in further defining the relationship of the tumor to the liver vessels.

Tumors arising from primordial germ cells may occur at any location within the abdomen but are most commonly found in the ovary. Their histology may vary from a benign mature teratoma (40% of all ovarian tumors) to a highly malignant choriocarcinoma. The peak age range for ovarian tumors is 10–14 years. These patients may be asymptomatic or have abdominal pain. Acute, severe pain may be due to tumor rupture or ovarian torsion. Most cases of ovarian torsion are felt to occur in conjunction with the presence of either an ovarian tumor or a cyst. An ultrasound can demonstrate the presence of a large ovarian cyst; the finding of a solid ovarian mass should prompt a more extensive workup. Serum α-fetoprotein is a useful tumor marker as is β-HCG (human chorionic gonadotropin).

Soft tissue sarcomas, of which rhabdomyosarcoma is the most common type, may originate in the retroperitoneum (8%) and can present as an abdominal mass. CT scans are used to determine resectability and the presence of metastatic disease.

Although lymphoma is relatively common in children, it rarely presents with an abdominal mass. Aggressive non-Hodgkin’s B-cell lymphomas (e.g., Burkitt’s lymphoma), however, usually originate in the abdomen and occasionally present as an abdominal mass.

D. An *intra-abdominal abscess* may manifest as a mass. The most common etiology in a child would be an appendiceal abscess. Ultrasound or CT scan may confirm the diagnosis. Some surgeons treat an appendiceal abscess initially with antibiotics and occasionally percutaneous drainage followed, perhaps, by an interval appendectomy. A patient with inflammatory bowel disease may have an abdominal mass secondary to bowel inflammation or a walled-off perforation of a segment of diseased intestine. Differentiating this from an appendiceal abscess may be difficult. In a neonate, complications of necrotizing enterocolitis, such as perforation, abscess, or a fixed loop of obstructed bowel, can present as an abdominal mass. These are indications for operative intervention. Other causes of intra-abdominal abscess include a tuboovarian abscess or intestinal perforation from a foreign body.

E. A *pancreatic pseudocyst or hematoma* of the rectus abdominus, retroperitoneum, or solid organ may develop after blunt abdominal trauma and present as an abdominal mass. Ultrasound or CT scan should confirm the diagnosis. Hematomas are generally treated nonoperatively. The possibility of child abuse should be considered if the history does not match the clinical findings.

F. *Enlargement of any intra-abdominal or retroperitoneal organ*, the spleen or liver in particular, may present as an abdominal mass. The causes are varied and may be due to metabolic disorders (e.g., glycogen storage disease or Gaucher’s disease), vascular etiologies (e.g., portal hypertension), or infiltrative diseases, either neoplastic (e.g., leukemia) or non-neoplastic (e.g., sarcoidosis). An ultrasound is a good screening tool when considering these diagnoses. These conditions are generally treated nonsurgically.
Pediatric Abdominal Masses

A. General Considerations
   - pain/fever?
   - constipation?
   - imperforate hymen?
   - jaundice?
   - antecedent trauma?

   ultrasound

   additional radiographs as needed

B. Congenital Anomalies
   - GU-polydysic kidneys
   - hydronephrosis
   - enlarged bladder
   - imperforate hymen

C. Tumors
   - neuroblastoma
   - Wilm's tumor
   - hepatic tumor
   - ovarian tumor
   - sarcoma
   - lymphoma

D. Infection
   - appendiceal abscess
   - inflammatory
   - bowel disease
   - NEC
   - tuboovarian abscess

E. Trauma
   - pseudocyst
   - rectus hematoma

F. Organomegaly

GI-duplications
   - mesenteric cysts
   - meconium cysts
   - hepatobiliary cysts
Esophageal Atresia/Tracheoesophageal Fistula

Rashmi Kabre and Srikumar Pillai

A. Incidence/Associated Defects. Esophageal atresia (EA) and tracheoesophageal fistula (TEF) are relatively common malformations occurring in 1 in 3,500 births. Although the etiology is unknown, approximately half of these cases occur with other anomalies, with most infants having more than one malformation. Cardiac defects alone account for 35% of these anomalies. The VACTERL association (vertebral defects, anorectal malformations, cardiac defects, tracheoesophageal anomalies, renal anomalies, and limb defects) has been used to describe the array of anomalies found in these patients. With high clinical suspicion, prompt diagnosis and appropriate clinical management can improve survival in these infants.

B. Anatomic Variants. Using the gross classification system, five basic variants of EA/TEF are encountered: (1) Type A—pure esophageal atresia, (2) Type B—esophageal atresia with proximal tracheoesophageal fistula, (3) Type C—esophageal atresia with distal TEF, (4) Type D—esophageal atresia with both proximal and distal TEF, and (5) Type E—tracheoesophageal fistula without esophageal atresia, more commonly known as “H type.” Type C occurs most often (85%) with a fistula that passes at the level of the carina to the lower esophageal segment. Type A is the next most common (8%), followed by Type E (4%), Type D (2%), and Type B (1%). Type E fistulas tend to be located high on the trachea.

C. Presentation. Infants with esophageal atresia present with varying degrees of respiratory distress, excess oropharyngeal secretions, and marked feeding intolerance shortly after birth. The secretions are classically described as fine, white, and frothy bubbles of mucus in the mouth or nose that return despite aggressive suctioning. The infant may have episodes of coughing, choking, cyanosis, and aspiration resulting from esophageal obstruction. Those with a distal fistula may have a distended abdomen and may aspirate refluxed gastric contents. If no fistula exists, the abdomen will be scaphoid. A patient with a tracheoesophageal fistula alone without esophageal atresia (H type) may present at a somewhat older age and with more subtle symptoms such as intermittent choking, cyanosis with feeding, or recurrent pneumonia.

D. Diagnosis. If esophageal atresia is suspected, a radiopaque 8 French (in preterm infants) or 10 French (in term infants) nasogastric tube should be passed into the stomach, which is normally located ~17 cm from the mouth. Inability to pass a nasogastric tube into the stomach of a newborn, with an x-ray of the entire abdomen demonstrating the tube to be coiled in the neck or upper chest, confirms the diagnosis of esophageal atresia. Intraluminal gas within the abdomen suggests the presence of a distal tracheoesophageal fistula (Type C or D). The absence of intraluminal gas under the diaphragm suggests either that no fistula exists (Type A) or that the fistula is to the proximal esophagus only (Type B). In a gasless abdomen, bronchoscopy may help determine whether the patient has a proximal fistula. A contrast study is rarely necessary but if performed should be done with only a small amount of dilute barium, as the risk of aspiration pneumonitis and reactive pulmonary edema is high. In the older infant in whom an H-type fistula is suspected, a plain film is usually normal. This diagnosis can be confirmed with an esophogram and/or bronchoscopy, although several attempts may be required to demonstrate the tract.

E. Medical Management. Once the diagnosis has been established, aspiration precautions should be taken, including continuous suctioning of the upper pouch with a sump tube, elevation of the infant’s head, intravenous fluid resuscitation, and oxygen therapy as needed. With the suspicion for sepsis or pulmonary infection, broad-spectrum antibiotics should be administered. Because of the high incidence of associated congenital anomalies, a thorough cardiac assessment (echocardiogram) as well as renal ultrasound should be performed prior to any surgical undertaking. Plain films will aid in detection of any skeletal abnormalities. Delayed surgical repair is preferable for infants with low birth weight, pneumonia, or other major anomalies. If ventilation becomes a problem for the premature infant with a fistula, the endotracheal tube can
be passed deeply to cover the tract, or the fistula can be ligated without attempting to repair the esophagus.

**F. Surgical Therapy.** Surgical treatment involves (1) primary repair with an esophagoesophagostomy when possible and (2) ligation of the fistula when present. The surgical approach is via a right lateral thoracotomy incision. The tracheoesophageal fistula is identified, usually behind the azygous vein, and divided. The upper esophageal pouch is identified and can be mobilized if needed, with care being taken to identify an upper fistula if present. Further length of the upper pouch can be achieved by performing one or two circular myotomies. The lower length of esophagus should be subjected to minimal dissection because of its segmental blood supply. A single layer full-thickness anastomosis is performed with interrupted sutures. Some surgeons place a transanastomotic nasogastric feeding tube so that early enteral feedings may be instituted. A chest tube may also be placed to drain any subsequent anastomotic leaks. Depending on surgeon’s preference, an esophogram may be obtained 5–7 days after surgery. Oral feedings are initiated if no leak is demonstrated.

If the patient is found to have a wide-gap atresia, two other scenarios may occur. Those with pure esophageal atresia without a fistula (Type A) undergo gastrostomy tube placement initially with subsequent esophageal dilations. Patients with wide-gap atresia with a fistula will also undergo gastrostomy tube placement, in addition to ligation of the fistula. Elective repair of both groups of patients is performed 3 months later. If at this time, the gap is still too wide, the patient may subsequently require reconstruction with the use of an esophageal substitute (e.g., gastric pull up, reversed gastric tube, and colon or small intestine interposition).

Type E defects (without esophageal atresia) can be approached simply via a left cervical incision through which the fistula is identified and divided. Identification of the fistula can be made easier by placing a catheter or stent across it under direct bronchoscopic visualization performed prior to the neck exploration.

**G. Complications.** An anastomotic leak is an early complication of surgical repair, especially if the repair was under tension or ischemia from mobilization occurred. This should resolve with hyperalimentation (or distal enteral feedings) as long as the leak is well drained with chest tubes. Esophageal strictures develop as a late complication and are usually secondary to an early anastomotic leak. These strictures generally respond to dilatations (usually repeated) and rarely require subsequent resection. Tracheomalacia, felt to result from in utero compression of the trachea by the distended upper esophageal pouch, can also be a problem for these patients. Most neonates who undergo repair will have some degree of esophageal dysmotility. Long-term, 60–80% of patients with repaired esophageal atresia will suffer from gastroesophageal reflux. Of these, one half respond to medications, with the remainder of patients requiring surgical correction.

**H. Outcomes.** The outcome for patients with esophageal atresia depends on a number of factors but most important are the associated anomalies, particularly the presence and severity of a cardiac defect. The patient’s gestational age, weight, and degree of pulmonary insufficiency also impact on the clinical outcome.
A. Incidence, associated defects
1/3500 births
VACTERL anomalies

B. Anatomic Variants
Atresia with fistula to lower esophagus in 85%

C. Presentation
Respiratory distress
Excess secretions
Feeding intolerance

D. Diagnosis
Pass NG tube
Abdominal X-ray
Bronchoscopy
UGI - use with caution

E. Medical Management
Aspiration precautions
IV fluids
Antibiotics
Cardiac evaluation

F. Surgical Therapy
Ligate fistula
Resect atresia
Primary esophageal repair

G. Complications
Anastomotic leak
Stricture
Tracheomalacia
Esophageal reflux

H. Outcomes
A. Introduction. Intussusception is the invagination of one segment of intestine—the intussusceptum—into another adjacent segment of intestine—the intussuscipiens. Most cases of intussusception are idiopathic, though identifiable causes are increasingly common as patient’s age increases. Identifiable causes are usually intraluminal or intramural anatomic abnormalities that are “picked up” by the bowel mucosa and passed downstream, carrying the attached bowel along with it and thus causing the invagination. In the pediatric population, an anatomic lead point is identified in <10% of cases, whereas in the adult population, a lead point is identified in as many as 97% of cases. When an identifiable lead point is present, patients are less likely to respond to non-operative therapies. The age at which a lead point becomes more common is 4 years, after which lead points are found in over 50% of patients. Among children, the most common anatomic lead point is a Meckel’s diverticulum, followed by polyps of the ileum and colon, hamartomas associated with Peutz-Jeghers syndrome, submucosal hemorrhages associated with Henoch-Schonlein purpura, lymphoma, lymphosarcoma, enteric cysts, inverted appendiceal stumps, and anastomotic suture lines. About half of adult cases of intussusception are due to malignancy.

Surgeons evaluating children under the age of 5 for acute abdominal pain should have a high suspicion for the presence of intussusception. This diagnosis is responsible for up to 25% of abdominal surgical emergencies in this age group. Of all diagnosed cases of intussusception, 80–90% occur in children aged 3 months to 3 years. The “classic” triad of intermittent abdominal pain, emesis, and currant-jelly stools is seen in less than 20% of cases. In one series, the most common symptoms associated with intussusception were vomiting (up to 90%), intermittent abdominal pain (up to 90%), fever (50%), currant-jelly stools (25%), palpable abdominal mass (15%), and hematemesis (10%). Thus the most typical presentation is emesis with intermittent abdominal pain. The pain is often described as colicky, short in duration, and returns in 20–30-min intervals. Each episode may be associated with pulling the knees up toward the abdomen, as well as fever, diaphoresis, and a bowel movement. Once the pain has subsided, the child may appear well until the next episode. As the episodes continue, the child will appear increasingly ill and distended, with continued emesis and signs of small-bowel obstruction. Ultimately if the blood supply to the intestinal mucosa is sufficiently compromised, it will begin to slough and the “red currant-jelly” stools will be notable. Continued delay in diagnosis and therapy can lead to shock and ultimately to cardiovascular collapse. It is important to remember the spectrum of time associated with this disease, as it is easy to evaluate a patient during one of the quiescent episodes and not be impressed with the severity of the illness.

B. Diagnostic Evaluation. By the time the child is evaluated, often several episodes have occurred and though the child often appears normal, he/she may also appear lethargic or even hypotensive and tachycardic. Notable findings on physical exam are usually limited to the abdomen where one can often feel an abdominal mass described as “sausage like” that is located in the right or supraumbilical abdomen. Another pertinent finding, the sign of Dance, is the inability to palpate the cecum in the right lower quadrant. Both of these findings can be difficult to elicit, especially if the child is crying or guarding. On rectal exam, most children will have gross blood, mucoid bloody stools, or hemoccult positivity. On rare occasions the examiner may even note the intussusceptum prolapsed through the anus.

Abdominal radiographs often reveal a normal or nonspecific bowel gas pattern. As the duration of symptoms progresses, however, the clinician is more likely to find films consistent with a small-bowel obstruction. In 25–60% of cases, one may even see a soft-tissue density in the right upper quadrant. This finding may be confirmed by careful review of the lateral film that may show displacement of upper abdominal gas inferiorly, or a distinct separation of bowel gas between the upper and lower abdomen with the intervening soft-tissue density. Other plain radiograph findings include a poorly visualized cecum and lateralization of the small bowel into the right lower quadrant. Obviously, any child with clinical or radiographic evidence for
peritonitis or perforation should not undergo an enema, but rather should go directly to an exploratory laparotomy.

C. Enema. Given adequate concern for the presence of an intussusception coupled with clinical stability, the child is then advanced to a contrast enema. This can be performed in the fluoroscopic suite with direct visualization of the colon during administration of contrast. If an intussusception is seen, the radiologist can attempt to reduce it in one of three ways.

Hydrostatic reduction involves placement of a noninflatable rectal tube and administration of barium or another contrast material by gravity. Depending on the contrast chosen, the height of the fluid column should be adjusted to create an intraluminal pressure of no greater than 120 mmHg. Pressures at this level or less will not reduce gangrenous bowel. This should be carefully monitored under fluoroscopic visualization, as failure of progressive reduction of the intussusception can lead to a risk of perforation. Successful reduction should be confirmed by reflux of contrast into the terminal ileum, though on occasion edema of this region may hinder this finding. Considering this possibility, some suggest close observation rather than operative therapy if reduction is believed to have been successful, but reflux of contrast into the terminal ileum is not visualized.

The second method of reduction also consists of administration of an enema, with constant observation by ultrasound. This method uses a saline and water-soluble contrast solution so that reflux of contrast into the terminal ileum can confirm successful reduction with an abdominal flat plate film. This method is noted to have similar rates of success, with less radiographic exposure. The limitations of this method include its dependence on a talented ultrasonographer, as well as the presence of a significant bowel obstruction as the air in the bowel will obstruct the US signal.

The third method of reduction is pneumatic. Air is insufflated into the colon to a starting pressure of 80 mmHg, and again under fluoroscopic guidance, the intussusception is visualized, and progressive reduction is constantly monitored. The pressure may be increased to 120 mmHg just as in the hydrostatic method, and success is determined by reflux of air into the small bowel. It is believed by some that this method is safer as it allows constant monitoring of the intraluminal pressure, as well as creates a more evenly distributed pressure along the length of the colon.

Several studies have shown that it is safe to continue attempts at reduction via enema as long as continued progression of the reduction is seen. If there is not any progression, the attempt should stop after 3–5 min. Longer attempts simply place the child under increasing risk of perforation without any improvement in the odds of reduction.

D. Operative Therapy. Children who have clinical or radiographic evidence of peritonitis or perforation, and children who fail radiologic reduction, should undergo operative reduction. After appropriate fluid resuscitation and broad-spectrum antibiotics, the operation is typically begun with a right lower quadrant transverse incision that can be extended across the midline if necessary. Once the intussusception is identified, it should be reduced by gentle compression of the distal end, rather than pulling from the proximal end, much in the way that toothpaste is squeezed from a tube. Traction may result in tearing of compromised bowel, compelling an unnecessary resection. Once the intussusception is reduced, it must be carefully inspected for viability and tears. A few cases of unrecognized longitudinal tears along the anti-mesenteric border of the colon under the taenia have been described. If vascular compromise is suspected, one should observe the bowel for 10–15 min prior to performing a resection. At this time, the surgeon should also inspect the bowel carefully for lead points that can be removed, being careful, however, to recognize that neither an edematous ileocecal valve nor a Peyer patch should be mistaken for a resectable mass. Finally, given the location of the incision, an appendectomy should be performed. If gangrenous bowel is encountered, a resection and primary anastomosis is still a viable option unless the patient is ill enough that one should minimize the duration of anesthesia, in which case the surgeon should bring out an ileostomy and mucus fistula.

Each of the described non-operative therapies is associated with a risk of perforation, and clearly if this complication is suspected, one should progress immediately to surgery.

E. Recurrence. The risk of recurrence is 5–10% after non-operative therapy, almost two thirds of which occur within the first 3 days of reduction. Recurrences have been noted, however, to occur as long as 3 years after the initial reduction. Recurrence after operative therapy is lower, usually less than 5%. As with the initial occurrence, recurrent intussusception should also initially be treated with attempts at non-operative reduction, unless a surgically correctable lead point is suspected. These cases include children over the age of 3 with multiple recurrences and children with polyposis syndromes.

Finally, age is an important factor when considering the diagnosis and treatment of intussusception. Neonates, like adults, are more likely to have a surgical lead point for an intussusception—a diagnosis that is rare in this age group. Given that this likelihood is 60–75%, that enema reduction has a low rate of success, and that the risk of perforation is higher, surgery is recommended once the diagnosis is made.
Intussusception

A. Clinical Manifestations
1.) intermittent abdominal pain
2.) emesis
3.) fever
4.) abdominal mass
5.) currant-jelly stools

B. Diagnostic Evaluation
1.) Physical exam
2.) abdominal radiographs

C. Enema
1.) Hydrostatic
2.) US-Guided
3.) Pneumatic

D. Operative Therapy
1.) Manual Reduction
2.) Resection with anastomosis
3.) Resection with ileostomy and mucous fistula
4.) Appendectomy

E. Recurrence
Repeat enema unless lead point suspected then proceed to operative therapy
**Definition.** The defining characteristic of Hirschsprung’s disease (HD) is the absence of ganglion cells over a varying length of intestine. It always involves the rectum and extends proximally for an individually variable distance. The disease is limited to the recto-sigmoid colon in 75–80% of cases, though it may extend to the transverse colon in as many as 17% of cases, to the entire colon and terminal ileum in 5–8% of cases, and rarely extend to include the entire intestinal tract to the duodenum in the rarest of cases. Male:female ratio is ∼4:1 for all cases, though this ratio trends toward 1.5:1 as the length of involved intestine increases. The incidence is estimated to be 1 in 5,000 live births.

**Etiology.** The disease is defined by the absence of ganglion cells. Ganglion cells are involved in bowel relaxation and motility, and their absence leads to chronic contraction of the affected region of bowel causing a functional obstruction. This can lead to chronically thickened and dilated proximal bowel. Although the absence of ganglion cells is clearly implicated in the etiology of HD, there are many theories as to the cause of this absence.

The first theory proposes that the disease is due to a failure of the proper migration of neural crest cells during fetal development. Neuroblasts appear in the developing esophagus around the 5th week and migrate down the intestinal tract outside of the circular muscle layer reaching the anus around the 12th week. The longitudinal muscle layer then forms while these neuroblasts differentiate into the myenteric plexus. At the same time in the 12th week, again beginning at the esophagus and progressing distally until the 16th week, the newly differentiated myenteric plexus migrates across the circular layer into the submucosa and mucosa, further differentiating into the submucosal plexus. As these two plexi ultimately control bowel motility, failure of this migration at any point along this process will result in a variable length of affected bowel, beginning at the most distal point and extending over a caudal to cranial length.

A second theory proposes that the absence of ganglion cells is not due to a failure of migration, but rather to a failure of differentiation of neuroblasts secondary to changes in the local microenvironment. It has been shown that certain extracellular matrix proteins affect the path of neuroblast migration, while others affect maturation and differentiation. Absence or abnormalities in any of these proteins could therefore affect proper development and function of the intestinal ganglion plexus.

Others have suggested that an immunologic mechanism may be responsible for HD given the presence of overexpression of ICAM-1 and MHC class II antigens on hypertrophic nerve trunks. Finally, others have suggested that HD has a genetic component given its familial relationship, its association with Down’s syndrome, and its mapping in the dominant form to chromosome 10. In addition to demonstrated autosomal dominant events, it has also been shown to be autosomal recessive, as well as polygenic.

**Clinical Presentation and Physical Examination and Immediate Management.** A. Children normally pass meconium within the first 24 h of life. The most common presentation in neonates is failure to pass meconium within these first 24 h, a finding present in 90% with the diagnosis of HD. Other common signs on presentation include abdominal distention and bilious emesis. Often if a rectal examination or rectal irrigation is performed, the meconium will pass with relief of symptoms for a time followed by a return of obstructive symptoms in a few days to weeks. Children who present with these mild symptoms should progress to the radiologic workup below.

**Diagnostic Evaluation.** B. Radiologically, the diagnosis can be suggested both by abdominal radiographs and by barium enema. Plain abdominal films may show a pattern consistent with a bowel obstruction with dilated loops of bowel and air-fluid levels. Closer review of these films, however, may reveal air in a nondistended rectum, especially on a lateral film. It is important also to evaluate carefully the plain films for pneumatoses as well as signs of perforation.

C. A smaller percentage of children may present with enterocolitis. Rectal examination on these children will often result in a telltale release of explosive gas and foul-smelling liquid stools. Their initial complaints can be either constipation or diarrhea, or if it is allowed to progress prior to presentation,
with shock secondary to toxic megacolon. These children should be promptly admitted to the ICU for intravenous (IV) fluid resuscitation, electrolyte replacement, broad-spectrum antibiotics, nasogastric (NG) decompression, and rectal irrigation. Once these children are clinically stable, they may progress through the normal workup for HD.

D. Prolonged delay in presentation, however, can also present with perforation. This group of children, after resuscitation, should be taken to the operating room as soon as possible.

Presentation is sometimes subtle, delaying diagnosis for months to even years. Changes in feeding regimens, chronic use of laxatives, enemas, and suppositories all can help mimic normal health and prevent prompt presentation. This is especially true of children with “short-segment” HD.

It is also important to be aware of other syndromes that are associated with the diagnosis of HD and the presence of one should prompt a search for the others. These include trisomy 21, Waardenburg syndrome, and cartilage-hair hypoplasia. Additionally, there is a very rare association with HD and congenital central hypoventilation syndrome (Ondine’s curse)—only 48 cases of which have been reported. Of these, a few have also been found to have neuroblastoma, an association termed neurocristopathy.

E. Historically, the barium enema was used for diagnosis and is still quite indicative of the presence of HD. Rectal irrigation and manipulation prior to the contrast enema can impede interpretation as these maneuvers may dilate the usually contracted rectum. Films that suggest the presence of HD typically have a narrow rectum with a cone-shaped portion of bowel extending up into a dilated proximal bowel. Cases in which the barium enema is difficult to interpret include “short-segment” HD, as well as total colonic HD.

F. Rectal biopsy has been the gold standard in the diagnosis of HD. Rectal biopsy is usually completed at the patient’s bedside via a suction rectal biopsy gun. An absence of ganglion cells, the presence of hypertrophied nerve fibers, and an excess of immunohistochemical staining for acetylcholinesterase are indicative of HD.

G. Some institutions are using anorectal manometry to help in the diagnosis of HD. Findings on this exam were first reported in 1964 and are described as an absence of relaxation of the internal sphincter after dilation of the rectum. Although many agree to its sensitivity in older children, there is still debate as to the sensitivity in neonates.

Operative Therapy. As stated above, the initial management of a child with HD and enterocolitis includes IV fluid resuscitation and electrolyte correction, NG decompression, and broad-spectrum antibiotics. Patients may also benefit from rectal irrigation and decompression via a rectal tube. As decompression is the goal, it is important to be sure that at the very least all of the irrigant is recovered. In assessing a patient’s operative risk, it is also important to evaluate for other potential congenital anomalies.

H. The two main principles in treating children with HD and enterocolitis that have had tremendous impact on the successful improvement in outcome have been the administration of total parenteral nutrition and the creation of a diverting colostomy. The importance of the colostomy should not be overlooked—this has been the mainstay of therapy and often the life-saving maneuver. Historically, an operation in a newborn was a much more morbid undertaking than it is today. Children therefore typically underwent a diverting colostomy with decompression and biopsies, followed by a definitive pull-through operation at 6–12 months of age.

I. Three procedures have dominated treatment and remain the definitive treatment for HD today—the Swenson, the Duhamel, and the Soave pull-throughs. The Swenson procedure was first published in 1948 and involves complete resection of the aganglionic bowel to within 1.5 cm from the anorectal line anteriorly and 1.0 cm posteriorly, thus preserving the internal sphincter. An end-to-end anastomosis is created externally between the normal proximal end of bowel pulled through and sutured to an everted rectal stump. The Duhamel procedure was first described in 1956 and modified over the subsequent 10 years. In this procedure, the aganglionic rectum is not resected, and the proximal functional bowel is brought into the pelvis posteriorly. The two lumens are then unified using a long GIA stapler, creating a single common rectal reservoir. Finally, the Soave procedure, described in 1964, involves resection of a tube of mucosa and submucosa from the aganglionic bowel to within 1.0 cm of the anorectal line. The normal proximal bowel is then brought through the remaining muscular sleeve and an anastomosis is created at the anus. The proposed advantage of the Duhamel and Soave procedures is their avoidance of dissection along the anterior rectal wall, which may disrupt pelvic nerves responsible for continence and sexual function.

Given improvement in neonatal and perinatal diagnosis of HD, coupled with significant improvements in anesthetic techniques and perioperative neonatal care, many surgeons now perform primary pull-through operations rather than continuing with the two-staged approach. The introduction of laparoscopic surgery is also beginning to redefine operative time, extent of dissection, length of hospital stay, time to recovery, and degree of complications.

Complications. J. Even after a definitive pull-through, children still are at an increased risk for development of enterocolitis. The reason for this is unknown, but it is important to remember this possibility in any child who has the diagnosis of HD and presents with bowel complaints. As before, these children should be treated with IV fluid resuscitation and electrolyte correction, NG decompression, bowel rest, and broad-spectrum antibiotics. Often these children will again need rectal irrigation and decompression, either at the bedside or in the operating room. If a child does not improve given these maneuvers, one must always consider returning to the basic principles of total parenteral nutrition and diverting colostomy.
Hirschsprung’s Disease

A. Neonate
- No meconium in 24 hours
- Abdominal Distention
- Bilious emesis

B. Abdominal Series
- Free Air?
- Enterocolitis?
- Pneumatosis?
- Sepsis?

D. IV Fluid Resuscitation
- Electrolyte replacement
- Broad spectrum antibiotics
- NG tube decompression
- Exploratory Laparotomy with leveling colostomy and open BX

C. IV Fluid Resuscitation
- Electrolyte replacement
- Broad spectrum antibiotics
- NG tube decompression
- Rectal tube decompression/irrigation

E. Barium Enema

F. Rectal suction Bx

G. No ganglion cells
- Acetylcholinesterase
- Hypertrophied nerve trunks
- Rectal manometry

H. Dx Hirschsprung’s Disease

I. Swenson
- Duhamel
- Soave
- Laparoscopic

J. Clinical decompensation
- Sepsis
- Profound recurrent enterocolitis
Necrotizing Enterocolitis

Kathryn D. Bass

A. Pathophysiology. As neonatal intensive care improves, the survival of infants of early gestation (<28 weeks) and very low birth weight (VLBW <1,000g) increases. Necrotizing enterocolitis (NEC) is a disease that has evolved in part from these advances. Ninety percent of all cases occur in the premature infant; the other major risk factor is early formula feedings. Feedings that are advanced at a rapid rate and volume create the greatest risk. Perinatal events resulting in hypothermia, hypotension, and hypoxia are associated with the onset of NEC. Untreated cardiac shunts such as patent ductus arteriosus (PDA) and the medical therapy of indomethacin for ductus closure have been cited. Umbilical catheters may result in embolic events to the gut. Cocaine exposure may result in vasospasm and relative ischemia of the splanchnic circulation. High-dose vitamin E used to treat retinopathy of prematurity has been shown to impair intracellular bacterial kill by leukocytes and is associated with higher incidence of NEC. Xanthine derivatives (theophylline and aminophylline) are known to slow gut motility and create oxygen radicals as part of their metabolism which may damage enterocytes. Although these agents and events occur commonly during the newborn period, in the setting of the vulnerable premature host receiving formula feedings, NEC must be considered early in the differential diagnosis of an infant with a worsening clinical condition.

The premature infant has an immature gut barrier and poor motility as a baseline. Early feedings of formula or medicines may result in a relatively hyperosmolar load to the bowel. Prematurity also results in a moderate to severe hypogammaglobulinemia (IgA and IgM), poor phagocyte function, and deficient complement levels. Stasis after feedings results in bacterial overgrowth, invasion through the bowel wall, poor bacterial clearance, and the onset of the inflammatory cascade. Elevated levels of platelet-activating factor and tumor necrosis factor promote mucosal bowel wall injury, creating a portal for more bacterial invasion. Transmural bowel injury occurs which can result in necrosis, perforation, sepsis, and possible death.

B. Physical Findings. Infants with NEC may present with lethargy and poor peripheral perfusion reflecting sepsis. A systolic murmur may be present. Abdominal distension is usually found with visible bowel loops in some cases. Focal or diffuse tenderness occurs as peritonitis progresses. The abdominal wall may be edematous with a doughy texture. Abdominal wall color changes reflect the underlying bowel pathology and may start as erythema and progress to gray or blue usually associated with dead bowel. Stools may be bloody as a result of sloughing gut mucosa.

C. Laboratory Findings. Leukocytosis is variable depending on the age of the infant and the severity of disease. Leukocytopenia (total count <6,000) is a common finding in gram-negative infection. Counts below 1,500 reflect significant sepsis and carry a poor prognosis. Thrombocytopenia is consumptive, the end result of the inflammatory cascade and the release of prostaglandins and cytokines. Disseminated intravascular coagulation may also occur accounting for persistent consumption of platelets. Hypovolemia results from the capillary leak of sepsis leading to metabolic acidosis. Acidosis that persists following adequate volume resuscitation is suspicious for necrotic bowel. The majority (70%) of infants with NEC have carbohydrate malabsorption. Carbohydrates are passed into the colon where they are fermented. The three methods of detecting malabsorption include testing stool for reducing substances with Clinitest tablets, testing urine for lactate, a by-product of fermentation, and testing breath for hydrogen excretion, which will be elevated in malabsorption.

D. Radiographic Findings. Plain abdominal radiographs, anteroposterior, and decubitus films (or cross table laterals) are the gold standard of diagnosis. Findings commonly include (1) bowel distension, (2) pneumatosis intestinalis, (3) portal venous gas, (4) ascites and/or gasless abdomen, (5) fixed bowel loop on serial films, and (6) pneumoperitoneum. If NEC is suspected, serial radiographs at 8-h intervals will help to make the diagnosis. Bowel distension is the first sign to appear followed by pneumatosis, which occurs early in the course of disease (within 12–24h) and may disappear. Portal venous air is usually associated with more severe disease. Ascites that progresses to a gasless abdomen is an ominous
sign of progressive sepsis. Pneumoperitoneum occurs when necrosis progresses to perforation and is an absolute indication for surgical intervention. Contrast studies may be used in the early phase of NEC if the diagnosis is in question. Lower gastrointestinal (GI) studies are avoided because of possible perforation. Ultrasound is useful during paracentesis to safely access intra-abdominal fluid.

E. Medical Treatment. The first course of treatment for NEC should always be nonoperative and consists of bowel rest, nasogastric decompression, broad-spectrum antibiotics (including anaerobic coverage), and vigilant surveillance. Metronidazole is favored for anaerobic coverage because of the virulence of *Clostridium* species as pathogens in NEC. Serial physical exams, radiographs, blood gases, and platelet counts will allow the assessment of progressive disease. When the diagnosis of NEC is suspected, surgical consultation should be obtained.

F. Surgical Treatment. The two indications for surgical intervention are (1) failure of medical management and (2) pneumoperitoneum. Failure of medical management is defined as persistent metabolic acidosis following adequate fluid resuscitation and/or the continued need for platelet transfusions to support thrombocytopenia. Persistent acidosis and thrombocytopenia suggest ongoing sepsis from necrotic bowel. Paracentesis can be used in this setting to evaluate the abdomen for contamination (gram-negative organisms or frank succus entericus). Pneumoperitoneum mandates drain placement or laparotomy depending on the size of the infant. In infants weighing less than 1,000 g, Penrose drains can be placed in the right and left pelvis and lavage of the abdomen can achieve effective drainage of the contaminated peritoneal space. In some cases, this will be the only surgical procedure needed. In 24 h, if the infant is no better or worse, formal laparotomy is performed to resect necrotic bowel. In infants weighing greater than 1,000 g, laparotomy is better tolerated and is the primary operation recommended. Resection of dead bowel and decontamination of the peritoneal space is the goal. *Proximal stoma* formation and distal mucous fistula or Hartmann pouch are created. Very rarely will a primary anastomosis be possible when minimal intraperitoneal contamination has occurred and the infant is not overwhelmingly septic. Postoperatively, the infant is supported on parenteral nutrition until sepsis clears and bowel function resumes. Six to eight weeks are allowed for inflammatory wound healing. A second procedure is then done to anastomose bowel and create GI continuity.

In 30% of cases medical management is curative; however, when NEC resolves, colonic strictures may occur (20–30% of cases) presenting as feeding intolerance typically at 4–6 weeks. Barium enema is useful to make the diagnosis. Surgical intervention is indicated in 30–40% requiring laparotomy and resection of bowel. In 10%, short-bowel syndrome or fat malabsorption is created by the extent of resection. Stomal complications including prolapse, retraction, or stricture occur in 20%. Enterocutaneous fistulae rarely occur. Long-term neurodevelopmental delay is present in cases of severe disease. Overall mortality is 30–40% with worse prognosis in the VLBW infants.
Necrotizing Enterocolitis

A. Pathophysiology
- Premature
- Low birth rate
- Perinatal events
- Immature gut barriers

B. Physical Findings
- Lethargy
- Abdominal distention
- Tenderness
- Abdominal wall color

C. Laboratory Findings
- Leukocytosis
- Thrombocytopenia
- acidosis

D. Radiographic Findings
- Bowel distension
- Pneumatosis
- Portal venous gas
- Pneumoperitoneum

E. Medical Treatment
- Bowel rest
- NG tube
- Antibiotics
- Surveillance
  - Platelet
  - Blood gas
  - WBC

F. Surgery
- Medical failures
  - Low platelet
  - Persistent
  - acidosis
- Infant >1000gm
  - Laparotomy
- Infant <1000gm
  - Bedside Penrose
  - Abdominal irrigation
Hematuria

David L. Sobel and Lev Elterman

Hematuria is the presence of red blood cells (RBCs) in the urine and may be the first sign of a serious underlying disease of the urinary tract. Hematuria may be classified as either gross or microscopic. Gross hematuria is alarming to the patient and deserves a prompt, thorough investigation to determine its cause. Microscopic hematuria, however, is often an incidental finding on routine examination. Nevertheless, microscopic hematuria also deserves an investigation as to its cause. Microscopic hematuria is defined as three RBCs per high power field or greater on microscopic evaluation of the urinary sediment from two of three specimens.

A. History and Physical Examination. An accurate history and physical examination are essential components of the diagnostic workup; collectively, they are extremely helpful in elucidating the cause of the hematuria. A proper history should focus on the duration and character of the hematuria, associated symptoms, age of patient, past medical history including history of diabetes and nephrolithiasis, history of urological procedures, smoking, radiation, carcinogen exposure, and trauma. Irritative voiding symptoms (i.e., urgency, frequency, and dysuria) are suggestive of an infection; in fact, hematuria in sexually active females is most often caused by cystitis secondary to a urinary tract infection. Hematuria accompanied with severe flank pain is suggestive of nephrolithiasis, whereas painless hematuria is concerning for a tumor or renal parenchymal disease. Lower urinary tract symptoms, such as weakened stream and hesitancy, connote an obstructive process that may be secondary to benign prostatic hyper trophy (BPH). The prostatic urethra in a patient with BPH is friable and has a tendency to bleed; moreover, patients with an obstructive process are more likely to develop cystitis.

When gross in nature, the character of the hematuria can be very helpful in isolating a source of bleeding. Gross hematuria can be classified as initial, total, or terminal, depending on when the hematuria appears during micturition. Initial hematuria is indicative of urethral bleeding distal to the external urinary sphincter. Total hematuria suggests bladder or kidney bleeding. Finally, terminal hematuria is indicative of urethral bleeding proximal to the external urinary sphincter (i.e., prostate or bladder neck).

A complete physical examination should be performed with special attention focused on the patient’s vital signs, abdominal exam, and genitourinary exam. Pyelonephritis may present with costo-vertebral angle (CVA) tenderness as well as an elevated temperature (>101.5°F). Nephrolithiasis associated with ureteral obstruction often presents with acute CVA tenderness which may radiate to the lower abdominal/genital region with or without nausea and vomiting. An enlarged prostate on digital rectal exam may indicate BPH, whereas an exquisitely tender prostate on exam suggests prostatitis.

B. Urinalysis. Urine dipstick and microscopic examination should be performed on all patients with hematuria. The urine dipstick assesses the presence of hemoglobin that is either within the urinary RBCs or free in the urine. A positive test is indicated by a specific color change on the dipstick; this oxidation reaction is catalyzed by hemoglobin. It is important to note that false positives may be the result of myoglobin as well as oxidizing contaminants such as Betadine. Urinary dipsticks have a sensitivity of 91% in detecting asymptomatic microscopic hematuria; their specificity is however limited and has been reported to be as low as 65%. As a result, each positive dipstick needs to be confirmed with a microscopic evaluation.

C. Cytology. Cytological examination of a voided specimen is useful in detecting bladder cancer (sensitivities range from 40 to 76%). As such, a voided urinary cytology is recommended for all patients with risk factors for transitional cell carcinoma (TCC), the most prominent form of cancer of the bladder. These risk factors include smoking, occupational exposure to chemicals or dyes, prior history of gross hematuria, age greater than 40, previous urological disease, history of irritative voiding symptoms, history of urinary tract infection, analgesic abuse (e.g., phenacetin), pelvic irradiation, and cyclophosphamide exposure.

D. Imaging. An imaging study is indicated in all patients with hematuria. Traditionally, the intravenous pyelogram (IVP) was the radiographic test of choice for visualizing the proximal
urinary tract. However, computed tomography (CT) has replaced the IVP as the initial radiological evaluation. CT is the single best imaging modality for the detection of urinary stones and the evaluation of renal or perineal infections. When IV contrast is administered during the CT scan, a KUB can be obtained at the end of the CT examination. This will allow the renal pelvi, ureters, and bladder to be examined in a fashion similar to an IVP. Patients with diminished renal function can undergo an MRI or a combination of renal ultrasound with retrograde pyelogram.

E. Cystoscopy. A careful visual inspection of the urethra and bladder with a cystoscope is a vital aspect of the workup for hematuria. In those instances where an individual presents with asymptomatic microscopic hematuria and does not have any of the risk factors for TCC (see Sect. C), a cystoscopy may not be necessary if the urinary cytology is negative.

Renal parenchymal disease can sometimes present as hematuria. When significant proteinuria, RBC cases, renal insufficiency, and/or a predominance of dimorphic RBCs are found in the urine, a nephrologist should be consulted.

Approximately 20% of the time, hematuria is documented but no identifiable cause of the hematuria can be found. Close, yearly follow-up of these idiopathic patients is indicated.
Hematuria

A. History and Physical exam
   irritative symptoms (urgency, frequency, dysuria)
   obstructive symptoms (weak stream, hesitancy)
   Pain present?
   timing of gross hematuria (initial, total, terminal)

B. Urinalysis
   dipstick microscopic

C. Cytology
   sensitivity 40-76%

D. Imaging studies
   indicated in all patients with hematuria
   CT scan – test of choice

E. Cystoscopy

Risk factors for transitional cancer:
   exposure to chemicals
   history of gross hematuria
   age greater than 40 years
   previous urologic disease
   irritative voiding symptoms
   history of urinary tract infections
   phenacetin abuse
   cyclophosphamide exposure
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Nephrolithiasis

Christopher L. Coogan

A. Renal calculi are usually asymptomatic until urinary obstruction occurs, causing flank pain, hematuria, infection, nausea, and vomiting. The pain is usually severe and may radiate to the flank, groin, testes, or tip of the penis depending on the level of obstruction. Stones occur three to four times more often in males, are more common in whites than blacks, and usually occur initially between the ages of 30 and 60 years (70%). In a patient who has passed one stone, the likelihood of passing a second stone is ~15% over 3 years and 50% by 15 years. The average interval between stone events is 9 years. The incidence of stones vary geographically and seasonally, with an increased incidence in the Southeastern USA and in the summer when dehydration is more common.

B. The etiology of nephrolithiasis is multifactorial, involving various physiological and chemical disturbances. Inherited disorders such as primary hyperoxaluria, cystinuria, and renal tubular acidosis may all cause stones, but occur less than 1% of the time. Primary hyperparathyroidism is the most common hypercalcemic condition associated with urolithiasis and is responsible for stone formation in 5% of the patients. A history of urinary tract infection, especially with urease producing organisms (most commonly Proteus sp.), predisposes to formation of struvite stones (magnesium ammonium phosphate), in which case urine pH is usually greater than 8.0. Infection stones account for ~15–20% of stones and are usually large stones within the renal pelvis (staghorn calculi). A history of benign prostatic hyperplasia with or without a neurogenic bladder may contribute to forming bladder stones secondary to high urine residuals and stasis of urine. Calcium stones occur more commonly in immobilized patients. Patients with an increased urinary oxalate (hyperoxaluria) experience an increased incidence of calcium oxalate stones. Hyperoxaluria is caused by enteric (secondary) hyperoxaluria, primary oxaluria, and rarely excessive dietary intake. Secondary hyperoxaluria is seen in patients with inflammatory bowel disease affecting the terminal ileum who undergo bowel resection. Most patients, however, do not have physical findings related to their stone disease and laboratory studies are required to determine the etiology.

C. A urinary pH of less than 5.5 eliminates distal renal tubular acidosis as a cause of stone formation; however, a low urinary pH is often found in patients with uric acid stones. High urine pH is found in patients infected with urease-producing bacterial organisms. Red blood cells and white blood cells are usually found in the urine of patients with urolithiasis. Certain crystal forms are diagnostic for the types of stones formed.

D. Laboratory evaluation in a first-time stone former consists of a serum calcium, creatinine, urea nitrogen, electrolyes, phosphorous, uric acid, ionized calcium, and parathyroid hormone level. In general, a CMP is usually ordered. A more detailed analysis is usually performed in patients who form their second stone, but may also be performed in selected first-time stone formers. An elevated serum calcium is suggestive of primary hyperparathyroidism but should be confirmed by a repeat calcium level and serum parathyroid hormone level. If an elevated parathyroid hormone level is found, patients should undergo parathyroidectomy prior to treatment of the stone.

E. Eighty percent of kidney stones are visualized on plain x-ray (radiopaque). A CT is necessary to demonstrate the degree of obstruction and document the location of the stone. CT should not be performed in patients with contrast allergy or with an elevated creatinine (> 1.8–2.0 mg/dl); an ultrasound or non-contrast CT scan may be performed in these patients.

F. The size and location of the stone determines the likelihood of its passing spontaneously. Approximately 90% of stones less than 4 mm will pass. Only 20% of stones greater than 6 mm will pass spontaneously. Stones are most likely to obstruct at three locations: at the ureteropelvic junction, at the level of the iliac vessels, and at the ureterovesical junction. Stones at the ureterovesical junction are more likely to pass than stones in the proximal ureter.

G. The majority of stones may be managed on an outpatient basis. Patients may be discharged from the emergency room if they are able to tolerate fluids, the pain is controlled with oral analgesics, and do not have temperatures greater than 101.5 °F. These patients, however, must have a repeat abdominal x-ray
in ~1 week and should strain their urine to retrieve any passed stones. They should drink plenty of fluids and be treated with antibiotics to cover gram-negative bacteria and oral pain medication.

H. Patients require hospitalization if they have high-grade ureteral obstruction, high temperatures (>101.5°F), uncontrollable pain, severe dehydration or vomiting, or a single functioning kidney. Cystoscopy and retrograde stent placement may be necessary. If a stent cannot be placed in a retrograde fashion, a percutaneous nephrostomy tube may be necessary. Placement of a stent will relieve the pain and obstruction and the stone may then be treated on an elective basis.

I. *Extracorporeal shock wave lithotripsy (ESWL)* is indicated for stones which are unlikely to pass spontaneously. ESWL was introduced in the early 1980s and is currently the most common treatment for renal calculi. Success rates for ESWL range between 60 and 90% depending on size, location, and type of stone. ESWL is performed on an outpatient basis. Mid and distal ureteral stones are removed by ureteroscopy and intracorporeal lithotripsy if necessary. The stones are removed by placing a ureteroscope into the ureter and using a basket to grasp the stone under direct vision.

J. Staghorn calculi are unlikely to be cured with ESWL. Large stones and cystine stones are best treated by placing a tube percutaneously into the kidney. A nephroscope is then placed through this channel into the kidney and the stone is broken up and removed. Patients are admitted to the hospital for 2 days after the procedure.

K. If a stone fragment is passed, patients are instructed to submit it for stone analysis. Approximately 80–85% of stones are composed of calcium oxalate, 10% are struvite, 5–10% are uric acid, and 1% is cystine. Patients who are recurrent stone formers should submit a 24-h urine collection. The volume of the urine, creatinine, and calcium, phosphorus, uric acid, and oxalate and citrate levels are analyzed.
Nephrolithiasis

A. Nephrolithiasis

B. History/physical exam

C. Urinalysis
D. Laboratory evaluation

E. CT

24-hour urine analysis

1. Recently passed stone
2. Normal
3. Consider non-urologic source

F. Urologic evaluation

G. Outpatient management

H. Hospitalization
- Cystoscopy +/- stent

I. ESWL vs. Ureteroscopy

J. Percutaneous nephrolithotomy

K. Stone analysis

Unlikely to pass

Passed stone

> 2.5 cm
A. Prevalence. Prostatism refers to symptoms of infravesical obstruction from benign prostatic hyperplasia (BPH). Symptoms may be classified into obstructive or irritative components and the prevalence varies by age as follows:

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Prevalence (in %)</th>
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<tr>
<td>41–50</td>
<td>25</td>
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<td>51–60</td>
<td>33–50</td>
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<td>61–70</td>
<td>41–69</td>
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<td>71–80</td>
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B. Symptoms. Obstructive symptoms include weak urinary stream, straining, hesitancy, incomplete bladder emptying, and terminal dribbling. Irritative symptoms consist of urgency, frequency, nocturia, urge incontinence, and dysuria. Patients may also suffer from chronic or acute urinary tract infections, hematuria, urinary retention, and acute or chronic renal failure. Symptom questionnaires (American Urologic Association Symptom Index for BPH) are available which ask seven questions, which are then ranked on a scale of 0–35. The symptom score is useful in determining which patients need treatment, which treatment modality is best utilized, and in quantifying response to treatment.

All patients should undergo a thorough physical examination with special attention to the genitourinary system. Percussion in the suprapubic region may reveal a distended bladder. A digital rectal examination (DRE) should be performed to assess the prostate; the normal gland is about the size of a chestnut and measures ~4 cm in width and 3 cm in length. The size of the prostate is estimated in the number of grams (normal prostate is ~20 g) or on a scale of normal through 4+. Size does not correlate with symptoms. Firmness is suspicious for cancer, which should be ruled out prior to treatment of prostatism.

C. Tests. Laboratory evaluation includes urinalysis and, in select patients, urine culture, serum creatinine, and serum prostate-specific antigen (PSA) should be obtained. PSA may be falsely elevated in infection, retention, BPH, and following urethral instrumentation. Optional testing includes measurement of urinary flow rate, post void residual (PVR), and pressure flow. Ultrasound or CT scanning of the abdomen and pelvis is indicated for patients with hematuria, urinary tract infection, renal insufficiency, and history of urolithiasis or prior urinary tract surgery. For patients with an elevated creatinine (>1.8 –2.0 mg/dl), an ultrasound should be performed rather than CT.

D. Treatment. Prostatism is not life threatening. Those with mild BPH (AUA score <7) should be followed with watchful waiting, while those with moderate BPH (AUA score 8–19) should be offered therapy and monitored closely for worsening symptoms or complications. Patients with an AUA score >19 should be counseled regarding the benefits of medical or surgical therapy but may also be treated with watchful waiting. Patients are instructed on behavioral therapy such as limiting fluid intake after the evening meal and avoiding decongestants.

Two main classes of medical therapy are available: (1) Alpha blockers (doxazosin, prazosin, and terazosin), which inhibit alpha-1-adrenergic-mediated contraction of prostate smooth muscle. Side effects include orthostatic hypotension, tiredness, dizziness, and nasal congestion. (2) Finasteride is a selective 5-alpha reductase inhibitor and is responsible for the conversion of testosterone to dihydrotestosterone, the active component of testosterone. It can reduce the size of the prostate and decrease the symptoms of BPH in some men. The side effects include decreased libido, ejaculatory dysfunction, and impotence. It is important to remember that finasteride will lower the PSA by 50%.

Absolute indications for surgery include urinary retention, recurrent urinary tract infection, gross hematuria, bladder stones, renal insufficiency, and failure of medical management to control symptoms. Prior to surgical intervention, cystourethroscopy should be performed for patients with hematuria, urethral stricture, and bladder cancer or prior lower urinary tract surgery (i.e., transurethral resection of the prostate; TURP). Cystourethroscopy may be performed in the office with a flexible cystoscope using local anesthesia.
prostatectomy is performed through either a suprapubic or a perineal approach. Indications include the presence of a large gland (75–100 g), coexisting problems requiring open bladder surgery (stone removal or diverticulectomy), or inability to place the patient in the lithotomy position. During open prostatectomy, the adenoma (BPH) is enucleated while the capsule and true prostate remain; thus these patients may still develop prostate cancer. TURP is performed if medical therapy fails, usually with a regional anesthetic. The interior of the prostate is resected through a cystoscope placed into the urethra. Complications include the TURP syndrome (2%) as a result of dilutional hyponatremia from absorption of irrigation fluid (usually 1.5% glycine). Patients may experience mental status changes, bradycardia, hypertension, visual changes, and nausea or vomiting. Additional complications include bladder neck contracture (2%), BPH recurrence (10% at 5 years), bleeding requiring transfusion (<5%), infection (15%), retrograde ejaculation (3%), and possibly impotence. Transurethral incision of the prostate (TUIP) may be performed in small glands with less morbidity.

Minimally invasive techniques may also be applied. These include (1) prostatic stents, (2) hyperthermia, (3) transurethral needle ablation (TUNA), (4) high-intensity-focused ultrasound (HIFU), and (5) laser treatment. Excellent short- and long-term results are seen with many of these procedures as an alternative to medical therapy or TURP.
Prostatism

A. Prevalence
   increases with age

B. Symptoms
   obstructive
   irritative

urinalysis
urine culture
serum creatinine
PSA

C. Tests

ultrasound
CT scanning
post-void residual

D. Treatment

AUA score < 7
AUA score 8-35

watchful waiting

medical therapy
   -alpha blockers
   -finasteride
   failed
   surgery*
   cystourethroscopy

*indications for surgery
   failure of medicine
   urinary retention
   recurrent UTI's
   hematuria
   stones
   renal insufficiency

open prostatectomy
large gland (75-100 g)
bladder surgery needed

transurethral resection

minimally invasive
techniques
A. Differential Diagnosis. The differential diagnosis of a painless testicular mass includes spermatocele, hydrocele, varicocele, inguinal hernia, and testicular tumor. Testicular torsion, epididymitis, trauma, and incarcerated inguinal hernia may also be confused with a testicular tumor, but these are usually associated with pain. A rapidly growing tumor or tumor that bleeds suddenly may cause testicular pain. Testicular tumors are the most common tumors in men aged 15–35 years; fortunately, however, they are currently one of the most curable solid neoplasms.

B. History and Physical Examination. A thorough history and physical examination should be performed on all patients. The usual presentation of a testicular tumor is a painless mass; some patients (30–40%) will complain of a dull ache or heaviness in the scrotum. Ten percent of patients will have signs or symptoms of metastasis. Gynecomastia is seen in 5% of patients as a result of hormone secretion by the tumor. Testicular tumors are associated with cryptorchidism (10%), maternal exposure to diethylstilbestrol (DES), trauma, atrophy, and familial predisposition. A history of urinary tract infections, fever, voiding symptoms, or urethral discharge is indicative of epididymitis.

Physical examination of the testes is performed by first examining the normal testicle with one or two hands. Any hard or fixed area is considered cancer until proven otherwise. One can usually differentiate between testicular tumor, epididymitis, spermatocele, varicocele, hydrocele, or testicular torsion by the history and physical examination alone. Varicocele is a dilation of the pampiniform venous plexus; it usually occurs on the left side and will often increase in size when one moves from the supine to a standing position or strains. A hydrocele is a fluid collection within the tunica vaginalis and is usually diagnosed by transillumination of the scrotum. A scrotal ultrasound may be needed to establish an accurate diagnosis, especially in patients with large hydroceles, which may make palpation of the testis difficult.

C. Laboratory Tests. Urinalysis may be helpful and may differentiate epididymitis from tumor. The most definitive studies are the tumor markers alpha fetoprotein (AFP) and the beta subunit of human chorionic gonadotropin (B-HCG). AFP is elevated in patients with embryonal cell carcinoma, teratocarcinoma, yolk sac tumors, or mixed tumors, but not in patients with choriocarcinoma or pure seminoma. AFP has a serum half-life of 5–7 days. B-HCG has a half-life of 24–36 h and is elevated in all patients with choriocarcinoma, 50% of patients with embryonal cell carcinoma, and 5–10% of patients with seminoma. The majority of seminomas do not exhibit any tumor marker elevation. In addition, 10–15% of nonseminomatous germ cell tumors will not exhibit tumor marker elevation.

D. Ultrasound Evaluation. A scrotal ultrasound is performed to examine masses of questionable etiology. Testicular tumors are visualized on ultrasound as hypoechoic lesions arising within the tunica albuginea. Epididymitis, torsion, hydrocele, spermatocele, and varicocele may also be diagnosed by ultrasound.

E. Orchiectomy. Removal of a testicular tumor is performed through an inguinal approach to permit early clamping and high removal of the spermatic cord. Orchiectomy should not be performed through a scrotal approach.

F. Cancer. If cancer is confirmed, staging studies for all patients should include a CT scan of the abdomen and pelvis and a chest x-ray. Testicular cancer spreads first to the retroperitoneal lymph nodes; this area must be closely examined during the CT scan. A CT scan of the chest is performed in select patients. Ninety-five percent of testicular tumors are of germ cell origin. Germ cell tumors of the testes are divided into two general classes: seminoma and nonseminomatous germ cell tumors.

Seminoma is the most common testicular tumor in adults and accounts for 40–60% of testicular tumors. Low-stage seminoma (no retroperitoneal adenopathy or low-volume retroperitoneal disease) is treated with radiation therapy to the retroperitoneal lymph nodes. Twenty to twenty-five percent of seminomas present with metastatic disease. High-stage seminoma is treated with cis-platinum-based combination chemotherapy.
Nonseminomatous germ cell tumors consist of embryonal cell carcinomas, teratomas, choriocarcinomas, and yolk sac tumors alone or in combination. Fifty to seventy percent of nonseminomatous germ cell tumors present with metastatic disease. Testicular tumors that contain both seminoma and nonseminomatous germ cell tumors are treated as nonseminomatous germ cell tumors. Clinical Stage I disease may be treated with either surveillance or retroperitoneal lymph node dissection. The incidence of positive nodes in patients without evidence of adenopathy on CT scan is 30%. Patients with high-volume retroperitoneal disease or lung metastasis are treated with cis-platinum-based combination chemotherapy.
Testicular Mass

A. Differential Diagnosis
   spermatocèle, hydrocele, varicocèle, inguinal hernia, tumors

B. History and Physical Exam

C. Laboratory Tests

D. Ultrasound of the Scrotum
   - cystic
     - careful follow-up
   - indeterminate
     - surgical exploration vs. follow-up ultrasound
   - solid
     - E. Radical Orchietomy
       - benign
         - stop
       - cancer stage with CT scan of abdomen and pelvis
         - non-seminoma
           - clinical stage I and IIA
             - surveillance vs. retroperitoneal node dissection
           - clinical stage IIA and III
             - cisplatin-based chemotherapy
         - seminoma
           - clinical stage I/II
             - radiation therapy to retroperitoneum
           - clinical stage II/III
             - cisplatin-based chemotherapy
Urinary Tract Infections

Kalyan C. Latchamsetty

A. Initial Management. A urinary tract infection (UTI) is defined as an inflammatory response of the urothelium to bacterial invasion that is usually associated with bacteriuria and pyuria. More than 7 million office visits are due to UTIs each year, and over 1 million hospital admissions are complicated by UTIs. These statistics reflect the broad prevalence of this diagnosis, which requires a different workup and treatment plan respective of the patient’s age, sex, and the overall clinical scenario.

UTIs should be categorized as complicated or uncomplicated. Uncomplicated UTIs are those that occur in a patient with a structurally and functionally normal genitourinary tract. Complicated UTIs occur in patients with a structural or functional abnormality that would reduce the efficacy of antibiotic therapy. A UTI in any male patient is considered a complicated UTI and requires further workup. Potential etiologies of treatable UTIs are vast and include chronically infected calculi, chronic prostatitis, ureteral duplication, ectopic ureters, vesicoureteral reflux (VUR), foreign bodies, urethral diverticula, papillary necrosis, and unilateral-infected atrophic kidneys.

A good history and physical exam (PE) is the first step in diagnosis and treatment. Adult patients may present with fevers, chills, nausea, vomiting, flank pain, dysuria, frequency, urgency, and other irritative voiding symptoms. Clinical symptoms in children may be similar but can also include poor feeding, irritability, and diarrhea. Older children may present with dysuria, suprapubic pain, and incontinence. In infants, the typical finding is failure to thrive. The PE should include careful palpation of the abdomen, flanks, suprapubic area, and a thorough genitourinary exam to evaluate for any abnormalities. Furthermore, a digital rectal examination should be performed on all adult male patients.

B. Urinalysis. A urinalysis with urine culture should be sent on all patients suspected of having a UTI. Suprapubic aspiration of urine is the most reliable means of obtaining a urine sample, but is invasive. A midstream-voided specimen is used instead. In the ambulatory setting, a dipstick examination revealing the presence of white blood cells (WBCs), bacteria, leukocyte esterase (from breakdown of WBCs in the urine), and nitrites (from gram-negative bacteria reducing nitrate to nitrite) in the urine all support a diagnosis of a UTI. Microscopic examination for red blood cells (RBCs) and WBCs is much more reliable than a dipstick examination, and the presence of >5 WBCs per high-power field is indicative of a UTI.

C. Pediatric Population. Treatment in children is based on age and may be subdivided into infants, young children, and school-age children. Those who are systemically ill require parenteral antibiotics until they defervesce and are to complete a full 2-week course of antibiotic therapy according to the cultures and sensitivities. Infants and young children who are asymptomatic should complete a 7-day course of antibiotic therapy, while school-age children who do not appear to be systemically ill may be treated with a 3–5-day course of antibiotics.

After treatment in the pediatric patient, a voiding cystourethrogram (VCUG) should be obtained to assess the presence of VUR or other anatomical abnormalities. If the VCUG is normal, a renal/bladder ultrasound (U/S) is obtained to further evaluate the genitourinary system. However, if the VCUG is positive for reflux, the urology service should be contacted for possible medical or surgical treatment. Pediatric patients found to have VUR, frequent reinfections, partial urinary tract obstruction, and those who are immunocompromised may need long-term antibiotic prophylaxis. It should be noted that imaging in children with UTIs is a controversial topic and some authorities advocate omitting imaging in female children with uncomplicated UTIs. Therefore, it is suggested that imaging be tailored to each individual clinical scenario in the pediatric population.

D. Adult Males. UTIs in young adults are 30 times more common in women than men, but with advancing age, there is a decrease in the ratio of women to men with bacteriuria. Twenty percent of women over the age of 65 have bacteriuria compared to 10% of men. The prevalence also increases with hospitalization or presence of other disease processes. An asymptomatic young male with a UTI has, by definition, a complicated UTI and warrants a U/S examination. If this is normal, no
further workup is necessary, and the patient should be treated with a 5–7-day course of antibiotic therapy. If symptomatic, however, the male patient needs further urologic evaluation including a cystoscopy and possible computed tomography (CT) scan.

E. Adult Females. Females are at higher risk for UTIs than males because of anatomical differences including a shorter and straight urethra and proximity of the introitus to the anus. There is great variability and controversy regarding treatment in females, but the following recommendations are generally accepted. Asymptomatic females need only a 3-day course of antibiotic therapy without further workup. If the female patient is symptomatic or is having recurrent or resistant UTIs, a full workup including imaging (U/S or CT scan) and cystoscopy should be obtained and treated accordingly.

Acute pyelonephritis is a diagnosis that may be categorized under UTIs; it may present with fevers, chills, and flank pain. Routine imaging is not necessary unless there is a clinical suspicion of renal/perirenal abscess or if the patient fails to respond to antibiotic therapy after 3 days. Patients may or may not require intravenous antibiotics and hospitalization depending on the acuteness of the illness. Acute uncomplicated pyelonephritis should be treated for 14 days while complicated cases are treated with 21 days of antibiotic therapy.
Urinary Tract Infections

A. Initial Management
   History & Physical Exam

B. Urinalysis
   >5 WBCs per high power field is diagnostic

C. Pediatric
   Systemically ill
     Yes
       Parenteral antibiotics then 2 week course
     No
       3-7 days antibiotics

   Voiding cystourethrogram ultrasound

D. Adult Males
   Asymptomatic
     Ultrasound 5-7 days antibiotics
   Symptomatic
     Cystoscopy Possible CT 5-7 days antibiotics

D. Adult Females
   Asymptomatic
     No work-up
   Symptomatic
     Imaging and cystoscopy
     Antibiotics for 3 days
Painful Scrotum

Christopher L. Coogan

A. The etiology of scrotal pain can usually be determined on the basis of history and physical examination. In particular, the duration of pain (acute vs. chronic) and age of the patient (child/adolescent vs. adult) are helpful. Laboratory tests, radiological exam, and occasionally scrotal exploration may also be necessary to confirm the diagnosis.

B. The history and physical exam are helpful in differentiating among torsion, orchitis, or epididymitis. Spermatic cord torsion may occur at any age but predominantly occurs in neonates and adolescents. Epididymitis and orchitis typically occur in adolescents and adults. The onset of pain is sudden in torsion, but is more gradual in patients with epididymitis or orchitis. A history of urinary tract infection, vomiting, dysuria, trauma, sexually transmitted disease, diabetes, previous swelling or pain, or recurrent infection should be determined.

C. Examination of a painful scrotum can be extremely difficult; one should begin by examining the unaffected side first. The consistency, texture (nodularity), and position (i.e., high riding) of the testicle as well as ability to transilluminate (hydrocele) are all helpful in determining the etiology. The absence of a cremasteric reflex (stroking of medial thigh resulting in elevation of the scrotum) is often seen in torsion and the testicle may be high riding. In contrast to epididymitis, manual elevation of a torsed testicle will not relieve the pain (negative Prehn’s sign). Careful examination may reveal a darkened area of the scrotal skin (blue dot sign) consistent with torsion of a testicular appendage. Rarely, testicular tumors will present with testicular pain, but must always remain in the differential diagnosis.

D. Laboratory evaluation should include a urinalysis in all patients and a complete blood count (CBC) in selected patients. Pyuria indicates a urinary tract infection and is also seen in some patients with epididymitis or orchitis. Profound leukocytosis in a patient with fever and severe testicular pain is an indication for hospitalization and intravenous antibiotics. The urinalysis is usually normal in torsion.

E. The two modalities utilized in the evaluation of scrotal pain include a radioisotope scan and duplex ultrasound. The former is performed by injecting technetium-99 followed by examining testicular blood flow. Torsion causes decreased blood flow producing a defect in isotope distribution. Duplex ultrasound of the scrotum determines the presence or absence of testicular blood flow, the presence of a mass, increased epididymal blood flow (epididymitis), and the presence of intestinal contents in the scrotum (hernia).

F. Epididymitis and orchitis can be seen in all age groups. It is often preceded by either a viral or a urinary tract infection with subsequent testicular or epididymal involvement. The pain is usually gradual in nature and in cases of epididymitis is localized to the posterior aspect of the scrotum. Patients may also have fever, dysuria, and leukocytosis; the testes and epididymis are firm, tender, and swollen. A duplex ultrasound or isotope scrotal scan will reveal increased blood flow to the affected testes. Patients are treated with intravenous (IV) or oral antibiotics, scrotal elevation, and bed rest.

G. Testicular torsion may occur at any age, but typically affects neonates (extravaginal torsion) or adolescents (intravaginal torsion). The onset of pain is usually sudden, and patients may complain of previous episodes. The pain will often radiate to the groin and lower abdomen and may be associated with nausea and vomiting. Physical examination will reveal a very high-riding, swollen, and exquisitely tender testicle. The cremasteric reflex is often absent and patients will not have relief of the pain with elevation of the testicle (Prehn’s sign). A duplex ultrasound or radioisotope scrotal scan will reveal diminished blood flow to the testicle. Patients with testicular torsion should undergo immediate scrotal exploration and orchiectomy of both testicles. The incidence of infarcted testicles increases significantly after 6h of spermatic cord torsion.

H. Patients with scrotal gangrene often present with pain and swelling of the external genitalia in association with signs of
systemic illness such as fever, chills, and generalized malaise. The onset of symptoms may be insidious especially in patients with a history of diabetes or ethanol abuse. Physical examination is diagnostic and usually reveals erythema of the scrotum and penis in addition to crepitis, necrosis, or purulent drainage. Treatment should consist of hemodynamic stabilization, broad spectrum IV antibiotics, and emergent surgical debridement of all necrotic and devitalized tissue.

I. Incarcerated inguinal hernias may also cause sudden scrotal pain. Physical examination is often diagnostic, but occasionally CT scan and/or ultrasound is necessary for definitive diagnosis. Depending on the clinical scenario, reduction may be attempted and, if this is successful, repair is generally undertaken during the same hospitalization. If reduction was unsuccessful or if viability of reduced intestine is in question, emergent surgery is indicated.

J. Hydroceles are defined as the presence of a persistent processus vaginalis. Occasionally a hydrocele will suddenly increase in size and result in testicular pain. The majority of hydroceles do not need treatment other than reassurance and careful examination. In selected patients, ultrasound of the scrotum is necessary to ensure there is no other pathology. Hydroceles may “communicate” with the peritoneal cavity through a hernia sac, or they may be of the “noncommunicating” variety.

K. Varicocele represents a dilation of the spermatic cord veins. The incidence of varicoceles is about 15% in the general population of adult males and may result in subfertility. There is a marked prevalence for the left side, which is due to the variance in the venous anatomy of the left gonadal vein. Examination of a varicocele should be performed by the patient in both the supine and standing position to allow dilation of the pampiniform plexuses. Varicoceles are rarely symptomatic and seldom require treatment except in the presence of subfertility or pain.
A. Painful Scrotum
B. History
C. Physical Exam
D. Laboratory Studies/UA
E. Radiological Evaluation
   - Radioisotope scan vs. Duplex US
F. Epididymitis / Orchitis
   - Antibiotics
   - Bed rest
   - Scrotal elevation
G. Torsion
   - Immediate operative intervention
   - Scrotal exploration
   - Bilateral orchiopexy (Torsion) or Incision and Drainage (Gangrene)
H. Gangrene
   - Conservative vs. operative Management
I. Hernia
J. Hydrocele
K. Varicocele
   - Treat in presence of subfertility
Part VIII
Gynecology
Acute Pelvic Pain

Eric R. Brown

A. Initial Evaluation. Acute pelvic pain is one of the most common reasons women present to the emergency room for evaluation. Patients should have a thorough evaluation to rule out a surgical emergency and to assess hemodynamic stability. Blood pressure and pulse should be monitored immediately. Unstable patients should be appropriately resuscitated following the ABCs of trauma protocols. Immediate vascular access should be obtained and prompt administration of isotonic fluids instituted to correct volume deficits. Once the patient is stabilized, continued evaluation of the physical exam should follow. If peritoneal signs are present (rigid abdomen, rebound, involuntary guarding), surgical evaluation should be considered.

B. Laboratory Testing. All patients of reproductive age and potential should undergo pregnancy testing with urine or serum human chorionic gonadotropin testing. In addition, blood counts, coagulation parameters, and type and cross-matching should be obtained. If pregnancy testing is positive, an ectopic pregnancy needs to be excluded. Additionally, patients who present with concomitant vaginal bleeding should also be evaluated for a threatened, incomplete, complete, or missed abortion. Ultrasonography can greatly assist in this evaluation.

C. Pelvic Imaging. Nonpregnant patients who present with acute pelvic pain should undergo imaging studies, preferably pelvic ultrasonography. For many of these patients, pain precludes a satisfactory physical examination; therefore, an accurate diagnosis must rely on imaging studies.

Patients with a normal pelvic ultrasound should have nongynecologic sources for their symptoms ruled out. The differential diagnosis includes gastrointestinal causes (acute appendicitis, diverticulitis, perforated peptic ulcer, cholecystitis, pancreatitis, inflammatory bowel disease, bowel obstruction, and strangulated hernias), genitourinary causes (nephrolithiasis, pyelonephritis, cystitis, and urethral syndrome), musculoskeletal causes, vascular causes (aneurysm and mesenteric thrombosis), and myofascial causes. Additionally, gynecologic causes that may be associated with normal ultrasounds include pelvic inflammatory diseases (salpingitis, oophoritis, or endometritis), adenomyosis, and dysmenorrhea.

For patients with abnormal pelvic ultrasound findings, the differential diagnosis depends on what is discovered. When pelvic fluid is found in the cul de sac, its character should be ascertained.

Clear fluid (hypoechoic on ultrasound) suggests a ruptured ovarian cyst. If there is no evidence of bleeding, these patients can be conservatively managed with analgesics and observation. Those who do not improve should undergo surgical evaluation. If the fluid is hyperechoic, hemoperitoneum should be considered.

The differential diagnosis includes a ruptured hemorrhagic cyst and ectopic pregnancy. Hemodynamic assessment and serial serum hemoglobin checks should be obtained. Should the patient become hemodynamically unstable, or if there is a significant drop in the hemoglobin or hematocrit, surgical evaluation should be considered after stabilization.

If a mass is discovered, an ovarian origin should be ruled out. Doppler studies can be done to rule out an ovarian torsion. If no flow is detected on Doppler studies, or if the patient’s symptoms are suspicious for ovarian torsion, immediate surgical evaluation should be undertaken. Doppler studies unfortunately have a high false-negative rate; therefore, clinical management should be guided by the patient’s symptoms. Patients with ovarian torsion tend to have pain that is colicky in nature. Traditionally a salpingo-oophorectomy was recommended as definitive management for ovarian torsion. Recently there have been numerous reports and studies that demonstrate conservative management (untwisting of the ovary) can be successful.

If the patient’s symptoms are not consistent with an ovarian torsion and Doppler studies are normal, a trial of analgesics should be considered. If there is no improvement in symptoms after a trial of analgesics, then surgical consultation is warranted.

Gynecologic masses that can cause acute pelvic pain include degenerating fibroids, tuboovarian abscess, and endometriomas. These should be managed accordingly.
Acute Pelvic Pain

A. Initial Evaluation
   assess stability
   begin resuscitation

surgery consult ← unstable patient

B. Laboratory Tests
   pregnancy test
   complete blood count
   type and cross

consider ectopic pregnancy
pregnant → consider threatened, missed, incomplete, or complete abortion

not pregnant

C. Pelvic Imaging
   ultrasound

normal → rule out non-gynecologic source
   -GI: appendicitis, ulcers, diverticulitis, cholecystitis, pancreatitis, IBD
   -GU: nephrolithiasis, cystitis
   -musculoskeletal
   -vascular

abnormal → -fluid present?
   determine character of fluid
   -mass present?
   rule out ovarian torsion
Evaluation of the Adnexal Mass

Eric R. Brown

A. Pelvic Mass. Between 5 and 15% of women undergo surgery for an adnexal mass. It is the fourth most common reason for hospital admission in the USA. An adnexal mass can be of gynecologic or nongynecologic origin. The former may originate from the cervix, uterus, or ovaries, while the latter may arise from any other pelvic structure. The key management goal is ruling out malignancy. Fortunately, the majority of these masses are benign and are discovered during a history and physical exam. The patient’s age, menstrual status, symptoms, history/physical exam, and results of imaging studies are important elements in the formation of a differential diagnosis. While some of these can be satisfactorily evaluated by a combination of abdominal and bimanual pelvic examination, the majority require adjunctive imaging studies for further characterization.

B. Ultrasonography is an important tool. Transvaginal or transrectal scanning offers an advantage over transabdominal route in assessing smaller adnexal masses. Ultrasound has a negative predictive value (80–100%) in determining malignancy. The addition of color Doppler velocimetry studies appears to offer no significant benefit. Computed tomography (CT) or magnetic resonance imaging (MRI) studies can be used in evaluating solid masses suspicious for malignancy; however, they are inferior to ultrasonography in the initial evaluation of cystic masses. The ultrasonographic features important in assessing whether a mass is benign or malignant include its size, echogenicity, wall structure, mobility, laterality, and the presence of septations, internal papillations, and/or solid components.

C. Pregnancy. All women of reproductive age should have a pregnancy test performed. An initial urine human chorionic gonadotropin (hCG) test is usually sufficient. A positive pregnancy test coupled with an adnexal mass and absent intrauterine gestational sac should raise suspicion for an ectopic pregnancy. These patients should be further evaluated and managed accordingly.

D. Cystic. A mobile, unilateral, unilocular, ≤3-cm cystic mass with a smooth thin wall and absent papillation or solid component is highly likely to be benign. Simple unilocular cysts can usually be managed conservatively. Most of these cysts are functional ones that spontaneously regress. Simple appearing ovarian cysts less than 6–8 cm can be observed for 6–8 weeks. A follow-up ultrasound during the follicular phase should be done to ensure resolution or improvement. The addition of hormonal therapy, such as oral contraceptives, does not appear to confer additional benefit. The risk of ovarian torsion increases as cyst size increases. Additionally, the risk of malignancy increases with cyst persistence, increased size, or if complex features are present. As such, for those cysts greater than 6–8 cm, or for those less than 6 cm that persist after observation, surgical evaluation should be considered.

E. Non-ovarian. Of the cystic adnexal masses, it is important to differentiate those of ovarian and non-ovarian origin. Non-ovarian cystic adnexal masses include hydro- or hematosalpinx, paratubal cysts, tubo-ovarian abscesses, and appendiceal abscess.

F. Solid Adnexal Masses. Solid adnexal masses can be of gynecologic or nongynecologic origin; the differential diagnosis of the latter includes pelvic kidney, lymphomas, retroperitoneal, gastrointestinal, or genitourinary neoplasms. These should be managed accordingly. Regarding the former, a solid adnexal mass in the pre- or early adolescent patient should raise suspicion for germ cell tumors, Mullerian abnormalities, or gonadal dysgenesis. Tumor markers (germ cell tumors), additional imaging (Mullerian abnormality), or chromosomal studies (gonadal dysgenesis-increased risk of malignant transformation with gonadoblastoma) can be done for further evaluation.

Gynecologic solid adnexal masses can be benign or malignant. The presence of ascites discovered on ultrasound should raise suspicion for malignancy. Meig’s syndrome, which presents with ascites, fibroma/thecoma, and pleural effusion, is a benign mimic. Benign solid or complex gynecologic adnexal masses include leiomyomas, fibromas, thecomas, Brenner tumors, endometriomas, hemorrhagic cysts, and benign cystic teratomas (dermoid cysts).
G. Malignancy Suspected. Solid or complex gynecologic adnexal masses suspicious for malignancy are further evaluated with tumor markers. Additional imaging can be done as well. Common tumor markers include CA-125 (epithelial ovarian cancer), alpha fetoprotein (germ cell tumors), beta hCG (choriocarcinoma), and lactate dehydrogenase (dysgerminomas). Tumor markers can also be used in concert with the evaluation of complex ovarian cysts. Solid gynecologic adnexal masses and complex ovarian cysts have a high risk of malignancy.

H. Surgery. Adnexal masses can be surgically managed via either laparoscopy or laparotomy. The choice is dependent upon the risk of malignancy, the patient’s history, and the surgeon’s skill level. Laparoscopy usually confers less patient morbidity when compared with laparotomy. Low-risk masses (persistent simple ovarian cysts) can be evaluated laparoscopically if no contraindication exists. Moderate- to high-risk masses are usually evaluated via laparotomy but are sometimes evaluated laparoscopically. Cytological washings should be obtained, as well as a frozen-section assessment of the mass. If a malignancy is discovered, appropriate surgical resection and staging should be performed.
Evaluation of the Adnexal Mass

A. Pelvic Mass
B. Ultrasound
C. Exclude Pregnancy
D. Cystic
E. Rule out non-ovarian source
F. Solid

G. Possible malignancy
H. Surgery

- Low risk for cancer
  - Laparoscopy
  - G. Possible malignancy

- High risk for cancer
  - Laparotomy
  - H. Surgery
  - Cytology, oophorectomy with frozen section

- Cancer
  - Resection staging (+)
  - Cytology, oophorectomy with frozen section

Simple unilocular
Observe follow-up ultrasound
Surgery if not resolved

Germ cell Tumors
Mullerian abnormalities
Gonadal dysgenesis

Tumor Markers
Imaging
Chromosomal studies

Adolescent patient

Adult
A. Pathogenesis. The diagnosis and management of pelvic inflammatory disease (PID) is based upon clinical signs and symptoms. PID encompasses infections of the uterus (endometritis or myometritis), fallopian tubes (salpingitis), ovaries (oophoritis), broad ligaments (parametritis), and pelvic peritoneum (peritonitis). PID affects up to one million women each year in the United States and is the number one reason for hospital admissions among women of reproductive age. PID is a polymicrobial disorder that results from ascending infection of bacterial flora from the vagina and cervix. The two initiating pathogens include *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. PID initiated by *N. gonorrhoeae* tends to be more acute and severe than that caused by *C. trachomatis*. Risk factors for PID include young age, history of multiple sexual partners, and a history of sexually transmitted diseases. Patients with PID commonly present with acute or subacute diffuse abdominopelvic pain and fever during or after their menses. Additional clinical criteria for PID include cervical motion or adnexal tenderness, and cervical or vaginal discharge.

B. Diagnosis. Laboratory evaluation that supports the clinical diagnosis of PID include an elevated white blood cell count, elevated C-reactive protein or sedimentation rate, or positive cervical cultures for *N. gonorrhoeae* and/or *C. trachomatis*. If the diagnosis of PID is confirmed, these patients should be offered testing for syphilis, hepatitis B and C, and human immunodeficiency virus (HIV). These tests are used to support a clinical diagnosis of PID based on the presence of cervical motion tenderness or discharge.

C. Differential Diagnosis. If laboratory and clinical diagnoses are not entirely consistent with a diagnosis of PID, an alternate diagnosis needs to be explored. The differential diagnosis includes appendicitis, diverticulitis, inflammatory bowel disease (IBD), nephrolithiasis, ectopic pregnancy, ovarian torsion, ruptured ovarian cyst, or degenerating fibroids.

D. Triage. Once the diagnosis is established, it is important to determine whether a patient needs to be admitted for intravenous antibiotic therapy. Generally patients who require hospitalization include pregnant patients, patients unable to tolerate oral intake, noncompliant patients, those with tubo-ovarian abscess, those who are immunodeficient, and patients in whom the diagnosis is uncertain.

E. In-patient care. Those admitted for intravenous antibiotic therapy can be started on one of the following regimens:

(a) Cefotetan 2 g IV q 12 h or cefoxitin 2 g IV q 6 h PLUS doxycycline 100 mg IV q 12 h.
(b) Clindamycin 900 mg IV q 8 h PLUS gentamicin IV/IM load of 2 mg/kg body weight then 1.5 mg/kg q 8 h (normal renal function).
(c) Ofloxacin 400 mg IV q 12 h PLUS metronidazole 500 mg IV q 8 h.
(d) Ampicillin/sulbactam 3 g IV q 6 h PLUS doxycycline 100 mg IV q 12 h.
(e) Ciprofloxacin 200 mg IV q 12 h PLUS doxycycline 100 mg IV q 12 h PLUS metronidazole 500 mg IV q 8 h.

*Only regimen (b) can be used in pregnant patients. All other regimens contain antibiotics that are contraindicated in pregnancy.*

Patients are continued on parenteral antibiotics until they have been afebrile for at least 24–48 h, have improvement in their symptoms, and are able to tolerate oral intake. If these criteria are met, patients can be discharged on one of the following regimen of oral antibiotic therapy:

(a) Ofloxacin 400 mg OR levofloxacin 500 mg daily PLUS metronidazole 500 mg twice a day for 14 days
(b) Doxycycline 100 mg twice a day for 14 days
(c) Cleocin 450 mg four times a day for 14 days

*Treatment of the sexual partner and use of barrier contraception should be encouraged.*

These patients should be reevaluated within a week. If they continue to show improvement, they should be encouraged to complete their entire antibiotic course. Upon completion they can be followed routinely.
F. Outpatient Care. Patients who met criteria for outpatient therapy upon initial evaluation can be treated with one of the following regimens:

(a) Ceftriaxone 2 g IM once OR cefoxitin 2 g with 1 g probenicid once OR third-generation cephalosporin THEN doxycycline 100 mg twice a day for 14 days.
(b) Ofloxacin 400 mg twice a day OR levofloxacin 500 mg daily PLUS metronidazole 500 mg twice a day for 14 days.

_Treatment of the sexual partner and use of barrier contraception should be encouraged._ For patients who do not improve, imaging studies should be done to rule out an abscess.

G. Imaging Studies: If no abscess is identified on imaging studies, other diagnoses, including those mentioned in Sect. C, should be considered. These patients should undergo surgical evaluation via laparoscopy or laparotomy. Surgical assessment is the gold standard for diagnosing PID; it facilitates direct culture of the pelvis. Furthermore, it facilitates evaluation of other causes for the patient’s symptoms. Endometrial biopsy can also be done as an adjunct to confirm the disease.

H. Abscess. When imaging studies reveal the presence of an abscess, one of the following management protocols can be pursued:

(a) Laparoscopy or laparotomy with removal of the abscess. These patients should be counseled preoperatively about the possibility of a hysterectomy and/or possible removal of the fallopian tubes and ovaries.
(b) Image-guided drainage of the abscess with continued antibiotic therapy, which should include adequate anaerobic coverage.
(c) Broaden spectrum of parenteral antibiotic treatment to ensure adequate anaerobic coverage. If there is no significant improvement in 24–48 h, these patients should undergo surgical assessment.

Once the diagnosis of PID is made, patients should be counseled about the following sequelae of the disease:

(a) Women with PID have an increased infertility rate. The degree of infertility appears to be directly proportional to the number of episodes of PID. Women with three or more episodes have infertility rates up to 40%.
(b) PID increases a woman’s risk of an ectopic pregnancy. These patients should be encouraged to undergo early pregnancy evaluation to confirm intrauterine gestation.
(c) Patients with PID appear to have an increased incidence of chronic pelvic pain. The incidence of this appears to be directly proportional to the number of episodes and the severity of the disease.
Pelvic Inflammatory Diseases

A. Pathogenesis
   ascending vaginal or cervical infection
   *Neisseria Gonorrhea, Chlamydia Trachomatis*
   Risk factors: multiple partners, history STD's

B. Diagnosis
   cervical motion tenderness
   vaginal discharge, positive cultures
   C-reactive protein, ESR

C. Differential Diagnosis
   appendicitis, diverticulitis, ectopic pregnancy, ovarian cyst or torsion, IBD, kidney stones

D. Triage

E. In-patient Care
   IV antibiotics (see text)
   monitor progress, fever
   - improves
   - no improvement
     if patient clinically improves, prepare for discharge on oral antibiotics (see text), treat sex partner, educate patient about risk of ectopic pregnancy and infertility

F. Outpatient Care
   oral antibiotics (see text)
   treat partner
   barrier contraception

G. Imaging Studies

H. Abscess
Part IX
Thoracic Problems
A. Definition. Solitary pulmonary nodules (SPNs) are defined as solitary lesions surrounded by normal lung parenchyma, not associated with atelectasis or hilar enlargement. In the past, lesions as large as 6 cm were classified as SPN, but now an appreciation that lesions $\geq 3$ cm are almost always malignant has led to the belief that a 3 cm cutoff may be more useful. Lesions $>3$ cm are sometimes referred to as solitary mass lesions. They are most often found on routine chest x-ray (CXR; incidence 0.1–2%; 130,000 new SPN/year) and are usually asymptomatic.

B. Differential Diagnosis. The differential diagnosis of SPN includes both benign and malignant processes. Granulomas make up 90% of benign nodules and most commonly indicate a coccidiodoma, histoplasmoma, or tuberculoma, incidences varying by geographical location. Hamartomas are the second most common.

Malignant lesions are most often primary bronchogenic carcinomas (88%), all cell types may present as an SPN. Peripheral nodules are most commonly adenocarcinomas or large cell carcinomas. Centrally located nodules are usually squamous or small cell carcinomas. Metastatic cancer (head and neck, colorectal, breast, kidney, and sarcoma) represents 10% and carcinoid 2% of malignant SPNs.

C. Evaluation. If the SPN is greater than 3 cm, it should be considered malignant until proven otherwise. If it is less than 3 cm and old CXRs show that it is stable in size, a follow-up CXR in 1 year is indicated. However, if this lesion is increasing in size then it should be considered malignant. If a lesion is less than 3 cm in size and old CXRs are not available for comparison, it is handled according to the risk factors set forth by history and radiographic features.

Low-risk characteristics include age <35, no smoking history, doubling time of the lesion of $<30$ days or $>400$ days, no history of prior malignancy, and a benign pattern of calcification (smooth and well-demarcated edges). High-risk characteristics include age $>35$, smoking history, doubling time of 30–400 days, history of a prior malignancy, and a malignant radiographic appearance (spiculated calcifications and lobulated appearance).

Patients at low risk should undergo follow-up CXR in 1 year, and those at high risk should be treated as if their lesion was malignant. Those patients with an intermediate risk should undergo CT scan of the chest. Management of these patients will depend on the CT characteristics of the lesion. CT scan features of benign lesions include smooth, well-demarcated edges, the presence of calcifications, or the presence of fat (hamartoma). Malignant lesions may be lobulated or spiculated, or have blurred edges or bubblelike areas of decreased density. Lesions with benign characteristics are followed closely; those with ominous features are treated as though they were cancer. Lesions which remain indeterminate are evaluated further.

D. Preparation for Surgery. Patients whose nodules are still indeterminate or likely malignant should have prompt intervention. Thoracotomy for diagnosis and treatment is indicated unless the patient is deemed to be unfit for operation (severe CAD, COPD). Preoperative workup includes pulmonary function testing (PFTs) and CT scan of the chest and abdomen to assess for other pulmonary lesions, for hilar adenopathy and for distant metastases. Patients at higher risk for thoracotomy are the elderly, the smokers, and the obese. In addition, patients with a preoperative FVC $<30\%$ of predicted or FEV1 of $<1$ L are at especially high risk. Patients scheduled for lobectomy or pneumonectomy should have a predicted postoperative FEV1 of at least 800 ml to be considered surgical candidates. The addition of a perfusion scan is very helpful in patients with borderline studies. Patients should stop smoking before surgery (even 5–7 days shows substantial benefit), and bronchodilators and respiratory muscle conditioning should be instituted.

Positron Emission Tomography (PET) has become a very important tool in the evaluation of lung nodules. A radioisotope, fluorodeoxyglucose (FDG), is injected into the patient’s bloodstream and is taken up by cells with a high metabolic rate (i.e., tumor cells). There the FDG is trapped.
because it cannot participate in glycolysis, and it begins to decay by releasing positrons. These are detected by the scanner and show up as “hot spots.” Tumors with a low metabolic rate, such as bronchoalveolar carcinomas and carcinoids, can produce falsely negative scans. Active infectious or granulomatous processes can lead to false-positive scans. Overall, however, the sensitivity and specificity of PET scanning is >90% for lesions >7 mm in size. A PET scan should be considered for all indeterminate lesions before proceeding with more invasive tests and for all lesions that are likely malignant for proper staging and to rule out metastatic disease.

Transthoracic needle aspiration (TTNA), under either fluoroscopic or CT guidance, can occasionally aid in the decision-making process. The presence of benign or indeterminate histology does not exclude malignancy, and this severely limits the usefulness of TTNA. There is a subset of patients with SPN in whom TTNA is warranted: patients who are not operative candidates yet require a definitive diagnosis, patients with prior malignancies and a new SPN (differentiate between a new primary lung cancer and a solitary met), and patients whose lesion is suspicious for small cell cancer (SCLC). Bronchoscopy also has limitations in diagnosing malignancy, especially lesions located in the periphery. Indications for bronchoscopy with biopsy and sputum sampling parallel those for TTNA. In general, a patient who can tolerate an operation should have surgical assessment of suspicious SPN.

E. Surgery. A posterolateral thoracotomy in the fifth or sixth intercostal space is most often employed. An anterolateral thoracotomy in the third intercostal space can be used in patients with low pulmonary reserve. Frozen section of the lesion should first be done if the diagnosis is still in question. Next, resectability must be determined. Pleural metastases, extensive mediastinal lymph node involvement, and direct extension of the tumor render the lesion unresectable. Approximately 80–100% of SPN are resectable at the time of thoracotomy. The goal of a curative resection is to remove the tumor with adequate margins and to preserve the maximum amount of normal tissue. Lobectomy is the procedure of choice for malignant SLNs. Alternatively, bilobectomy or pneumonectomy can be considered depending on the size and location of the tumor. Segmentectomy and wedge resection should be reserved for the patient with small lesions and high operative risk. Operative mortality is 3–7% for malignant lesions (pneumonectomy 6.2%, lobectomy 2.9%, wedge resection 1.4%) and 1% for benign lesions.

Survival from lung cancer depends on the stage and cell type of the lesion. In non-small cell lung cancer (NSCLC), the estimated 5-year survival is as follows: Stage IA—70–80%, Stage IB—60–70%, Stage IIA—50–60%, Stage IIB—40%, Stage IIIA—20–30%, Stage IIIB—5%, and Stage IV—0–5%. This underscores the importance of prompt evaluation and treatment of patients with SPN. SCLC behaves as a systemic disease and is usually metastatic by the time it is diagnosed. For this reason, surgery is not indicated and multimodality treatment aimed at reducing symptoms is employed. Two-year survival with SCLC is 10%. The exception to this rule is the rare occasion when SCLC is picked up as an SPN and subsequent test confirm that there is no evidence of other disease. In this case, it should be treated similarly to NSCLC, and lobectomy yields similar results based on postoperative stage.

Video-assisted thoracoscopy (VAT) is a technique that has greatly assisted the surgeon in evaluating indeterminate SPNs. A wedge resection of the nodule can be done with minimal morbidity and mortality. VAT cannot assess centrally located lesions or those near the hilum; in these cases, thoracotomy must be done.
Solitary Pulmonary Nodules

A. Definition
solitary lesion surrounded by normal parenchyma

B. Differential Diagnosis
benign: coccidioidoma, histoplasmosoma, tuberculoma
malignant: primary lung tumor, metastasis

C. Evaluation
obtain old CXR

> 3 cm
consider malignant

D. Prepare for Surgery
CT chest, abdomen
PFT’s
PET scan
needle aspiration needed?
bronchoscopy

E. Surgery
thoracotomy vs.
VATS

< 3 cm

size stable by old CXR
follow-up CXR in 1 year

increased size compared to old CXR
consider malignant, prepare for surgery

no old CXR’s available

low risk for cancer

intermediate risk

high risk for cancer

CXR in 1 year

CT scan, if indeterminant or malignant, prepare for surgery

consider malignant, prepare for surgery
A pleural effusion develops when the volume of fluid entering the pleural space exceeds the amount that can be removed via the lymphatics. Effusions result from either increased pleural fluid formation or decreased lymphatic clearance, or a combination of these two. The most common causes are congestive heart failure (CHF), pneumonia, cancer, and pulmonary embolism.

Pleural fluid originates from three major sources: the pleural capillaries, the pulmonary interstitial spaces, and the peritoneal cavity. The normal rate of entry of fluid into the pleural space approximates 0.01 cc/kg/h, while the average rate of lymphatic clearance is around 0.30 cc/kg/h. Movement of fluid across pleural membranes is determined by hydrostatic and oncotic pressure differences across these membranes (Starling’s law). Fluid may accumulate in the presence of increased capillary hydrostatic pressure (CHF), decreased plasma oncotic pressure (hypoalbuminemia), increased capillary permeability (pneumonia), increased intrapleural negative pressure (atelectasis), or decreased lymphatic clearance from obstruction (tumor).

A. Initial Evaluation. Symptoms of pleural effusions include dyspnea, pleuritic chest pain, and cough. Physical examination may reveal dullness to percussion, decreased or absent breath sounds, and absence of fremitus.

Plain chest x-ray (CXR), including a lateral decubitus view, is the easiest method of confirming the diagnosis. Ultrasound and CT scan are also useful imaging modalities. If the distance between the chest wall and the lung is greater than 10 mm (by lateral decubitus CXR, ultrasound, or CT scan), a diagnostic thoracentesis is indicated. Loculated fluid collections are suggested by a lack of fluid layering on imaging studies. CT scan can provide additional information about the pleura, lung parenchyma, and mediastinum.

B. Thoracentesis. Thoracentesis should be performed for all symptomatic or undiagnosed pleural effusions. The fluid may be bloody (cancer, pulmonary embolus, trauma, and pneumonia), turbid (chylothorax), clear or purulent (empyema). The pleural fluid should be analyzed for protein, albumin, lactate dehydrogenase (LDH), glucose, cell count, culture, AFB, and cytology. Serum protein, albumin, and LDH should be drawn simultaneously. Differentiating transudative versus exudative effusions at this point will determine the most effective treatment strategy (Table 78.1).

C. Transudates. Transudative effusions are due to systemic changes such as increased hydrostatic pressure or decreased colloid oncotic pressure (CHF, cirrhosis, and pulmonary embolism). The pleura itself is free of disease. Treatment is directed toward the underlying cause. Repeat thoracentesis may be employed for recurrent, symptomatic transudative effusions, while aggressive systemic treatment addresses the underlying etiology. Failure of repeated thoracentesis is treated by either chest tube suction or VATS (video-assisted thoracoscopic surgery) to obliterate the pleural space, followed by chemical and/or mechanical pleurodesis. Continued aggressive medical therapy is mandatory.

D. Exudates. Exudative effusions result from pleural or lymphatic disease processes. Common examples include pneumonia, malignancy, pulmonary embolism, and empyema. Tuberculosis is a less common, but important, example of an exudative effusion. Clinical history and physical exam, sputum culture, PPD, and spiral CT scan may be very helpful in providing diagnostic clues. Note that pulmonary embolism can cause either transudative or exudative effusions and should be ruled out whenever the cause of any effusion eludes initial diagnostic studies. The treatment of exudative effusions varies greatly according to the diagnosis.

E. Malignant Effusion. Malignant effusions signal advanced disease and poor prognosis. Lung and breast cancers are the most common causes; ovarian and gastric cancers are less common causes. Patients with breast cancer may survive for several months to years from the onset of a malignant effusion, whereas patients with lung, gastric, and ovarian cancer usually survive only a few months. Interestingly, an effusate pH < 7.30 or effusate glucose < 60 mg/dl will reliably predict a survival of only a few months versus ~1-year survival for...
Video-Assisted Thoracoscopic Surgery (VATS) may be utilized for exudative effusions in patients who may eventually undergo thoracotomy for any reason, such as very dense adhesions, and should not be used in patients with malignant effusions. VATS is highly effective for patients with malignant effusions (not possible with a bedside chest tube) and consists of gently rubbing the pleural surfaces with an abrasive material to help produce an inflammatory response (a cautery scratch pad is an example). Chemical pleurodesis should also be utilized to prevent recurrence of the effusion. Talc poudrage or slurry can be very evenly distributed with the aid of direct visualization, providing an extremely effective pleurodesis that only rarely has to be repeated postoperatively for recurrent fluid accumulation. A chest tube is inserted after the procedure and is required for only a day or two. In contrast, bedside chest tubes may require several days while the separate processes of drainage and chemical pleurodesis take place. Thus, VATS helps reduce length of hospital stay for these terminally ill patients.

The final advantage of VATS over traditional chest tube placement is pain control. The entire procedure is performed under general anesthesia and requires only one or two very small incisions. The chest tube can be placed directly through one of these incisions. Additionally, intercostal nerve blockade provides very effective postoperative analgesia. This is in contrast to bedside chest tube placement and pleurodesis, both of which are inherently painful processes. Proper chest tube placement can be performed almost painlessly, but pleurodesis nearly always is uncomfortable for the patient no matter how well performed. Bedside chemical pleurodesis is often unevenly distributed, resulting in higher rates of recurrent effusions, which require additional treatment (repeat pleurodesis or chronic tube drainage).

The disadvantages of VATS are the need for general anesthesia and a skilled thoracic surgeon. Not all patients will be able to tolerate general anesthesia or the single-lung ventilation that is employed during the operation. Therefore, classic tube thoracostomy followed by observation until fluid drainage is minimal, followed by pleurodesis at the bedside, is still the most commonly practiced method of treatment for malignant effusions.

Failure of the above methods to prevent fluid reaccumulation and dyspnea usually requires the use of a chronic chest tube. Small tubes or pigtail catheters should be utilized if at all possible, and many can be attached to drainage bags or bulbs to allow discharge of the patient from the hospital. Unfortunately, these are difficult effusions to treat satisfactorily.

### Table 78.1. Exudate effusions (must meet one criterion).

<table>
<thead>
<tr>
<th>Criteria</th>
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<tbody>
<tr>
<td>Protein ratio (effusion:serum) &gt; 0.5</td>
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<tr>
<td>LDH ratio (effusion:serum) &gt; 0.6</td>
</tr>
<tr>
<td>Effusion LDH &gt; 2/3 normal serum LDH</td>
</tr>
<tr>
<td>Serum-effusion difference ≤ 1.2 g/dl</td>
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<tr>
<td>Malignant cytology</td>
</tr>
</tbody>
</table>

### Table 78.2. Chemical pleurodesis procedure.

1. Complete fluid drainage
2. IV narcotic/sedative
3. 1% lidocaine (4 mg/kg) instillation
4. Doxycycline (500 mg/50 cc) or talc slurry
   - 2 gm/100 cc
5. Clamp tube, rotate patient
6. Reapply tube suction
7. Remove tube when output < 100 cc/day
Pleural Effusions

A. **Initial Evaluation**
   - Symptoms: dyspnea, pleuritic chest pain, cough
   - Physical exam: decreased breath sounds
   - Imaging: chest X-ray, CT or ultrasound

B. **Thoracentesis**
   - Transudate vs. exudate

C. **Exudate**
   - Check protein, albumin, LDH, glucose, cell count, culture, cytology, AFB

D. **Exudate**
   - e.g. pneumonia, cancer, empyema, PE

E. **Cancer**
   - Tissue diagnosis then systemic chemotherapy, possible radiation

F. **Tube thoracostomy**
   - Non-loculated chemical pleurodesis
   - Failed

G. **VATS**
Acute Respiratory Distress Syndrome

Edie Y. Chan

A. Definition
Acute respiratory distress syndrome (ARDS) was first described in 1967 by Ashbaugh and colleagues and was initially associated with trauma, sepsis, or aspirations. In 1994, the American-European Consensus Conference on ARDS created the widely used definition of ARDS as follows: (1) acute onset of severe hypoxemia with a PaO₂ to FIO₂ ratio of 200 mmHg or less, (2) bilateral infiltrates on chest x-ray, and (3) no evidence of left atrial hypertension or a pulmonary wedge pressure less than 18 mmHg. A less-severe form of ARDS known as ALI (acute lung injury) was also defined at this conference and was distinguished from ARDS by a PaO₂ to FIO₂ ratio of less than 300 mmHg.

Incidence and Risk Factors. The estimated incidence of this disease is 1.5–12.9 cases per 100,000 people. Risk factors associated with the development of the disease include pneumonia, sepsis, gram-negative infections, aspiration, trauma, pancreatitis, blood transfusions, smoke inhalation, and drug toxicity. The presence of multiple risk factors increases the risk of the development of the disease. The risk of mortality in a patient who develops ARDS is greater than 30% and the most common cause of death from ARDS is multiple organ failure.

B. Clinical Features of ARDS
ARDS is commonly associated with three phases. The first phase, the acute or exudative phase, is characterized by damage to the alveolar-capillary barrier, which leads to flooding of the alveolar spaces with fluid, inactivating surfactant, leading to widespread inflammation and producing gas exchange abnormalities and loss of lung compliance. Clinically, this is characterized by the development of bilateral infiltrates on chest x-ray. Pathologically, this is characterized by diffuse heterogeneous areas of alveolar and capillary damage, and exposed alveolar-epithelial basement membrane. The alveolar spaces are filled with edema fluid and inflammatory cells. Following this is the fibrosis phase, which is associated with persistent hypoxemia, pulmonary hypertension, and further loss of compliance. Chest x-ray may show linear opacities associated with evolving fibrosis. Pathologically, the lungs demonstrate deposition of collagen, acute and chronic inflammation, and persistent edema. Finally, the patient may progress to the recovery phase, which is associated with resolution of radiographic abnormalities and improvement in oxygenation and lung compliance. However, microscopic fibrosis persists in the lung spaces. It is important to note that ARDS does not affect all areas of the lung homogeneously and there may be areas of unaffected alveolar spaces.

C. Ventilator Treatment Strategies in ARDS

Lung Protective Ventilation (LPV). Five randomized controlled trials have been published on LPV. Three of the trials found no significant difference in mortality. One study by Amato et al. used both pressure- and volume-limited ventilation in association with higher positive end-expiratory pressures (PEEP). This study was stopped early secondary to a significant reduction in 28-day mortality. In 2000, a large multicenter randomized controlled clinical trial sponsored by the National Heart, Lung, and Blood Institute enrolling 861 patients found that low tidal volumes (6 ml/kg) with a plateau pressure limit of 30 cm of H₂O versus traditional tidal volumes (12 ml/kg) with a plateau pressure limit of 50 cm of H₂O decreased the mortality from 40 to 31%; p = 0.007. This became the basis for the use of LPV.

Permissive hypercapnia LPV may lead to an elevation of arterial carbon dioxide levels. Currently, there is no evidence that demonstrates that controlled elevations of arterial carbon dioxide are harmful to human beings.

High PEEP. A trial evaluating the use of higher PEEP in the setting of LPV demonstrated no difference in mortality compared to the control group and only a modest effect on oxygenation.

High-Frequency Oscillatory Ventilation (HFOV). Trials comparing HFOV versus conventional ventilation demonstrated no difference in adverse events and no difference in 30-day mortality or survival. However, patients with the most severe hypoxemia appeared to have a trend towards a benefit with HFOV.

Prone Positioning. The use of prone positioning is believed to have potential beneficial effects on respiratory mechanics, recruitment of underutilized alveoli, and increasing secretion.
drainage. Trials evaluating prone positioning demonstrated no difference in mortality but modest improvements in oxygenation. 

_Nitric Oxide (NO)._ Inhaled NO leads to pulmonary vasodilation, which may improve ventilation-perfusion mismatches, hypoxemia, and pulmonary hypertension. Multiple large trials investigating the use of inhaled NO have found that inhaled NO led to improvements in oxygenation but no survival benefit.

**Pharmacologic Strategies in ARDS**

_Prostaglandin E._ A meta-analysis demonstrated no effect of prostaglandin E on early mortality. Secondary to significant adverse events, most trials were stopped early, which led to a lack of data on late mortality and other secondary outcomes (length of ventilation).

_N-acetylcysteine._ Meta-analysis showed no difference in early mortality. There were few adverse events from these studies; however, there were not enough data to report on late mortality or secondary outcomes.

**High-Dose Corticosteroids.** There were two randomized studies evaluating high-dose corticosteroids in patients with ARDS. There was no significant difference in early mortality with the use of steroids.

_Surfactant._ Several studies randomized patients to surfactant therapy. Again, there was no difference in early mortality in ARDS. There were not enough data to evaluate late mortality or secondary outcomes.

_Corticosteroids in Late ARDS._ Meduri et al. randomized 24 patients with late-phase ARDS to steroids in an attempt to attenuate the fibrotic response. The study demonstrated a significant improvement in mortality, but this study needs to be reviewed with caution. This was an extremely small trial with several operator biases. However, this has led to the Late Steroid Rescue Study, which randomizes 180 patients to steroids with late-phase ARDS. However, the results of this study have not yet been published.

_Pentoxifylline._ Pentoxifylline is a phosphodiesterase inhibitor that prevents neutrophil chemotaxis and activation. This drug was evaluated in 30 patients with metastatic cancer and ARDS. There was a significant reduction in 1-month mortality in the treated group. However, this study also needs to be approached with caution secondary to small sample size and lack of definition of ARDS in this study.

**Conclusions.** ARDS is a disease entity with a significant morbidity and mortality. Identifying patients early in the disease process may potentially allow for changes in managing mechanical ventilation and reviewing therapies for best outcomes in patients.
A. Definition
1. PaO₂/FiO₂ < 200 mmHG
2. Bilateral infiltrates on chest X-ray
3. Pulmonary wedge pressure < 18 mmHg
   No evidence of left atrial hypertension

B. ARDS
1.) 3 phases

C. Ventilator Strategies
LPV (6ml/kg)
Plateau Pressure ~ 30cm of H₂O
Increased Plateau Pressure
1.) decrease tidal volume
2.) increase permissive
Permissive hypercapnia

Life threatening
Hypoxemia
1.) HFOV
2.) Increase PEEP
3.) Prone Ventilation
4.) Inhaled NO

Pneumonia
1.) Antibiotics

CHF
Cardiac Intervention
Nosocomial Pneumonia

Jennifer E. Foster

A. General Considerations. Nosocomial pneumonia, or hospital acquired pneumonia, occurs 48 h or more after admission. It is estimated that its incidence is ~4–8 episodes per 1,000 hospitalizations. With a mortality rate between 33 and 50%, nosocomial pneumonias have the highest mortality rate of all types of nosocomial infections. Additionally, the development of a nosocomial pneumonia increases the cost of hospitalization by $40,000 per patient. Several predisposing endogenous host factors and exogenous conditions have been identified. Host risk factors include age >70, chronic lung disease, depressed consciousness, immune deficiency, organ failure, smoking, and low serum albumin. Exogenous risk factors include mechanical ventilation, chest surgery, the presence of an intracranial pressure monitor or nasogastric tube, transport from the ICU for procedures, reintubation, and hospitalization during the fall or winter. Medications such as H-2 blockers or antacid therapy, previous antibiotic exposure, steroids, and immunosuppressive drugs may also predispose patients to nosocomial pneumonia. While most cases occur in nonventilated patients, the rate of infection is increased 20-fold in ventilated patients.

The most important step in the pathogenesis of nosocomial pneumonia is colonization of the oropharynx and stomach by a pathogenic bacteria, virus, or fungus. Although healthy human patients are able to aspirate small amounts of pathogens without sequelae, this can be fatal for hospitalized patients. If the aspirated inoculum is small, mucociliar clearance and alveolar macrophages will be able to remove the pathogen. A larger inoculum aspirated by a patient with a deficient host defense will allow pathogen proliferation and pneumonia will develop. The most common organisms associated with nosocomial pneumonia are aerobic gram-negative bacteria especially Pseudomonas aeruginosa, Enterobacter, Klebsiella pneumoniae, Escherichia coli, and Acinetobacter. Other common organisms include gram-positive cocci such as Staphylococcus aureus and Streptococcus pneumoniae. Viral and fungal pathogens are uncommon in immunocompetent patients, but should be considered in immunosuppressed patients.

B. Diagnosis. The clinical findings diagnostic of pneumonia are fever, purulent sputum, and an elevated white blood cell count in combination with a new infiltrate on chest radiograph. Because of the sequelae of delayed treatment, clinical findings alone are sufficient to initiate empiric antibiotic therapy. Confirmation of the pneumonia, though, is necessary to provide appropriate therapy while avoiding overtreatment. Sputum cultures are often unreliable as they will grow multiple pathogens because of contamination. The addition of an appropriately interpreted gram stain to a sputum culture as well as quantifying pathogens will help to determine the predominance of one type of bacteria. Obtaining lower respiratory tract cultures, such as endotracheal aspirates, bronchoalveolar lavage, or protected specimen brush, remain the mainstay of diagnosis.

C. Treatment. Once cultures have been obtained, empiric antibiotic therapy should be started immediately. Empiric antibiotic therapy that is delayed or inadequate in spectrum may increase mortality. The initial empiric regimen is guided by whether the patient is at risk for multidrug-resistant organisms, such as methicillin-resistant Staphylococcus aureus (MRSA), P. aeruginosa, and Acinetobacter species. Risk factors for multidrug-resistant organisms include hospitalization of greater than 5 days, antimicrobial therapy in the preceding 90 days, high frequency of antibiotic resistance in the hospital unit, and immunosuppressive disease or therapy. All empiric antibiotics should be administered intravenously to ensure maximum efficacy; monotherapy is recommended for patients without risk factors for infection with multidrug-resistant organisms. Recommended monotherapy includes ceftriaxone, levofloxacin, moxifloxacin, ciprofloxacin, ampicillin/sulbactam, or ertapenem. For patients at risk for multidrug-resistant pathogens, empiric therapy should include a combination of three antibiotics to provide adequate coverage. Two antibiotics should be directed toward coverage of aerobic gram negatives, especially P. aeruginosa. Choices for the first antibiotic are either an antipseudomonal cephalosporin (e.g., cefepime, ceftazidime) or an antipseudomonal carbepenem (e.g., imipenem, meropenem) or a B-lactamase inhibitor (e.g., piperacillin-tazobactam). The second antibiotic
should be either an antipseudomonal fluoroquinolone (e.g., ciprofloxacin, levofloxacin) or an aminoglycoside (e.g., amikacin, gentamicin, tobramycin). The third antibiotic should be either linezolid or vancomycin to provide coverage against gram-positive cocci, especially MRSA. Supplemental oxygen, physical therapy, bronchial toilette, and adequate fluid replacement is necessary in addition to antibiotics.

To avoid overtreatment of patients, it is necessary to reassess the patient’s respiratory cultures and clinical status 48–72 h after the initiation of empiric therapy. If the patient has not clinically improved, the clinician needs to search for other pathogens, complications, or sites of infection. If the cultures are negative and the patient has clinically improved, antibiotics should be discontinued. For all positive cultures, therapy should be de-escalated. Appropriate antibiotics should be continued for 7–8 days unless *P. aeruginosa* is the etiologic agent in which case therapy should be continued for 15 days.

**D. Prevention.** Recommendations for the prevention of nosocomial pneumonia include staff education, compliance with alcohol-based hand washing, and isolation of patients with multidrug-resistant pathogens. Additionally, avoidance or minimization of mechanical ventilation reduces the incidence of nosocomial pneumonia. Assessing patient’s swallowing ability and consciousness, removing nasogastric tubes, and abstinence from smoking prior to surgery decrease a patient’s risk of developing nosocomial pneumonia. Pre- and postoperative respiratory care and semirecumbent positioning also aid in preventing nosocomial pneumonia.
Nosocomial Pneumonia

A. General Considerations
   Predisposing factors:
   - Age > 70
   - Chronic lung disease
   - Depressed consciousness
   - Immune deficiency
   - Organ failure
   - Mechanical ventilation
   - Smoking
   - Chest surgery
   - Medications

B. Diagnosis
   - New or progressing CXR infiltrate
     AND
     - TWO of:
       - Fever > 38°C
       - Leukocytosis
       - Purulent secretions

   Obtain lower respiratory tract culture and gram stain

C. Treatment
   Start Empiric Antibiotics promptly.
   Is patient at risk for multidrug-resistant organism?
   - Yes
     Antipseudomonal cephalosporin
     Or
     Antipseudomonal carbepenem
     Or
     B-lactamase inhibitor
     -PLUS--
     Antipseudomonal fluoroquinolone
     Or
     Aminoglycoside
     -PLUS--
     Linezolid
     Or
     Vancomycin
   - No
     Ceftriaxone
     Or
     Fluoroquinolone
     Or
     Ampicillin/sulbactam
     Or
     Ertapenem

   Improved clinical status after 48-72 hours?
   - Yes
     Narrow antibiotic therapy and treat for 7 days. If p. aeruginosa, treat for 15 days
   - No
     Search for other pathogens, complications, or infections. Adjust antibiotics based on cultures available

D. Prevention
Part X
Breast Disorders
Palpable breast lumps are the most common breast problem for which women or men seek medical attention. The discovery of a breast lump by the patients themselves or their physician causes a great deal of anxiety and fear, and a marked sense of urgency for both the patients and their practitioner. The likelihood of a lesion being malignant depends in large part on the patient’s age, increasing as one gets older. Approximately 2% of all breast cancer cases occur in women younger than 30 years, increasing to ~70% in women over the age of 50.

A. History and Physical Exam. The approach and workup of a breast mass should be done in a systematic manner in order to avoid unnecessary tests or procedures. As with any other medical problem, the workup begins with a thorough history and physical exam. In women younger than 25 years, the mass is most likely a fibroadenoma or fibrocystic mass. In the 30–40-year-old group, cystic breast disease is more common than cancer; however, cancer is most likely in a postmenopausal woman. One should inquire into the family history to detect the possibility of a genetic risk for breast cancer. Other important aspects of the history include a menstrual history, history of trauma to the breast, the use of hormone replacement therapy, and whether the patient has had radiation therapy to the chest in the past. Specific questions pertaining to the mass should include the length of time it has been present, at what point in the menstrual cycle the lesion was noted and whether it has changed after completing a cycle, and if there is tenderness associated with the mass and if so does the pain change with the menstrual cycle.

A thorough physical exam should be performed with attention to the supraclavicular and axillary lymph node basins. The patients should ideally be examined 3–10 days after the onset of their menses. One cannot always distinguish a cyst from a solid lesion on clinical exam as a very tense cyst will feel quite hard. In women younger than 30 years, the “mass” may actually be an area of asymmetric, tender, and fibrocystic tissue. An area of asymmetric thickening in a postmenopausal woman, however, should raise suspicion for malignancy. It is important to remember that postmenopausal women on hormone replacement therapy (HRT) can continue to have premenopausal lesions.

B. Diagnostic Evaluation. Once the history and physical exam have been performed, one must determine what radiologic studies should be obtained if any. For the patient who is younger than 30 years, an ultrasound of the breast is preferred. Mammograms of women in this age group are rarely helpful as the breast tissue is too dense to allow differentiation of lesions from the surrounding stroma. A focused ultrasound over the lesion in question can quickly distinguish between a fluid-filled cystic lesion and a solid fibroadenoma. In women over 30, a mammogram should be obtained, followed by an ultrasound if further clarification of the lesion is needed. An ultrasound should not be used as a screening test, but rather as a focused diagnostic tool. If the patient has had a screening mammogram performed within the last 6 months, one may order a unilateral diagnostic mammogram of the affected breast, including magnification views if needed. If it has been longer than 6 months since the last mammogram and one is suspicious for malignancy, bilateral mammograms with a diagnostic study on the side of the lesion may be ordered.

The next step is to determine whether a biopsy is needed and, if so, what type of biopsy should be performed. The following guidelines may be helpful:

1. For patients younger than 30 years, the lesion is most likely a fibroadenoma or cyst. An ultrasound should be obtained to determine this. A cyst will appear characteristically as an anechoic, well-circumscribed, compressible lesion with through transmission and bilateral edge shadowing. A fibroadenoma will appear as a homogeneous, hypoechoic, well-circumscribed mass with minimal through transmission. If the ultrasound clearly shows a cyst or fibroadenoma, the mass could be followed clinically with repeat clinical exam and ultrasound in 3–6 months. If the cyst is causing pain or becomes larger, a needle aspiration of the cyst can be performed. If the ultrasound study is not clearly able to characterize the lesion in question then a biopsy should
be done, first with fine needle aspiration (FNA) and, if one does not obtain fluid, then a core biopsy. FNAs are frequently unreliable in diagnosing a fibroadenoma as the dense rubbery tissue does not easily yield cells for cytology. If a fibroadenoma is likely, based on the ultrasound appearance, then a core biopsy will be a more reliable method of obtaining tissue.

2. In premenopausal patients older than 30, the lesion is usually a cyst, but the risk of cancer is higher than in the previous group. If the mammogram and ultrasound clearly show a cyst, then the patient can be observed, especially if multiple cysts are present. Again, if the lesion is tender or enlarging or if there is a need for reassurance, the cyst can be aspirated. If the fluid appears benign (straw colored), it may be discarded. If this is a recurrent cyst or the fluid is turbid or bloody, then the fluid should be sent for cytology. If the lesion is solid, then either an FNA or a core biopsy should be performed. The FNA/core biopsy can be done by palpation if the lesion is easily felt or under ultrasound guidance if the lesion is indistinct. A fibroadenoma on biopsy may be observed with repeat exam in 3–6 months. If there are any atypical cells on biopsy, if the lesion becomes larger, or if the patient prefers, the lesion can be excised. If the lesion appears suspicious on mammogram or ultrasound, an FNA or core biopsy can be performed to confirm cancer and allow for a more thorough planning and discussion of the surgical options.

3. In the postmenopausal woman, a biopsy should be performed on all palpable masses as the incidence of cancer is much higher in this population. Again, the biopsy may be done by either an FNA or a core biopsy. If the core biopsy is not conclusive, then an incisional biopsy will need to be performed prior to definitive therapy.

In the management of palpable breast masses, it is important to remember that performing a biopsy is never a wrong approach, but one should consider doing this in the least invasive manner possible that will yield the information needed. All patients who do not undergo excision of the mass should return for follow-up 3–6 months after the initial evaluation.
Palpable Breast Mass

A. History and Physical Exam
   elicit risk factors for breast cancer

B. Diagnostic Evaluation
   ultrasound
   mammograms

Management

Age

< 30 years

- cyst
  → aspirate or observe at 3 month intervals

- fibroadenoma
  → observe, repeat ultrasound at 3-4 months, excise lesion if it enlarges

- indeterminate
  → FNA or core biopsy

≥ 30 years

premenopausal

- cyst
  → observe, repeat ultrasound in 3-4 months, aspirate if it enlarges

  → aspirate, if lesion recurs or is bloody, send fluid for cytology

- solid
  → FNA or core biopsy; atypical cells mandates excision of lesion

postmenopausal

→ FNA or core biopsy; if positive, plan treatment
A. Breast Imaging Reporting and Data System. Annual screening mammography for early detection of breast cancer has become a generally accepted and widely practiced test in the United States. Patients with abnormal mammogram seek the advice of their general/breast surgeons for management. In order to avoid uncertainty and provide uniformity of report, the American College of Radiology has devised BI-RADS (Breast Imaging Reporting and Data System), which is used by all radiologists to summarize mammograms (Table 82.1). Abnormal mammograms are broadly divided into (1) masses, (2) calcifications, (3) asymmetric densities, and (4) architectural distortions. Each one of these abnormalities may be graded on BI-RADS scale.

B. Clinical Risk Factors. In evaluating the patient’s risk for breast cancer, one also has to assess the individual clinical risk factors. This means obtaining a relevant history and performing a physical examination. This segment of the patient’s risk for breast cancer may be categorized as Low—for example, younger than 45 years, nulliparous, and having a second degree relative with breast cancer; Intermediate—prior radiation to the chest and neck, breast biopsy showing atypia, or receiving postmenopausal hormone therapy; High—history of ipsi- or contralateral breast cancer, first degree relative with breast cancer, and previous breast biopsy showing lobular carcinoma in situ.

At this point, the surgeon should combine the mammographic and clinical risk scores and advise the patient whether to observe or intervene according to the algorithm.

The introduction of ultrasound into surgical practice, and specifically for the evaluation of breast lesions, has drastically changed the management of patients with abnormal mammograms. Breast lesions detected as masses by mammography may be determined in the office by ultrasound to be either solid or cystic or simple or complex. As a general rule, simple cysts (characteristic sonographic features) are observed, complex cysts are drained, and solid masses are needle-core biopsied under local anesthesia with ultrasound guidance. Masses smaller than 5 mm and microcalcifications are difficult to visualize with ultrasound; these are sampled with stereotactic techniques. Wire localization and excisional biopsy is utilized whenever the means or expertise of image-guided technique is not available. It is now generally accepted that the accuracy of image-guided needle biopsy is equal to that of the open biopsy.

C. Image-Guided Biopsy. Only 20–25% of patients with abnormal mammograms have cancer, usually in the BI-RADS 5 category. Over the past decade minimally invasive, image-guided (stereotactic, ultrasound) needle biopsy has replaced wire localization and surgical excision for the diagnosis of abnormal mammograms. Magnetic resonance imaging (MRI) may have future application in this regard. As a rule, one attempts to establish the diagnosis percutaneously before taking the patient to the operating room. This will allow the surgeon to fully discuss the treatment options with the patient and her relatives. Several needle types of different sizes either manually operated or power driven (Mammotome) are employed to obtain tissue samples for histologic diagnosis. The procedure is performed under local anesthesia and aseptic conditions either in an office setting or in a surgicenter. Some image-guided needle biopsies are inadequate for histological evaluation, meaning that the pathologist does not find adequate breast tissue in the sample to make a diagnosis. Consequently, this has fostered the use of larger needles and a greater number of samples taken from the breast lesion which, not surprisingly, occasionally result in total removal of smaller lesions. Since only 20–25% of abnormal mammograms have malignancy (in situ and invasive) and another 5–10% reveal only atypia, the majority of breast biopsies are benign. Against that background, one has to interpret the pathologist’s report.

There are several caveats with respect to image-guided breast biopsies. First, if the biopsy is being done for microcalcification, adequate sampling must be verified by seeing calcifications in a specimen mammogram and by seeing the needle traverse the target on the x-ray. Second, there should be concordance between the image and the histologic reading. For example, if the needle biopsy of a BI-RADS 4 or 5
mammographic lesion is benign, either a repeat image-guided biopsy or wire localization and open biopsy should be considered. Concordance provides the clinician with a clear course of action. If core biopsy shows that the lesion is a fibroadenoma, and images confirm that this is the most likely diagnosis, the lesion may be followed with serial imaging studies. If the lesion gets bigger or if the patient has pain, the lesion should be removed. If core biopsy shows cancer, definitive treatment may be discussed. Third, if a needle biopsy of a lesion demonstrating mammographic architectural distortion or microcalcification reveals atypical cells, excisional biopsy should be recommended.

Table 82.1. BI-RADS™ mammographic assessment.

<table>
<thead>
<tr>
<th>Category</th>
<th>Assessment</th>
<th>Description and management recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Incomplete</td>
<td>Needs additional workup, such as spot compression or ultrasonography, prior to assigning final assessment</td>
</tr>
<tr>
<td>1</td>
<td>Negative (normal mammogram)</td>
<td>There is nothing to comment on. Routine screening</td>
</tr>
<tr>
<td>2</td>
<td>Benign finding negative</td>
<td>A definitely benign finding described. Routine screening</td>
</tr>
<tr>
<td>3</td>
<td>Probably benign</td>
<td>Very high probability of being benign. Short-term follow-up (usually 6 months) recommended to establish stability</td>
</tr>
<tr>
<td>4</td>
<td>Suspicious abnormality</td>
<td>Not characteristic, but has reasonable probability of being malignant. Biopsy should be urged</td>
</tr>
<tr>
<td>5</td>
<td>Highly suggestive of malignancy</td>
<td>High probability of being cancer. Appropriate action should be taken</td>
</tr>
</tbody>
</table>

Adapted from the American College of Radiology Breast Imaging Reporting and Data System (BI-RADS™)
Abnormal Mammogram

A. BI-RADS system used to summarize mammograms

1. average
   screen per American Cancer Society

2. low / intermediate
   imaging in 6-12 months
   high
   imaging in 3-12 months

3. low
   imaging in 3-6 months
   intermediate / high

C. Image Guided Biopsy
   verify needle placement
   image/tissue concordance
   presence of atypia

4. low / intermediate / high
   wire localized excision

B. Clinical Risk Factors

low - age < 45 years, nulliparous
intermediate - prior radiation to chest and neck, breast biopsy showing atypia, hormone replacement therapy
high - history of breast cancer or lobular carcinoma in situ, strong family history of breast cancer
Invasive Breast Cancer

Mehra Golshan

The estimated number of cases of invasive breast cancer in 2006 exceeded 214,000; nearly 40,000 will die each year from the disease. The vast majority of patients are women, although 1,700 men will be affected. Factors associated with increased risk include increasing age, early onset menarche, late menopause, nulliparity, late first pregnancy, and family history of breast and/or ovarian cancer. The genetic predisposition to breast cancer through the BRCA 1 or 2 mutations account for nearly 7% of the cases of breast cancer in the United States. The median age at diagnosis is 61.

The primary pathologic subtypes of breast carcinoma are invasive ductal (80%), invasive lobular (10–15%), and mixed ductal/lobular carcinoma. The overall survival and outcome is the same for these groups stage for stage. Rare types include medullary, mucinous, tubular, adenoid cystic, and metaplastic carcinomas.

The incidence of breast cancer continues to increase at a rate of 0.3% per year, while mortality has decreased by over 2% per year. With the advent of widespread screening mammography, breast cancer is detected at a much earlier stage than decades past. Surgical treatment has evolved from the Halsted radical mastectomy where the nipple, areola, breast, pectoralis muscles, and axillary lymph nodes were removed to localized treatment pioneered by Drs. Bernard Fisher and Umberto Veronesi with lumpectomy and quadrantectomy, respectively.

A. Clinical Stage I/II. For early-stage breast cancer (Stage I or II), usually surgery is undertaken first, although recent studies have begun to look at preoperative chemotherapy and hormonal therapy in this population. Surgical options include breast conservation with radiation and sentinel lymph node biopsy versus mastectomy with sentinel node biopsy. When mastectomy is performed, reconstruction is offered. The primary types of reconstruction include implants, latissimus flap, and TRAM (transverse rectus abdominis myocutaneous) flap reconstruction. The type of reconstruction depends on the patient’s body habitus and expectations for reconstruction.

The traditional management of the lymph nodes has been through a level I and II axillary lymph node dissection. In the early 1990s researchers at the John Wayne Cancer Institute, University of Vermont and Milan in Italy, began studying and reporting on the technique of sentinel lymph node biopsy. The concept of the sentinel node is that it is the first lymph node in the nodal chain that drains a breast cancer. If that node does not contain metastatic disease, then it is very unlikely that further lymph nodes are involved with breast cancer. The technique requires the injection of blue dye, lymphazurin, and/or technetium-labeled sulfur colloid into the breast prior to surgery and identification and removal of the labeled lymph node(s) for pathologic examination. In women diagnosed as clinically node-negative, the sentinel node has replaced the axillary lymph node dissection without detriment to survival or local recurrence. The morbidity of lymphedema, which was between 15 and 20%, is now below 2%. Axillary lymph node dissection is routinely performed when the sentinel lymph node contains metastatic disease or if the axilla is clinically positive.

Women with Stage I or II breast cancer are offered chemotheraphy and/or hormonal therapy and/or trastuzumab based on the nodal status, tumor size, hormone receptor status, Her2neu receptor status, and menopausal status. The type and duration are individualized by the patient’s particular cancer. In general, women with cancers greater than 1 cm in size are offered adjuvant chemotherapy and/or hormonal therapy.

B. Radiation. With breast conservation, radiation is necessary and this reduces local recurrence to 8–10%. Women who undergo mastectomy usually do not require radiation unless the tumor is greater than 5 cm or there are more than four lymph nodes positive for metastatic disease. Based on the results of seven large randomized prospective studies, with the two largest having over 20-year follow-up, survival is exactly the same should a woman choose breast conservation or mastectomy. Emerging data in the elderly population suggest that in patients with node-negative early-stage breast cancer treated with
lumpectomy and Tamoxifen, radiation may be forgone with no
difference in survival but an increased local recurrence.

C. *Adjuvant Therapy.* In general, for *premenopausal* patients
with tumors <1cm and negative nodes, usually no treatment
is given. Tamoxifen may be given, usually for 5 years. Larger
tumors and any patient with axillary nodal metastases are treated
with chemotherapy (see Algorithm), which is typically anthra-
cycline based; taxanes are added for the node-positive patient.
*Postmenopausal* women who are hormone receptor positive
are treated with aromatase inhibitors for 5 years (has replaced
Tamoxifen). Chemotherapy is given for any postmenopausal
woman with lymph node metastases, and it may be considered
for women diagnosed as node-negative with larger tumors.

D. *Stage III/IV Breast Cancer.* For patients with Stage III or
IV breast cancer, chemotherapy or hormonal therapy with or
without trastuzumab is generally offered. Women with Stage
III cancer who respond to therapy are then treated with a mod-
ified radical mastectomy; however, data support the use of
breast conservation followed by regional radiation therapy in
a select group. In patients with Stage IV or metastatic breast
cancer, surgery has no role except for palliation.

Inflammatory breast cancer constitutes 2–3% of breast
cancer cases in the United States each year. Clinical signs,
which can include skin thickening, peau d’orange, and nipple
inversion, are usually seen in a rapid sequence of changes.
A punch biopsy of the overlying skin will often show dermal
tumor lymphatic invasion. Initial therapy is with induction
chemotherapy followed by a modified radical mastectomy
and then radiation therapy. If the tumor is hormone positive,
hormonal therapy with Tamoxifen or an aromatase inhibitor
is utilized.
Invasive Breast Cancer

A. Clinical Stage I or II
- Surgery
  - Lumpectomy
  - Sentinel Node Biopsy
  - Mastectomy
  - Offer reconstruction

B. Radiation

C. Adjuvant Therapy

D. Clinical Stage III or IV

Pre-menopausal
- Tumor <1cm
  - (-) Nodes: No Treatment
  - (+) Nodes: Chemotherapy +/− Tamoxifen
- Tumor >1cm
  - (-) Nodes: Chemotherapy or Hormonal
  - (+) Nodes: Mastectomy and Nodal Evaluation

Post-menopausal
- Tumor <1cm
  - (-) Nodes: No Treatment Or Hormonal
  - (+) Nodes: Chemotherapy +/− Hormonal
- Tumor >1cm
  - (+) Nodes: Poss Chemo +/− Hormonal
  - (-) Nodes: No Treatment
Nipple Discharge
Neha D. Shah and Darius Francescatti

The human breast serves the physiologic purpose of production and secretion of milk. Milk that is secreted from the nipple navigates a complex ductal system to reach the nipple–areolar complex. This ductal system begins with microscopic milk-producing acini which are grouped into lobules. The acini have receptors sensitive to serum prolactin levels (highest during the late luteal phase of the menstrual cycle and during pregnancy) and produce milk accordingly. Once produced and secreted at the acinar level, milk travels toward the nipple through a network of intralobular terminal ductules which converge to form ducts. Beneath the nipple–areolar complex these ducts converge to form lactiferous sinuses which then lead to openings in the nipple. The histology of the ductal system is composed of a layer of A cells (milk-producing cells), B (or chief) cells (the energy source), myoepithelial cells, and an underlying basement membrane. Myoepithelial cells contract in response to serum estrogen, progesterone, and prolactin levels, with resultant secretion of milk from A cells. Also, oxytocin is secreted by the posterior pituitary gland in response to nipple stimulation; it results in contraction of myoepithelial cells, thus inducing milk to be secreted from the lobules to the ducts. The entire ductal and lobular network is invested in connective tissue stroma, lymphatics, and adipose tissue. In the postmenopausal breast, tissue parenchyma (ducts and lobules) will involute to variable degree in response to decreased hormonal stimulation.

Etiology of Nipple Discharge. A. Abnormal nipple discharge can be defined as spontaneous discharge from the nipple in a nonlactating woman. Approximately 5% of women seen in breast clinics complain of spontaneous nipple discharge. Although benign etiologies are most common, a physician’s first priority is to differentiate malignant causes (DCIS, LCIS, and invasive carcinoma) from benign causes (physiologic discharge, intraductal papilloma, duct ectasia, fibrocystic disease, hormonal abnormality, cyst, and iatrogenic).

B. The initial step in investigating the etiology of spontaneous nipple discharge is a thorough history and physical examination. The first goal of this initial assessment is to categorize the nipple discharge as either physiologic or nonphysiologic. Physiologic discharge is usually bilateral, arises from multiple ducts, and is milky in appearance. It can occur during pregnancy, prior to menses, with iatrogenic hormonal stimulation, or with abnormal serum hormonal levels. In contradistinction, nonphysiologic nipple discharge often appears serous, serosanguinous, or bloody in nature; it is usually unilateral and issues from a single duct. This latter type of discharge is more suspicious for malignancy.

Patient history may reveal an association between nipple discharge and the timing of menses, pregnancy, or trauma. The examiner may suspect a malignant process in a patient complaining of associated mass or weight loss. Visual changes may indicate a central nervous system lesion producing high levels of prolactin. Fever and chills are indicative of a possible infective process. A medication history may reveal an iatrogenic cause of nipple discharge (i.e., estrogenic medications such as oral contraceptives and hormone replacement therapy as well as antidopaminergic drugs). Patient demographics and risk assessment may provide additional help in formulating a differential diagnosis. Male patients with nipple discharge must be carefully evaluated. In a child the cause of nipple discharge is most likely a cyst. Cytologic evaluation of any suspicious or unusual nipple discharge is essential.

On physical examination, it is important to note whether the discharge is unilateral or bilateral, its character (i.e., milky, purulent, serous, serosanguinous, and bloody), and whether a single duct or multiple ducts are involved. Historically, identification of a duct or quadrant of the breast that may be the source of the nipple discharge was accomplished by serial compression of breast tissue toward the areola in a 360° arc until fluid is expressed; the location of the diseased duct is presumed within the area compressed. A complete examination of the breast may reveal an associated mass, dimpling of the skin, or lymphadenopathy. Tenderness and erythema may lead one to a diagnosis of abscess. Breast sonography should always be performed as an adjunct in the evaluation of nipple discharge. The echogenicity of breast lesions helps
showing ductal irregularity or dilation. Further evaluation should be considered.

*Diagnostic Approach.* C. Spontaneous nipple discharge should be evaluated immediately. A variety of etiologies can give rise to this finding including intraductal papilloma (~40% of patients with nipple discharge), breast carcinoma (~8%), duct ectasia, and fibrocystic disease and must be considered as well.

Nipple aspirate fluid (NAF) should be sent for cytology and a diagnostic mammogram ordered. However, mammography has been shown to be a less-sensitive test for malignancy in patients with spontaneous nipple discharge without other associated symptoms or physical findings. With this in mind, cytologic evaluation should be considered essential. Cytology for nipple discharge may be examined via NAF or by ductal lavage. In the former, a suction device is placed over the nipple to aid in identifying the source of the discharge and a sample is obtained for analysis. In the latter method, a thin cannula is inserted into each discharging duct and saline lavage performed. The resultant fluid is then aspirated and cytologically examined. Ductal lavage produces highly cellular samples for analysis. There is 87% reported sensitivity for detection of malignant lesions.

Cytologic atypia demands an etiologic workup. Cellular and noncellular components of lavage fluid can be analyzed to provide information beyond what traditional cytology has proved. Polymerase chain reaction (PCR) and fluorescent in situ hybridization (FISH) allow amplification of genomic information in the lavage sample enabling the pathologist to directly study premalignant and malignant chromosomal abnormalities. The noncellular component of lavage fluid can be analyzed for hormonal levels (estrogen, progesterone, and prolactin), tumor markers (PSA, CEA), and for the presence of growth factors (fibroblast growth factor-2) and receptors (Her-2/neu, ER, PR). As tests become more sophisticated and correlated with biopsy-proven malignancy, the sensitivity and specificity of cytology can only continue to increase.

If cytologic or mammographic studies are suggestive of malignancy, either a targeted excisional biopsy or further workup for malignancy should be pursued. Regardless of whether cytology or mammography is negative, the abnormal draining duct must still be explored, if possible, and excised. Historically galactography, the introduction of contrast dye into the discharging duct, was used to delineate the ductal tree in order to localize the duct for excision. Galactography has been shown to be helpful in localizing the abnormal duct by showing ductal irregularity or dilation.

Today, however, diagnostic and/or therapeutic intraluminal intervention can be aided via the ductoscopic exploration of the breast. The abnormal duct is entered utilizing a 0.9 mm ductoscope. This procedure can be done in the office or used as an adjunct in the operating room to guide excisional biopsy. Visualization has been reported to be successful in ~75% of cases. Ductoscopy is more sensitive than galactography in detection of ductal lesions. In one study ductoscopy detected approximately twice the number of pathologic ductal lesions as galactography and will facilitate the detection of lesions to an effective range of up to 10 cm distal to the areola (5 cm for ductography). Furthermore, the ductoscopic technique was shown to have a 57% sensitivity for detection of breast cancer based on ductal wall appearance (visualized irregularity or mass). The injection of methylene blue into the abnormally discharging duct can be useful in identifying the duct and its ramifications, thus facilitating a complete excision. A persistent abnormal discharge despite a thorough negative workup, which includes ductoscopy, mandates the continued pursuit of a diagnosis. Magnetic resonance imaging (MRI) should be considered in such cases.

As an emerging technique, further studies are required to thoroughly assess the clinical scope of ductoscopy. Ductoscopic visualization and histologic analysis of breast lesions may, in the near future, provide a less-invasive method of differentiating a benign from a malignant lesion. Ductoscopically directed surgical excision may reduce the need for reexcision for positive margins following lumpectomy. The technologies of ductal lavage and ductoscopy are rapidly advancing. These techniques can potentially revolutionize the screening, diagnosis, and treatment of established breast malignancy.

**C. Spontaneous nipple discharge**

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**D. Physiologic nipple discharge**

- **Diagnostic Approach.**
  - **D. Physiologic nipple discharge** (nonbloody, bilateral, and milky discharge from multiple ducts) can be the result of recent pregnancy, iatrogenic cause, or elevated serum prolactin (pituitary mass and hypothalamic tumor). Serum prolactin should be measured. The patient may require a CT scan for evaluation of the pituitary and hypothalamus.

  **E. Purulent drainage** may be indicative of an abscess, in which case gram stain and culture should be obtained to ensure appropriate antibiotic treatment. If persistent, despite antibiotic therapy, an abscess must be drained. Nipple discharge can also be a manifestation of systemic illness such as Cushing’s disease, renal failure, or hypothyroidism. Once a diagnosis of iatrogenic hyperprolactinemia has been made, it can be effectively treated by discontinuing a prolactin agonist drug or by prescribing a dopamine agonist (such as bromocriptine). Depending on the etiology benign nipple discharge can be effectively treated and if total resolution does not occur, a continued diagnostic workup is indicated.
A. Patterns of Pain. Breast pain (mastalgia) is the most common complaint associated with disorders of the breast, being present in 45–85% of women who present for evaluation of breast symptoms. It is important to note that the presence of breast pain does not exclude cancer, as some studies show that up to 5% of patients with breast cancer present with pain. The most common type is a cyclical pattern related to the hormonal changes of menses. These patients invariably have nodularity in the breast and pain that lasts for the week preceding menstruation. The pain is usually relieved by the onset of bleeding. The second most common pattern is noncyclical, which accounts for more than a fourth of women with breast pain. These patients describe a burning discomfort that is not relieved by menses. The amount of nodularity is much less prominent and there may be no palpable abnormality. Breast nodularity is probably a reflection of the complicated hormonal milieu, namely, ovarian estrogen production, diminished progesterone secretion, and hyperprolactinemia.

Chest discomfort is not always breast in origin. Tietze’s syndrome is pain at the costochondral junction. There is no palpable abnormality, and the pain is unilateral and runs a chronic course.

B. Management. The first step is a thorough history and physical examination. A dominant lump or lesion should be approached as if it contained cancer. A mammogram is essential. Most women with mastalgia have diffusely nodular breasts and management includes reassurance that the pain is physiological.

Nutritional therapy: Restricting the intake of methylxanthines such as caffeine may decrease the amount of pain. Reducing dietary fat improves cyclical breast tenderness. Evening primrose oil (EPO) causes a significant reduction in both cyclical and noncyclical pain.

Endocrine therapy: Danazol is an attenuated androgen which competitively inhibits estrogen and progesterone receptors. It is the only drug approved by the Food and Drug Administration (FDA) for mastalgia. Luteinizing hormone-releasing hormone (LHRH) agonists or analogs cause direct inhibition of ovarian steroidogenesis (essential for producing ovarian ablation). LHRG should only be used for acute and severe mastalgia because of multiple side effects including loss of trabecular bone.

Tamoxifen: Tamoxifen is an estrogen agonist–antagonist thought to competitively inhibit the action of estradiol on the mammary gland. Unfortunately the risk of endometrial carcinoma should relegate the drug to only severe cases when all standard therapies have failed.

Nonendocrine therapy: Bromocriptine has shown significant clinical response for cyclical breast pain by lowering prolactin levels.
Breast Pain

A. 
Patterns
1. cyclical-related to menses, relieved by onset of menses
2. non-cyclical-less nodularity

B. 
Management
order mammograms

mass present?

assess pain severity

no

minor
reassurance
diet modification

yes

exclude cancer

severe

evening primrose oil
hormonal:
-danazol
-LHRH analogues
-bromocriptine
-tamoxifen
A. Epidemiology and Risk Factors. Ductal carcinoma in situ (DCIS) is a form of noninvasive breast cancer which usually affects women in their sixth decade of life. The disease is characterized by a proliferation of malignant ductal epithelial cells without microscopic invasion through the basement membrane. The categories of DCIS include comedo and non-comedo. The comedo subtype is identified by the presence of necrotic cellular debris within the ducts and large pleomorphic nuclei with numerous mitoses. It is the most common form and also the most aggressive. Noncomedo DCIS has low mitotic rates and does not exhibit central necrosis. It is further classified into cribriform, solid, micropapillary, and papillary subtypes. It is believed that all forms of DCIS will eventually become invasive breast cancer if left untreated.

Risk factors for the development of DCIS, as well as any breast cancer, include nulliparity, late age at first pregnancy, young age at menarche, obesity, family history of breast cancer, previous history of benign breast disease, and moderate to high levels of alcohol use.

Over the past two decades, the incidence of DCIS has gone from less than 1% to over 17% of all new breast cancers. Over half of breast cancers diagnosed by screening mammography are DCIS. This rise in incidence is attributed to the increased utilization of screening mammography and improved imaging technology.

B. Diagnosis. Before the widespread use of screening mammography, patients with DCIS typically presented with a palpable mass or nipple discharge. For the patient with nipple discharge, incisional or excisional biopsy is appropriate for making the diagnosis. Recently, however, routine mammograms are detecting clinically occult lesions more frequently. Common radiographic findings in patients with DCIS include microcalcifications (72%), a soft-tissue density (10%), and microcalcifications in conjunction with a soft-tissue density (12%). DCIS is not detected radiographically in 6% of cases. Excisional biopsy of the breast tissue containing the microcalcifications is performed to confirm the diagnosis. Alternatively, stereotactic or core biopsies can be obtained especially if the microcalcifications are diffuse or multicentric; however, negative biopsies do not rule out DCIS as sampling error may miss the involved tissue.

C. Treatment. Treatment options vary and range from simple mastectomy (also known as total mastectomy) with or without reconstruction to breast-conserving surgery with or without radiotherapy. The National Cancer Database reports an increase in breast-conserving surgery with radiotherapy from 38 to 54% between 1985 and 1993. Breast-conserving treatment involves wide local excision of the tumor with clear margins and is more commonly used on smaller tumors of more favorable grade. The definition of clear margins is controversial; negative microscopic margins are acceptable in many institutions, while other centers require 1.0-cm margins. Breast-conserving surgery alone is considered adequate treatment for a single focus of DCIS, less than 1.0 cm, with clear margins. Radiotherapy is added to this regimen for tumors greater than 1.0 cm with clear margins and no evidence of comedo subtype or necrosis. The addition of radiotherapy lowers the recurrence rate (from 16.4 to 7%) and makes it more likely that the recurrence will be a noninvasive tumor. Simple mastectomy is advised for younger patients as well as those with larger tumors, comedo subtype with necrosis, nipple involvement, or radiographic findings of scattered or multiple foci of microcalcifications.

Axillary lymph node metastases are reported in less than 1% of all cases. Therefore, axillary lymph node dissection has not been traditionally recommended considering the morbidity of arm lymphedema. However, some centers are utilizing sentinel lymph node dissection for larger tumors or multicentric disease.

Follow-up mammography is recommended at 3 months and 6 months post-op, every 6 months for the following 2–3 years, and then yearly.
Ductal Carcinoma In Situ

A. Epidemiology, risk factors, pathology
- malignant proliferation of epithelial cells
- no invasion through basement membrane
- types: comedo (necrosis), non-comedo
- increased risk in patients with late first pregnancy, nulliparous, young menarche, obesity, family history breast cancer

B. Diagnosis
- physical exam
- mammography

palpable mass
mammographic abnormality (non-palpable)

excisional biopsy with clear margins
wire localized breast biopsy

review histology

C. Treatment

single focus DCIS < 1 cm, clear margins of resection, non-comedo subtype
breast conserving surgery alone

single focus DCIS > 1 cm, clear margins of resection, non-comedo subtype
breast conserving surgery and radiotherapy

multicentric disease, large tumor, comedo subtype
simple mastectomy +/- reconstruction, +/- sentinel node axillary dissection
Part XI
Head and Neck Disorders
Although the majority of facial masses are benign, all masses demand workup and tissue diagnosis. The history, physical exam, age of the patient, and chronicity of the lesion determine the differential diagnosis. Most pediatric lesions and adults with acute masses are likely to be caused by infectious diseases, while adults with slow-growing chronic lesions are at highest risk for malignancy.

A. Differential Diagnosis. Pediatric infectious lesions are most often due to mumps or Coxsackie virus, while infections in adults are often caused by Staphylococcus Aureus.

Noninfectious benign lesions can be broken up into epithelial and nonepithelial masses. Epithelial lesions include mixed tumor of the parotid (80%), Warthin tumor, or less commonly adenoma/oncocytoma. Nonepithelial causes of parotid masses include lymph nodes, cysts, lipoma, lymphoepithelial lesions, or capillary hemangioma in children.

Malignant lesions include mucoepidermoid, which is the most common malignant tumor, adenoid cystic CA, mixed malignant tumor, acinic cell CA, adenocarcinoma, and squamous cell CA.

B. History and Physical Exam. History should glean the presence of autoimmune disease, infectious disease, and any familial autoimmune or malignant disease. Smoking history and any recent history of facial trauma are also important.

Initial physical exam is directed at determining whether the lesion is intra- or extraparotid. This is done by palpating the mass and attempting to roll it over the styloid process and under the mandible. If the lesion is mobile and rolls under the mandible it is likely a lymph node. Parotid masses will not be freely mobile and will not move below the mandible. Additionally, care should be taken to examine the patient carefully for adenopathy.

Once the mass is located within the parotid, the exam seeks to determine if the deep lobe is involved by tumor. Cranial nerve exam of the facial nerve and its branches will delineate facial nerve involvement. Displacement of the tonsil medially is also a sign of deep-lobe involvement. Lack of either nerve involvement or tonsillar deviation does not however guarantee lack of extension of the deep lobe and further diagnostic tests and ultimately surgical resection/biopsy are necessary for delineating deep-lobe involvement.

C. Diagnostic Studies. While many surgeons choose to forgo any additional studies and proceed to the operating room for biopsy and/or resection, several other diagnostic studies can prove helpful. Ultrasound can diagnose cysts, but lacks sensitivity for deep-lobe involvement and has not been found to be cost-effective. CT scan is the gold standard and provides the most useful information regarding size, nerve, and deep-lobe involvement and nodal involvement.

Along with imaging studies, fine-needle aspiration (FNA) can provide useful preoperative histologic diagnosis. FNA can be performed in the office setting. It provides the benefit of ruling out nonepithelial pathology and predicting the need for extensive dissection and radical neck dissection. In the hands of an experienced cytopathologist, FNA is up to 80% accurate in diagnosing malignant disease. However, it is significantly less accurate in predicting tumor grade. If a malignant tumor that is likely to have high-grade features is diagnosed, the patient can be counseled about the need for facial nerve resection, radical neck dissection, and postoperative radiation. A negative FNA still necessitates surgical resection and biopsy.

D. Treatment. Treatment for epithelial lesions includes a superficial parotidectomy with frozen section. Further treatment is dependent on the histology, tumor grade, and involvement of the deep lobe and facial nerve.

If the lesion is a benign salivary gland tumor, no further treatment beyond superficial parotidectomy is necessary. If the lesion is a malignant low or intermediate grade tumor, total parotidectomy with nerve preservation (shaving of tumor off the nerve) and postoperative radiation is also considered. If the lesion is a malignant high-grade tumor, treatment includes extended or radical parotidectomy including resection of the facial nerve, a modified radical neck dissection, and postoperative radiation. Facial nerve excision should be performed in cases where there is functional impairment, or the nerve cannot be separated from tumor to provide clean margins.
Tumor can be shaved or cleaned from the nerve, leaving close margins, and postoperative radiation can be utilized to sterilize the close margin. When the nerve is resected, immediate nerve grafting should be attempted to regain some facial nerve function.

Postoperative radiation is considered for intermediate and high-grade malignant tumors and any tumors with incomplete or close margins. Controversy exists regarding the use of radiation in low-grade malignant lesions and its use should be based on patient age, comorbidities, and adequacy of resection.

E. Prognosis. Prognosis is based primarily upon the grade and type of tumor as well as the size and extent of involvement. Five-year survival rates are as follows: low-grade mucoeipidermoid—92%, acinic cell—76%, high-grade mucoepidermoid—49%, malignant mixed—40%, adenoid cystic—30%, and squamous cell—26%.
Parotid Mass

A. Differential Diagnosis
1.) Infections
2.) Noninfectious benign
3.) Malignant

B. History and Physical
1.) Autoimmune
2.) Infections
3.) Smoking
4.) Facial Trauma
5.) Intra or Extra Parotid
6.) Facial nerve exam
7.) Tonsil deviation

C. Diagnostic Studies
1.) CAT Scan
2.) FNA
3.) US not useful

D. Treatment
Epithelial Lesion
1.) Superficial Parotidectomy
2.) Frozen section
3.) Total Parotidectomy with preservation of facial nerve and post-op RT
4.) Extended or Radical Parotidectomy with modified neck dissection and post-op RT for High grade tumors

E. Prognosis (5yr Survival)
1.) Low grade mucoepidermoid 92%
2.) Acinic 76%
3.) High grade mucoepidermoid 49%
4.) Malignant-mixed 40%
5.) Adenoid cystic 30%
6.) Squamous cell 26%
Evaluation of the Suspicious Neck Mass

Patrick J. O’Leary

A. While most neck masses are benign, they are frequently the first manifestation of an aerodigestive tract malignancy. Neck masses occur in all age groups and can be categorized as congenital, inflammatory, or neoplastic. The differential diagnosis is dependent upon the patient’s age, medical and social history (smoking and alcohol), and the location of the mass. In the pediatric population, congenital and infectious causes predominate. In contrast, neck masses in patients over the age of 40 should be considered malignant until proven otherwise. Indeed, fully 85% of adult neck masses are neoplastic, and 85% of those are malignant.

B. Congenital causes. Congenital causes of neck masses include branchial cleft cysts, thyroglossal duct cysts, cystic hygroma, and hemangioma. Branchial cleft cysts may arise from any of the branchial arches and are numbered as such. First branchial cleft cysts are rare and are intimately associated with the external auditory canal and parotid gland. Second branchial cleft cysts are the most common and originate from the skin in the mid or lower neck along the sternocleidomastoid muscle. A fistula tract may run from the skin, between the internal and external carotid arteries, and open internally into the superior pole of the ipsilateral tonsil. Third branchial cleft cysts are lower on the neck and the fistula tract, when present, enters into the piriform sinus. Hallmarks for diagnosis include the pediatric age group, lateral location, and propensity for recurrent infections. Treatment is surgical excision.

Thyroglossal duct cysts are three times more common than branchial cleft cysts. They are remnants of the thyroglossal duct formed by the descent of the thyroid diverticulum from the foramen cecum of the tongue into the neck. The cysts are midline in location and can be found anywhere along the course of the duct. Surgical management requires removal of the cyst, the tract, and the central portion of the hyoid bone to prevent recurrence (Sistrunk procedure). A thyroid scan should be performed prior to excision to ensure the presence of a functioning thyroid gland.

A cystic hygroma is a congenital mass of dilated lymphatic channels commonly found in the neck. They are formed by a failure of the embryonic lymph channels to establish a communication with the venous system. Treatment is surgical excision.

Hemangiomas are classified as capillary, cavernous, and mixed. Diagnosis is based on their bluish coloration, warmth, easy compressibility followed by rapid refilling, and an occasional bruit. As most of these lesions will spontaneously resolve, observation is the treatment of choice.

C. Inflammatory Conditions. Acute lymphadenitis is the most frequent cause of a mass in the neck. It is an inflammatory enlargement of one or more lymph nodes usually secondary to a staphylococcal or streptococcal infection of the upper respiratory tract, oral cavity, scalp, or neck. The lymph nodes are tender, erythematous, and mobile. Treatment is with antibiotics and drainage of the node if it becomes fluctuant.

Mononucleosis is a viral disease caused by the Epstein-Barr virus. It is characterized by fever, malaise, a severe exudative pharyngitis, splenomegaly, and an increase in atypical white blood cells. Patients may present with diffuse cervical lymphadenopathy, usually of the posterior chain. The diagnosis is confirmed with the monospot test. Treatment is supportive with hydration and rest.

Cat scratch disease is a benign, self-limited disease caused by Rochalimaea hensleae. After 3–5 days, a characteristic papule appears at the site of inoculation. A tender regional adenopathy evolves over the next 1–2 weeks. Treatment is supportive.

Tuberculous lymphadenitis is characterized by a firm, usually asymptomatic mass in the neck. If suspected, a PPD and chest x-ray should be obtained. A fine-needle aspiration (FNA) can yield sufficient material for culture. Treatment is with long-term antibiotic therapy. The atypical mycobacterium may cause a unilateral, nontender enlargement of cervical lymph nodes, particularly in the pediatric population. A draining sinus may be present. Surgical excision is the preferred treatment.

Actinomycosis is a bacterial infection associated with dental extraction and tooth decay. Infection begins in the mouth and
spreads into the neck, forming abscess cavities and multiple fistula tracts. Diagnosis is made by identifying sulfur granules on microscopic examination. Treatment is with penicillin.

*Sialadenitis* of the submandibular gland may present as a submandibular neck mass. Pain is the most common complaint, made worse with eating. The mass is tender and warm. Treatment is with antibiotics, hydration, and removal of any ductal obstruction.

AIDS may cause a variety of cervical findings. The finding of marked lymphadenopathy is almost universal and is observed when stable. Other causes include Kaposi’s sarcoma, non-Hodgkin’s lymphoma, and a multitude of infectious etiologies.

D. *Neoplasms* of the neck may be either benign or malignant. Malignant tumors are far more common, and metastatic lesions are the most common of all. *Metastatic squamous cell carcinoma* should be suspected in any patient over the age of 40, particularly those with a history of tobacco or alcohol use. The most frequent primary site is the aerodigestive tract.

A thorough head and neck examination and triple endoscopy (laryngoscopy, bronchoscopy, and esophagoscopy) with random biopsies are used to localize the primary lesion. In up to 20% of cases the primary tumor cannot be identified. Evaluation of the presumed metastatic node with FNA or excisional biopsy is then performed to confirm the diagnosis. This may be followed with a neck dissection and radiation therapy to include regions that most commonly would harbor the primary lesion.

*Primary malignancies* of the neck are uncommonly encountered and are usually malignant lymphomas. Forty percent of patients with Hodgkin’s disease present with enlarged cervical lymph nodes which are painless and rubbery. Biopsy confirms the diagnosis.

*Benign neoplasms* of the neck are rare and include lipomas, lymphangiomas, and hemangiomas. A pulsatile mass in the neck is indicative of a carotid body tumor and biopsy should be avoided.
Evaluation of the Suspicious Neck Mass

A. Neck Mass 

0-15 Years

B. Congenital

- Branchial cleft cysts → Excision
- Thyroglossal duct cysts → Thyroid scan/Excision
- Cystic hygroma → Excision
- Hemangiomas → Observation

>40 Years

C. Inflammatory

- Acute Lymphadenitis → Antibiotics
- Mononucleosis → Monospot/Supportive Care
- Cat Scratch Disease → No treatment needed
- Tuberculosis → PPD, CXR, FNA culture, Antibiotics
- Atypical Tuberculosis → Excision
- Actinomycosis → Antibiotics
- Sialadenitis → Antibiotics, hydration, removal of ductal obstruction

16-40 Years

D. Neoplastic

- Benign (15%) → Observation or excision
- Lymphoma Suspected → Open Biopsy
- Metastatic Disease Suspected → Triple Endo FNA
- Chemo and/or RT

- Malignant (85%) → Primary Identified
- No Primary Identified
- Neck Dissection → RT
Part XII
On-Call Crises
89
Epistaxis

Mark El-Deiry

A. Introduction. Epistaxis is a common presenting symptom to many emergency rooms. Up to 64% of people will experience at least one episode of epistaxis in their lifetime. Usually this is easily controlled, but rarely more invasive measures are required.

The history is the key aspect in the treatment of epistaxis. Important details include mode of onset, duration, quantity, side, previous treatment, and treatment outcomes. The history will also uncover other underlying etiologies including blood dycraisias, trauma (usually by digital manipulation), exacerbation by cold or dry environments, chronic nasal steroid use, anticoagulation therapy, alcohol or cocaine abuse, liver failure, or dialysis. Other less common etiologies include hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease), Wegner’s granulomatosis, and neoplasia. In children it is important to consider foreign body as a cause of epistaxis. In unilateral epistaxis one must consider the possibility of neoplasia and in young adults juvenile nasopharyngeal angiofibroma (JNA) is considered. Finally, attention should be paid to whether the patient has had recent rhinological or sinus surgery.

The goal of the physical exam is usually to separate the bleeding into anterior or posterior sources. In order to adequately evaluate the nose, it is important to use proper instruments. These include a Frazier-tipped suction, nasal speculum, bayonet forceps, and a head mirror or head light. A topical decongestant mixed with anesthetic should be given prior to the examination. This can be delivered intranasally using an atomizer spray bottle. Another option includes placing intranasal cotton pledgets soaked in a 1:1000 mixture of topical anesthetic and epinephrine and removing them immediately prior to the examination. The exam should proceed from the anterior to posterior of the nasal cavity. Particular attention should be paid to the anterior septum, or Little’s area, usually considered to be the most common site of anterior epistaxis. Any crusting noted should be removed as this can often hide sources of bleeding.

Nasopharyngoscopy can be helpful in making the diagnosis, particularly in identifying posterior bleeding or an intranasal tumor. Both flexible and rigid endoscopes can be used. The benefit of a flexible nasopharyngoscope is that it allows visualization of the hypopharynx. A rigid endoscope can allow visualization of posterior bleeding and aid attempts at cautery. It is important to use a topical decongestant/anesthetic combination prior to endoscopy. Aggressive suctioning may be required to achieve adequate visualization.

B. Anterior epistaxis is most commonly associated with bleeding from Little’s area on the anterior septum. It usually accounts for 95% of all episodes of epistaxis. In this location, multiple blood vessels come together to form an arterial plexus called Kesselbach’s plexus. Despite often looking impressive, epistaxis from the anterior nose is rarely life threatening. In fact, it has been suggested that topical decongestant and pressure alone are adequate to control bleeding. The most common causes of anterior epistaxis are digital trauma and sensitivity to cold and dry environment. Patients with hereditary hemorrhagic telangiectasia often present with refractory anterior epistaxis. These patients commonly require treatment in the operating room for control of bleeding.

C. Posterior epistaxis is often a more complicated diagnosis. Commonly it involves branches of the maxillary artery, but may involve the internal carotid as well. Patients with sinonasal cancer often present with posterior epistaxis. Rarely, posterior epistaxis can be life threatening, particularly in cases of midfacial trauma. In this situation, the primary goal is to secure the airway. Once this is accomplished attention can be turned to the nose. Because life-threatening epistaxis usually involves the posterior nasal cavity, control of the bleeding can be achieved by placing two Foley catheters into the nasopharynx and inflating the balloons. Often it is difficult to achieve maximal pressure because of midfacial instability. Other considerations include ligation of the external carotid artery under local anesthesia to control bleeding. If the patient is stable, angiography with embolization can be used to achieve hemostasis. It is important to have the otolaryngology team involved early on in these cases. Additionally, because of the tendency for posterior packing to compromise the airway, all patients with posterior packs in place should be admitted for apnea and cardiac monitoring as well as antibiotics.
Epistaxis

A. Introduction
1.) Definition
2.) History
3.) Physical Exam
4.) Nasopharyngoscopy

B. Anterior Epistaxis
1.) Local compression with topical decongestant and anesthetic
2.) May require AgNO3 of site and sugicel
3.) Apply bacitracin bid to affected area, prn topical decongestant

C. Posterior Epistaxis
1.) Control airway
2.) Place foley catheters into nasopharynx and inflate balloons
3.) Unstable patient consider ligation of external carotid
4.) Stable patient consider embolization via angiography

Bleeding Continues
1.) Posterior packing
2.) Control in operating room

Epistaxis
Diabetic ketoacidosis (DKA) is caused by a deficiency in insulin combined with an increase in the counterregulatory hormones such as glucagon, catecholamines, cortisol, and growth hormone. These hormonal alterations lead to a state of severe volume depletion, acidemia, ketonemia, and electrolyte abnormalities that are fatal in up to 5% of patients. About 10% of diabetics will make their initial presentation while in DKA. It is most commonly associated with Type 1 (insulin-dependent) diabetes but may develop in Type 2 diabetes as well. Although poor compliance is a common cause of DKA, in most cases severe stress, such as sepsis, trauma, surgery, drugs, pregnancy, or myocardial infarction, is the initiating factor.

A. Signs and Symptoms. Initial symptoms usually consist of polyuria, polydipsia, nausea, vomiting, fatigue, and, occasionally, vaguely localized abdominal pain. Dehydration is constant as a persistent osmotic diuresis leads to volume depletion. Classic signs of Kussmaul’s respirations (rapid, shallow breathing indicating a respiratory compensation for worsening metabolic acidosis) and a fruity odor to breath may or may not be present. With worsening fluid and electrolyte depletion, tachycardia and hypotension will be evident and in more severe states of DKA, respiratory distress, shock, and even coma can occur.

B. Laboratory Confirmation. Initial laboratory evaluation should include serum electrolytes, a complete blood count with differential, urinalysis, and an arterial blood gas. If abdominal pain is present, it is prudent to include serum liver function tests and serum amylase and lipase. Essential to the diagnosis of DKA is the presence of hyperglycemia (> 250 mg/dl), metabolic acidosis (blood pH < 7.3), a low serum bicarbonate (< 15 mEq/L), ketonemia, and ketonuria. Despite total body potassium depletion from persistent polyuria or vomiting, serum potassium is often elevated on presentation secondary to acidosis. Similarly, total body sodium, magnesium, phosphorus, and chloride may be depleted. To identify precipitating factors, one should also obtain blood cultures, a chest x-ray, and an EKG.

C. Treatment. Effective treatment for patients with DKA requires both aggressive metabolic management and treatment of the underlying cause. Specifically, this includes improving tissue perfusion, decreasing serum glucose and osmolality, and correcting electrolyte abnormalities. It is important to start a therapeutic flow sheet that will allow one to assess chronologically how each therapeutic intervention affects metabolic indices like serum glucose, electrolytes, serum osmolality, bicarbonate, base deficit, and anion gap. In DKA, the anion gap will be elevated as ketoacids accumulate in the serum. The serum osmolality will be elevated secondary to hyperglycemia and if the value is over 320, the patient will often be comatose:

\[
\text{Serum osmolality} = 2(Na+K) + \frac{\text{Glucose}}{18} \\
\text{normal: 280–300 mOsm/L}
\]

\[
\text{Anion gap} = (Na)-(Cl+HCO_3) \text{ (normal: 8–12 mEq/L)}
\]

The cornerstone of fluid management in patients with DKA is repletion of the extracellular fluid volume. By restoring intravascular and extravascular fluid volume, the concentration of the serum glucose and the counterregulatory hormones will fall. Two to three liters of normal saline over the first 1–3 h (5–10 ml/kg/h) is recommended. One may substitute 0.45 NS initially for normal saline if the serum sodium is greater than 150. Following the administration of normal saline, 0.45 NS is given at a rate of 150–300 ml/h. Once serum glucose reaches 250 mg/dl, glucose should be added to the IV fluid to prevent iatrogenic hypoglycemia, provided insulin infusion is still required to treat the acidosis.

Once the diagnosis of DKA has been established, regular insulin should immediately be administered. Insulin replacement not only treats the hyperglycemia (and hyperosmolality), it more importantly corrects the ketoacidosis. This is accomplished by decreasing fatty acid delivery to the liver, decreasing ketone body production, and enhancing the clearance of ketone bodies from the blood. Standard regular insulin therapy consists of an initial bolus of 0.1 unit/kg followed by a continuous IV infusion of 0.1 unit/kg/h until the blood glucose level is ~250 mg/dl. The insulin bolus effectively primes the
insulin receptors and thus increases the therapeutic potential of the continuous insulin infusion. Increasing the insulin infusion rate beyond 0.1 unit/kg/h is usually not helpful as there is a limit to glucose uptake by peripheral tissues. If serum glucose levels do not fall by 50–70 mg/dl in the first hour, by 30% in 4 h, or by 50% in 8 h, the infusion rate should be doubled or an additional bolus of 10 units should be administered. This is repeated hourly until these parameters are fulfilled. The continuous infusion rate may be decreased to 0.05 units/kg/h (or roughly 1–4 units/h) once the serum glucose is less than or equal to 250 mg/dl. Control of ketoacidosis requires the achievement of at least two of three parameters: a serum bicarbonate concentration greater than 18 mEq/L, a venous pH of 7.3 or greater, and an anion gap less than 14 mEq/L. Treatment should not be discontinued until two of these parameters are met. It is important to have an initial serum potassium concentration before administering insulin as fatal arrhythmias may be induced in the rare patient who has hypokalemia on presentation.

Since estimated serum potassium deficits in DKA range from 3 to 5 mEq/kg, potassium replacement should be initiated as soon as an adequate urine output and a normal serum potassium are documented. With serum levels less than 5.5 mEq/L, the addition of 20–30 mEq/L to the intravenous fluids is appropriate. With levels less than 3.5 mEq/L or if bicarbonate is given, 40–80 mEq/h is recommended. As in the case of renal insufficiency, serum potassium repletion should be held if the serum levels do not fall below 5.0 mEq/L. Although phosphate replacement is seldom required in the treatment of DKA, levels less than 1 mg/dl should be replaced in the form of a potassium salt. Replacement must be done gradually, as rapid correction of serum phosphate may precipitate serum calcium into the tissues and lead to tetany.

Bicarbonate therapy is controversial and seldom necessary for correction of acidosis as insulin administration decreases the production of ketone bodies and promotes the regeneration of bicarbonate. Loose indications may include a pH less than 7.1 and serum bicarbonate below 10 mEq/L or to reduce the discomfort of Kussmaul respirations. Slow infusion of 1 amp over a 1-h period is the preferred method of administration.

Cerebral edema is the most well-documented complication of DKA therapy and has been associated with a mortality rate of up to 70%. Rapid correction of hyperglycemia and rapid rehydration markedly increase the risk for cerebral edema. It is most common with children, occurring in 1% of all children with DKA. The presence of headaches or mental status changes should alert the treating physician for this possible complication.
Diabetic Ketoacidosis

A. Signs and Symptoms
- polyuria, polydipsia,
- nausea, vomiting, fatigue,
- abdominal pain,
- dehydration, tachycardia,
- hypotension, coma

B. Laboratory Confirmation
- glucose > 250 mg/dl
- pH < 7.3
- bicarbonate < 15 mEq/l
- ketonemia
- ketonuria

C. Treatment

- determine and treat underlying condition
- monitor:
  - urine output (insert Foley catheter)
  - serum glucose hourly
  - serum electrolytes
  - serum osmolality
  - serum bicarbonate, base deficit
  - anion gap
- insulin replacement:
  - 0.1 units/kg bolus
  - 0.1 units/kg/hr continuous infusion until glucose reaches 250 mg/dl
  - 0.05 units/kg/hr when glucose < 250 mg/dl
  - double infusion rate if glucose levels do not fall 50-70 mg/dl in 1st hour or 30% in 4 hours or 50% in 8 hours
- hydration:
  - 0.9N NaCl or 0.45 NaCl if sodium > 150
  - add glucose when serum glucose reaches 250 mg/dl
- potassium replacement:
  - 20-30 mEq/hr when serum level < 5.5 mEq/l
  - 40-80 mEq/hr when < 3.5 mEq/l
- replace other electrolytes as needed
- bicarbonate replacement
A. Definition. Acute renal failure (ARF) occurs in ~5% of hospitalized patients. Oliguria is defined as urine output less than 400 cc over 24 h. The functional derangement associated with acute oliguria is a sudden decrease in the glomerular filtration rate (GFR), leading to increased serum urea and creatinine, sodium and water retention, hyperkalemia, and acidosis. The initial approach toward oliguria is to verify its presence by checking the most recently recorded urine outputs. Oliguria and impending ARF have ~50% mortality rate in surgical patients most commonly from sepsis and multiorgan failure. Oliguria is classified into prerenal, intrinsic, and postrenal causes. Prerenal and postrenal etiologies must be excluded first because they are often reversible if treated in a timely manner. Initial management entails a detailed history, physical exam, and blood and urine studies, which should be sent prior to instituting therapy.

B. Prerenal Causes. Prerenal ARF is characterized by diminished renal blood flow and accounts for 60–70% of renal failure cases. Prerenal renal failure often leads to intrinsic renal failure because of ischemia-induced acute tubular necrosis (ATN). Assessment of volume status, hemodynamics, and medications may reveal potential prerenal causes of oliguria. Laboratory findings demonstrate a fractional excretion of sodium (FENa) less than 1%, urine osmolality greater than 500 mOsm, and urinary sodium less than 20 mmol/L. These values are consistent with functional kidneys attempting to conserve intravascular volume through reabsorption of sodium and water.

Systemic circulatory impairment is the most common form of prerenal ARF. It is usually due to decreased renal perfusion pressure. An evaluation of the patient’s volume and cardiac status is extremely important. Patients may have depleted their intravascular volume, as with hemorrhage, gastrointestinal fluid loss (emesis, diarrhea, or nasogastric suction), sequestration of fluid (pancreatitis, bowel obstruction, or ascites), renal losses (diuretics or glycosuria), or insensible losses (burns or fevers). These patients have an inadequate preload volume and decreased cardiac output (CO). Signs of hypovolemia include sinus tachycardia, orthostatic and supine hypotension, mucocutaneous changes (decreased skin turgor and delayed capillary refill), and mental status changes (dizziness and decreased arousability). The underlying cause must be treated and the volume status returned to normal. Isotonic fluid replacement should be given as a fluid bolus; volumes and rates vary depending on the concern for causing, or worsening, pulmonary edema and hypoxia. Volume status can be monitored by following vital signs for resolution of tachycardia, orthostatics, hypotension, along with central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP).

Decreased renal perfusion may also be secondary to myocardial failure from myocardial infarction, pulmonary embolus, congestive heart failure (CHF), or cardiac tamponade. Patients may complain of chest pain and shortness of breath and physical findings may include an irregular pulse, a new murmur, jugular venous distension, rales, an S₃, and wheezing. Treatment is aimed at the underlying cause and to improve the patients’ CO and overall hemodynamic status. Inotropes may be necessary to increase the patients’ CO after the preload volume has been restored.

Decreased renal perfusion pressure in the setting of an adequate CO may be due to macrovascular causes such as renal artery or vein occlusion from thrombosis, thromboembolism, severe arterial stenosis from atherosclerosis, or a dissecting aneurysm. Depending on the cause and the timing, surgery may be considered as a treatment option.

Certain medications such as angiotensin-converting enzyme (ACE) inhibitors and nonsteroidal anti-inflammatory drugs (NSAIDs) cause prerenal oliguria through the inhibition of prostaglandin synthesis. These drugs disrupt renal autoregulation of blood flow by efferent arteriolar dilatation (ACE inhibitors) or by afferent arteriolar vasoconstriction (NSAIDs). Treatment entails identification and removal of the offending drugs.

C. Renal Causes. Renal or intrinsic ARF occurs when there is intrinsic damage to the renal parenchyma and accounts for
25–40% of the causes of oliguria. Laboratory studies typically reveal a FENa greater than 3%, urine osmolality between 250 and 300 mOsm, and urine sodium greater than 40 mmol/L. These values occur because the kidney has lost its ability to concentrate urine and the inability to maximally reabsorb sodium and water. Intrinsic ARF is divided into four categories, which include tubular, interstitial, glomerular, and vascular.

D. Tubular Causes. ATN accounts for ~85% of the intrinsic causes of ARF and is usually due to ischemia (50%) and toxins (35%). Ischemia is typically an extension of prerenal ARF, except that the GFR does not improve with restoration of blood flow. Toxins cause ATN by direct nephrotoxicity and other mechanisms. Antibiotics (aminoglycosides and vancomycin) and radiocontrast dye are the most common causes, but chemotherapeutic agents (cisplatin), amphotericin, cyclosporine, heme pigments (myoglobin and hemoglobin), solvents (ethylene glycol), and myeloma light chains also cause ATN. Treatment is mainly supportive and includes reversal of ischemia, removal of toxins, and maintaining euvolemia. Patients at risk for ATN include those with diabetes, CHF, or chronic renal insufficiency. Prevention of ATN entails adequate hydration, maintaining CO, and prevention of vasoconstriction.

E. Interstitial Causes. Acute interstitial nephritis usually presents with ARF, fever, rash, eosinophilia, and occasionally eosinophiluria. This usually occurs because of an allergic reaction to a drug, an autoimmune disease, an infection, or an infiltrative disease. Glomerulonephritis can present with hypertension, proteinuria, and/or hematuria; evaluation consists of various serologic tests. Microvascular disease of the kidneys may present with hypertension, hemolytic anemia, and thrombocytopenia. This occurs because of glomerular capillary thrombosis or occlusion. Treatment entails eliminating the possible offending agent, diagnostic biopsy, immunosuppression with corticosteroids, and plasmapheresis depending on the etiology.

F. Postrenal Causes. Postrenal ARF or obstructive uropathy occurs in 5–10% of the cases. This occurs when the urinary outflow tracts of both kidneys are obstructed or when the tract of a single functional kidney is blocked. It is important to rule out obstructive uropathy quickly because recovery is inversely related to the duration of the obstruction. These patients typically present with relative anuria and possibly with suprapubic distention and pain or overflow incontinence. Possible etiologies include benign prostatic hypertrophy, prostate cancer, cervical cancer, retroperitoneal fibrosis, intratubular obstruction, pelvic mass or malignancy, intraluminal bladder mass, neurogenic bladder, and urethral strictures. Bladder catheterization may be diagnostic and therapeutic for these patients. Patients with urinary drainage systems in place should have the catheters inspected for any kinks and irrigated gently to confirm patency. Renal ultrasound should be done to detect hydronephrosis and therapy should possibly include ureteral stenting or percutaneous nephrostomy.

Initial treatment for ARF should focus on reversing the underlying cause and correcting fluid and electrolyte imbalances. Focus should be placed on postrenal and pre-renal etiologies first. Volume status should be ascertained quickly. Hypovolemic patients should be given isotonic intravenous fluids, while hypervolemic patients should undergo diuresis. Hypervolemia presents with jugular venous distention, an S4 gallop, pulmonary rales, and/or peripheral edema. Often cardiac filling pressures need to be measured to assess the patient’s true volume status. Once preload (CVP/PCWP of 18–20 mmHg) and CO (4–8 L/min) have been determined to be adequate, diuretics and dopamine should be given. Loop diuretics should be given in large doses and dopamine should be started at a renal dose (3–5 mg/kg/min). These medications are given to prevent renal vasoconstriction, to increase intratubular flow, and to convert a patient from oliguric renal failure to nonoliguric renal failure, which is easier to manage from a fluid perspective. If these drugs do not increase urine output they should be discontinued. Furthermore, despite the prevalent use of renal dose dopamine for ARF, there is no literature to support its use and no evidence to suggest it speeds recovery of or improves survival after ARF. Hyperkalemia, acidosis, hypervolemia, and uremia are metabolic derangements seen with ARF and may need to be treated with dialysis depending on severity. A nephrology consultation is essential for the supportive treatment of ARF and in the decision of instituting dialysis.
Oliguria

A. Definition
urine output < 400 cc over 24 hrs

B. Prerenal Causes
FENa < 1%
urine osmolality > 500 mOsm
urine Na < 20 mmol/L

- hypovolemia:
  - bleeding, GI losses, diuretics, ascites
- cardiogenic:
  - infarction, failure, tamponade, embolus
- vascular:
  - renal artery occlusion

  treatment is aimed at restoring normal volume status, electrolyte levels and perfusion

C. Renal Causes
FENa > 3%
urine osmolality 250-300 mOsm
urine Na > 40 mmol/L

- Tubular Causes
  - ischemia
  - antibiotics, X-ray dye, chemotherapy, solvents

D. Interstitial Causes
  - drug allergy, autoimmune, infection, infiltrative disease

  eliminate offending agent

E. Postrenal Causes
obstruction

- prostatic hypertrophy
- prostate cancer
- neurogenic bladder
- urethral strictures etc.

  treatment is aimed at relieving obstruction
Postoperative Fever

Melissa K. Johnson

A. Differential Diagnosis. Postoperative fever is defined as a temperature elevation greater than 101 °F. Fever is common in the postoperative period, occurring in up to 50% of patients; however, an infectious source is identified in less than half of febrile patients. The release of pyrogenic cytokines in response to the trauma of surgery may elicit a febrile response in the absence of infection. Early postoperative fevers (within 48h) are usually due to the inflammatory response to surgery and are not associated with infections. In contrast, fevers due to infection tend to occur later in the postoperative period. Risk factors for infectious causes include the presence of cancer, an immunosuppressed state, and anything that interrupts normal host defense mechanisms. These fevers tend to be higher (>101 °F).

A discussion of postoperative fever must take into account the time period relative to the day of surgery. A differential diagnosis can then be formulated. The classic “Ws” of postoperative fever are as follows: Wind: atelectasis on POD 1–2; Water: urinary tract infection on POD 2–3; Wound: wound infection on POD 3–7; Walking: deep venous thrombosis (DVT)/thrombophlebitis on POD 5–7; and Wonder Drug: drug fever on POD >7.

B. Intraoperative Fever. Fever during the intraoperative period may be due to endocrine disturbances (Addisonian crisis, thyroid storm, or pheochromocytoma), transfusion reaction, drug hypersensitivity, preexisting infection, intraoperative manipulation of purulent material, or malignant hyperthermia. The incidence of fever in the intraoperative period is generally lower than during other periods, potentially due to the inhibition of the normal febrile response to pyrogens by general anesthesia.

C. Less than 24 Hours. Fevers in the early postoperative period (<24h) are most often due to the normal inflammatory response to surgery. Additional causes include atelectasis or necrotizing wound infections. In the presence of a high fever, a bedside assessment of the wounds is mandatory, inspecting for ominous changes such as discoloration, intense erythema, crepitation (soft tissue air), and brownish drainage. Necrotizing wound infections are rare, but may be caused by beta-hemolytic Streptococci or Clostridia. These wounds are especially painful. Treatment consists of emergent operative debridement and prompt administration of antibiotics, usually penicillin and/or clindamycin. Laboratory and x-ray workup of fevers in the immediate postoperative period is typically not indicated unless a patient is symptomatic. Treatment of fever in a noninfected patient should focus on returning the thermoregulatory set-point to normal with antipyretics.

D. Postoperative Days 1–2. Fevers on postoperative days 1–2 (24–48h) are usually due to atelectasis induced by general anesthesia and decreased diaphragmatic excursion from postoperative pain. Atelectasis is manifested by tachypnea, tachycardia, and decreased breath sounds on auscultation. X-rays are not needed to diagnose atelectasis unless fevers persist for 2–3 days in which case pneumonia should be suspected. Incentive spirometry, coughing, deep breathing, chest physiotherapy, and adequate pain control all help to prevent and treat atelectasis. Additional causes of fever in this time period include wound infection, thrombophlebitis, and central venous line sepsis. Intravenous (IV) sites and venous catheters should be inspected and changed if indicated. The wound should be inspected for any interval changes in color or drainage. Asymptomatic and low-risk individuals may be observed and treated with antipyretics. For high-risk patients, urine, blood, and sputum cultures should be obtained along with a chest x-ray.

E. Postoperative Days 3–7. Fevers on postoperative days 3–7 are more likely to be due to infectious causes than fevers occurring at other times. Risk factors which predispose patients to postoperative infections include diabetes, immune suppression, obesity, catheterization (urinary or central venous), and prolonged postoperative ventilation. The most common cause of postoperative infection is wound infection. Tenderness, erythema, drainage, and inflammation at the surgical wound are all indicators of possible wound infection. Urinary tract infections are more common in patients with indwelling urinary catheters. A urinalysis demonstrating pyuria and a
urine culture growing >100,000 colonies of bacteria indicate a urinary tract infection (UTI). Prolonged ventilation or persistent atelectasis predisposes patients to pneumonia; a chest x-ray and sputum culture should be performed if the patient’s physical exam supports this diagnosis.

Other causes of fever in the later postoperative period include central line sepsis, deep venous thrombosis/pulmonary embolism (DVT/PE), intra-abdominal abscess, anastomotic leak, *Clostridium difficile* colitis, and parotitis. Central lines should be inspected and changed if indicated. One should examine the extremities for pain and swelling, and obtain venous Doppler studies if DVT is suspected. Pulmonary embolism may be diagnosed with either nuclear medicine ventilation/perfusion scans or spiral CT scans of the chest. Imaging studies (ultrasound or CT) may be needed to rule out abscess or anastomotic leak. In the absence of identifiable infections, drug fevers and alcohol withdrawal should be considered. Antibiotics are the most common cause of drug fevers.

Overall, the workup of fevers in the postoperative patient should be guided by a thorough history and physical exam. In asymptomatic low-risk patients, further workup is usually of low yield and adds significant cost. Lab and x-ray studies should be ordered based on signs and symptoms, particularly for those patients in high-risk categories.
**Postoperative Fever**

### A. Differential Diagnosis
- Temperature greater than 101 degrees
- "wind, water, wound, walking, wonder drugs"

#### B. Intra-operative Fever
- Malignant hyperthermia
- Transfusion reaction
- Drug hypersensitivity
- Endocrine, e.g., Addisonian crisis
- Pre-existing infection
- Manipulation of septic focus

#### C. < 24 hours
- Atelectasis
- Necrotizing wound infection
- Bedside inspection of wound is mandatory

#### D. Post-operative Day 1-2
- Atelectasis
- Pneumonia
- Thrombophlebitis
- Central line sepsis

#### E. Post-operative Day 3-7
- Wound infection
- Urinary tract infection
- Pneumonia
- Central line sepsis
- DVT/PE
- Abscess
- Anastomotic leak
- *C. Difficile* colitis
- Parotitis
- Drug fever
- EtOH withdrawal

Febrile patients at high risk of infection are those with cancer, immune suppression, and those where either the gastrointestinal or genitourinary tract were entered during surgery. Under these circumstances, laboratory and radiographic tests are indicated in the assessment of the patient.
A. Differential Diagnosis. Hypercalcemia is rare in hospitalized patients, occurring at a reported rate of less than 1% of patients in this country. Hypercalcemia is the result of excessive skeletal calcium release, increased intestinal calcium absorption, or inadequate excretion of calcium. The two most common conditions that account for 90% of patients with hypercalcemia are primary hyperparathyroidism and cancer. Malignancies such as multiple myeloma, lymphoma, and metastatic breast cancer can invade bone and cause direct bone reabsorption leading to hypercalcemia. Other malignancies can release parathyroid hormone-like substances that cause hypercalcemia. Rarer causes of hypercalcemia include use of thiazide diuretics, lithium therapy, excessive vitamin D intake, immobilization, hyperthyroidism, and complicated renal failure.

B. Signs and Symptoms. Hypercalcemic crisis occurs when serum calcium levels rise above 14 mg/dl or serum-ionized calcium above 3.5 mmol/L. Serum calcium levels can be falsely elevated when the serum albumin is high; however, this will not affect the ionized (physiologic active) calcium measurement. Symptoms of hypercalcemia include nausea, vomiting, constipation, polyuria, muscle fatigue, weakness, and mental status depression that can lead to coma. Patients can also have pancreatitis, nephro lithiasis, and/or cardiac findings, specifically, a shortened QT interval on EKG. These symptoms can be present with serum calcium levels greater than 12 mg/dl (ionized calcium 3 mmol/L). However, they become life threatening at the hypercalcemic crisis levels and require prompt therapy.

C. Treatment. Regardless of the etiology of a hypercalcemic crisis, the first treatment is rehydration. Hypercalcemia causes an osmotic diuresis that leads to hypovolemia, elevating serum calcium levels even higher. Therapy begins with intravenous (IV) hydration using normal saline, with an initial bolus adequate to rehydrate and then a continuous rate of 200–300 cc/h. Patients should be placed on a cardiac monitor and moved to an ICU setting if necessary for close monitoring of calcium levels. Once the patient is hydrated, diuresis with furosemide should begin with 40–80 mg IV every 2–8 h, to achieve an hourly urine output of 100–200 cc. This urine output requires IV fluid replacement to avoid hypovolemia that would again exacerbate the hypercalcemia. Hydration and diuresis will correct the hypercalcemia in the acute setting. The underlying etiology of the hypercalcemia must still be found.

Surgical intervention is indicated for hyperparathyroidism whether caused by a solitary or multiple adenoma(s) of the glands. There is debate whether asymptomatic hypercalcemia caused by a parathyroid adenoma is an indication for surgery. Surgical intervention is indicated for hyperparathyroidism caused by hereditary multiple endocrine neoplasia (MENI) and MENIIa, even if the patient is asymptomatic.

Hypercalcemia caused by malignant invasion of bone requires further therapy to avoid future crises. This therapy includes calcitonin and bisphosphonates. Calcitonin, 4 U/kg IM/SQ q12H, will work immediately but only decreases the calcium, 0.5 mmol/L, in the short term. Pamidronate is a bisphosphonate that will decrease bone resorption with a dose of 90 mg continuous IV over 24 h. The peak effect is seen in 4–5 days and another dose can be given at that time.

Other causes of hypercalcemia are addressed according to the mechanism involved or with discontinuation of the aggravating medication. However, these rare causes of hypercalcemia even more rarely cause a hypercalcemic crisis.
Hypercalcemic Crisis

A. Differential Diagnosis
   primary hyperparathyroidism
   cancer
   thiazide diuretics, lithium therapy,
   vitamin D excess, renal failure

B. Signs and Symptoms
   nausea, vomiting, constipation,
   polyuria, fatigue, weakness,
   depression, mental status changes,
   pancreatitis, renal stones

C. Treatment
   intravenous saline hydration
   furosemide diuresis
   for cancer, administer
   calcitonin 4U/kg IM q 12h
   and biphosphonates
   (pamidronate 90 mg IV q 24h)
   for other causes of hypercalcemia, treat
   underlying process
   parathyroidectomy for hyperparathyroidism
Postoperative Chest Pain

Alberto Aviles

A. **Origin of Chest Pain.** Chest pain may be caused by a variety of problems involving the many anatomical structures in the thoracic cavity. Deep retrosternal or precordial pain originates from the roots of T1–T4. Posterior nerve connections in the sympathetic chain communicate with T5–T6 which innervate the diaphragm and peritoneal surfaces of the upper abdomen. Therefore, the six dermatome band (T1–T6) receives impulses from the thoracic visera (heart, aorta, pulmonary structures, esophagus, and mediastinum) as well as the diaphragm and upper abdominal organs (gallbladder, pancreas, stomach, and duodenum). The way in which chest pain presents in terms of character and duration can aid in choosing between cardiac, pulmonary, vascular, or gastrointestinal sources.

B. **Pain History.** In the postoperative period, it is essential that the clinician formulate a comprehensive picture of the patient which should include knowledge of the procedure performed, when it occurred, and a brief history of the patient’s preoperative condition. Such information, in conjunction with associated symptoms, can help establish whether the origin of pain is cardiac, pulmonary, vascular, or gastrointestinal. The history can identify cardiovascular risk factors such as hypertension, hypercholesterolemia, or diabetes. Chronic obstructive pulmonary disease and a history of gastroesophageal reflux disease (GERD), peptic ulcer disease (PUD), or cholelithiasis are pulmonary and gastrointestinal factors that may predispose to pain in the postoperative setting. Regarding when the chest pain occurs in relation to the surgery, myocardial infarctions (MIs) typically occur within the early postoperative period, while infectious and thromboembolic events occur later.

C. **Initial Evaluation.** Vital signs are measured and the patient’s stability assessed. Proper monitoring devices such as a pulse oximeter, telemetry, and blood pressure cuff are placed. A physical examination focusing on the neck, heart, lungs, abdomen, and peripheral pulses should be performed. The physical exam should also assess for jugular venous distension, presence of breath and adventitious sounds, new murmurs or gallops, localizing abdominal tenderness, and decreased distal pulses. Other findings may include friction rubs, deviated or pulsating trachea, subcutaneous emphysema, and Hamman’s sign. The latter two findings are suggestive of esophageal perforation. Initial tests should include ECG and cardiac enzymes to evaluate the heart; ABG and CXR to assess pulmonary status; CBC, liver function tests, and amylase and lipase for possible intra-abdominal problems.

D. **Cardiac Causes.** Symptomatic ischemic heart disease may present either as angina pectoris or as MI. The recently ambulating surgical patient who describes the pain as deep, steady, and lasting less than 20 min may be experiencing an anginal episode. Sublingual nitroglycerin (0.4 mg every 5 min) will relieve the ischemic pain of angina but not the pain of a MI. Deep and steady pain lasting more than 20 min is consistent with an MI and the patient should be given oxygen, morphine, and aspirin. Nitroglycerin may be continued either in sublingual form or intravenously depending on the severity of the pain and the response to treatment. Beta-antagonists are given unless there are contraindications to doing so (heart failure, bradycardia, heart block, and COPD). Chest pain occurring in combination with typical ECG findings (elevated ST segments, inverted T waves, and Q waves) or positive cardiac enzymes (CPK-MB, Troponin I, and myoglobin) justify a diagnosis of MI. Both CPK-MB and myoglobin are used to identify an early MI as they become abnormal within 3–6 h postinjury. Myoglobin disappears from the blood within 12 h; however, CPK-MB peaks at 24 h. Tissue damage in the postoperative patient may falsely elevate these two markers, whereas an elevated Troponin I (abnormal 8 h postinjury, peaks at 24 h) of >0.4 ng/ml is diagnostic of an MI. Three troponin-I levels obtained 12 h apart are highly sensitive and specific for detecting myocardial injury. The clinician should keep in mind that in the postoperative period, an MI infrequently occurs after the 3rd day, chest pain is present in only 30%, and thrombolytics are not indicated if an early MI is encountered (<6h). Angioplasty is the treatment of choice for the patient who experiences a postoperative MI.

E. **Pulmonary Causes.** Chest pain associated with pulmonary structures can differ from cardiac ischemia by location and...
associated symptoms. Cardiac pain is often central while inflammation of the parietal or diaphragmatic pleuras may present laterally. Pain exacerbated with coughing or breathing is also consistent with a pulmonary etiology. The pulmonary diseases to consider in the postoperative period are pneumothorax, pulmonary embolus, pneumonia, and pleurisy. Pneumothoraces may occur after upper abdominal surgery or laparoscopy and will often present in the immediate postoperative period. In contrast, a spontaneous pneumothorax can occur at any time. More than 70% of secondary spontaneous pneumothoraces occur in patients with COPD.

Pneumonia and pulmonary embolus typically occur several days into the postsurgical period. The former occurs in 5–20% of postoperative patients; the latter fortunately occurs in only 1%. Atelectasis can progress to pneumonia. Risk factors for pneumonia include previous lung pathology, age greater than 65 years, or more than 1,200 cc operative blood loss.

Risk factors for pulmonary embolus and deep venous thrombosis (DVT) include a prior history of DVT, obesity, age > 40, prolonged periods of immobilization, cancer, and long operative times. Pulmonary embolism (PE) may present with chest pain, hemoptysis, tachycardia, fever, or dyspnea. An arterial blood gas usually reveals hypercarbia with variable hypoxemia while an ECG reveals right axis deviation or atrial fibrillation. Further diagnostic tests include ventilation perfusion scans and spiral CT to confirm a perfusion defect or presence of a clot, respectively. Treatment involves anticoagulation (heparin, then coumadin) if the patient is greater than 24 h postsurgery; otherwise an inferior vena cava filter should be considered. Thrombolytic therapy is not indicated for the treatment of PE in the surgical patient because of the risk of bleeding. Pulmonary embolectomy (transvenous or open) is rarely indicated, but can be lifesaving for the patient in extremis.

F. Gastrointestinal Causes. Gastrointestinal and vascular etiologies should not be excluded as several abdominal organs are in close proximity to the diaphragm. The stomach and duodenum are potential sources of referred pain to the chest if inflammation or ulceration is present. The clinician should enquire whether there is a history of PUD or symptoms suggestive of this problem. Prompt removal of nasogastric tubes simultaneous with adequate antiacid therapy may reduce injury to these organs. The onset of oral intake, or lack thereof, introduces other considerations during the postoperative period. Absence of gut stimulation predisposes to biliary stasis that can progress to acalculous cholecystitis. Conversely, starting oral intake prematurely may cause emesis. Boerhaave’s syndrome can occur if the vomiting episodes are violent and this must be treated aggressively to minimize the risk of mediastinitis. CXR will typically reveal a left-sided pleural effusion and air in the mediastinum.

G. Vascular Disorders. Sudden, sharp pain that radiates to the back might indicate a ruptured or dissecting aneurysm. Distal pulses must be checked to evaluate the extent of dissection while vital signs are monitored carefully. Spiral CT or angiography are diagnostic and treatment is dictated by aneurysm location. Type A aneurysms involve the ascending aorta and are treated immediately with surgery to prevent rupture or pericardial tamponade. Type B aneurysms are those that do not involve the ascending aorta and can be treated medically using antihypertensive medication.

H. Procedure-Related Factors. Complications from surgery in the upper abdomen may produce referred pain; therefore, the clinician should be familiar with certain predisposing procedures. Residual gas retention from laparoscopy often creates pain within the first 24 h by distension and irritation of the diaphragm. Referred pain originating from the diaphragm usually presents in the scapular region. Contents from a gastric or biliary anastomotic leak can irritate the peritoneal surfaces of the upper abdomen after gastric, esophageal, or gallbladder surgery. Biliary leaks occur in 1–2.5% after laparoscopic cholecystectomy and can be diagnosed with a hydroxy iminodiacetic acid (HIDA) scan. Patients with a postcholecystectomy bile leak and those who had surgery in the upper abdomen (splenectomy or gastrectomy) are at risk for developing a subphrenic abscess or pancreatitis. Other procedures that may predispose to pancreatitis are endoscopic retrograde cholangiopancreatography (ERCP) cholecystectomy, vascular procedures that require extensive retroperitoneal dissections, and, rarely, parathyroidectomy.
Postoperative Chest Pain

A. Origins of Chest Pain

B. Pain History
duration, location, associated symptoms, comorbid conditions

C. Initial Evaluation
CXR, EKG, ABG, labs, bedside physical exam

D. Cardiac
angina
lasts < 20 minutes
nitroglycerin
myocardial infarction
lasts > 20 minutes
EKG, enzymes
only 30% have pain
rare after day 3

E. Pulmonary
pneumothorax
pneumonia
embolism
pleurisy

F. GI
esophagitis/perforation
gastritis/peptic ulcer
cholecystitis
pancreatitis

G. Vascular
ruptured aneurysm
dissecting aneurysm

H. Procedure Related
laparoscopy
gastric surgery
hepatobiliary
splenectomy
vascular

CO₂ retention
leaks
abscess
pancreatitis
A. The heart is influenced by multiple factors which affect its rate and rhythm, including neural, hormonal, metabolic, and drug effects. The major concerns when faced with an abnormal heart rate and rhythm are perfusion of vital organs and identifying and treating the arrhythmia. The first priority is to assess and ensure patient stability. Loss of consciousness requires prompt activation of advanced cardiac life support (ACLS) protocol. Signs and symptoms of shock, chest pain, palpitations, dyspnea, and syncope require thorough assessment. When a cardiovascular emergency is occurring or when a situation is evolving into one, it is essential to have adequate airway management and supplemental oxygen, adequate vascular access, ECG with rhythm strip, cardiac monitoring, and continuous pulse oximetry.

Evaluation and treatment begin with a detailed assessment of the patient. Relevant past history includes underlying cardiac disease (history of infarction, Wolff-Parkinson-White syndrome, angina, arrhythmia, and congestive heart failure), risk factors (diabetes, tobacco use, hypertension, and elevated cholesterol), prior cardiac surgery, home medications, and use of addictive substances (alcohol, benzodiazepines, and narcotics). On physical examination, one should have a current set of vital signs and focus on signs of cardiopulmonary dysfunction. Evaluating all wounds, drain and urine output, and analgesic adequacy are also essential. Useful studies include the following: ECG, arterial blood gas, hemoglobin, glucose, serum electrolytes, chest radiograph, cardiac enzymes, and drug levels in the blood. Always compare the current ECG and laboratory values with previous studies.

B. Tachycardia (>100 beats/min) is the most common postoperative arrhythmia. Tachycardia decreases diastolic filling and cardiac output causing hypotension and increases myocardial oxygen consumption, which can lead to myocardial ischemia. In evaluating tachycardia with a 12-lead ECG and rhythm strip, one should employ a systematic approach. Examine P-waves for rate, morphology, and relationship to the QRS complexes. Examine the QRS complex morphology and the R–R interval.

C. Sinus Tachycardia. Sinus tachycardia (ST) is an adaptive response and may be an appropriate response in the postoperative setting. The rate is between 100 and 180 bpm. The P-waves appear normal (upright in lead II and down in a ventricular fibrillation, VF) and precede each QRS complex. It is important to carefully evaluate the patient to determine the etiology of ST. Hypercatecholamine states such as anxiety, pain, fever, or ethanol and drug withdrawal may cause ST. To optimize oxygen and glucose delivery during hypoxemia, anemia, or hypoglycemia, ST will maintain delivery to peripheral tissues despite decreased levels. ST occurs in low stroke volume states such as hypovolemia and myocardial dysfunction to maintain cardiac output. Administration of certain medications (inhaled β-agonist, theophylline, and caffeine) may cause ST and withdrawal of others (β-blockers, clonidine, and steroids) may cause a rebound tachycardia. Sepsis, pulmonary embolus, hyperthyroidism, and pheochromocytoma are also potential etiologies.

The treatment for ST is directed at the underlying cause of the tachycardia. In the event of signs and symptoms of myocardial ischemia or infarction, ST requires treatment. In patients without hypovolemia or left ventricular dysfunction, β-blockers should be used and titrated for effect.

D. Narrow complex tachycardias require correct diagnosis for appropriate management. The differential diagnosis is junctional tachycardia, paroxysmal supraventricular tachycardia (PSVT), and ectopic or multifocal atrial tachycardia. If inspection of the ECG does not yield appropriate diagnosis, unilateral carotid sinus massage or other vagal maneuvers should be attempted to identify and possibly terminate the rhythm. Adenosine 6 mg IV push may be given if the rhythm persists. Adenosine causes a transient heart block at the AV node. The effect is a temporary arrest prior to the patient converting to a normal rhythm or the rate slowing to allow rhythm identification.

When the rhythm persists and is identified, the appropriate management is unique to the type of rhythm. Junctional tachycardia should never be cardioverted. With normal heart function, the medications of choice are β-blocker, calcium-channel blocker,
or amiodarone. In the presence of CHF or decreased ejection fraction, amiodarone alone is recommended. The treatment strategy for ectopic or multifactorial atrial tachycardia is almost the same.

In patients with PSVT refractory to vagal maneuvers or adenosine and in patients with normal cardiac function, a β-blocker, calcium-channel blocker, or digoxin should be administered. When the arrhythmia persists, DC cardioversion is appropriate. In patients with decreased cardiac function, DC cardioversion is the priority treatment followed by digoxin, amiodarone, or diltiazem.

E. Atrial flutter is a reentrant tachycardia characterized by a “sawtooth” pattern on ECG. The atrial rate ranges from 250 to 350 beats/min. A physiologic AV block typically occurs. On physical exam, flutter waves may be visible in the jugular venous pulsations. Atrial fibrillation is characterized by its lack of P-waves in the presence of an irregular ventricular rate. Treatment of both requires control of rhythm and rate. To control the rate, digoxin may be used regardless of cardiac function, while a β-blocker or calcium-channel blocker may also be used with normal cardiac function. The antiarhythmic agent amiodarone should also be considered. In the unstable patient or patient with rate greater than 150 bpm, synchronous DC cardioversion beginning at 100 J is the treatment. In the stable patient, DC cardioversion is also recommended if the rhythm persists after the rate is controlled.

F. Ventricular Tachycardia. (VT) There are two forms of ventricular tachycardia (VT): monomorphic and polymorphic. In monomorphic VT, the QRS complexes are uniform and broad. In polymorphic VT, there are multiple QRS morphologies, each beat usually different from the previous one. The R-R interval also changes. A subtype of polymorphic VT is “Torsade de pointes,” which has a variable morphology which transitions in a characteristic repetitive manner.

Regardless of the VT, if the patient is unstable, proceed with cardioversion. Synchronized cardioversion begins at 100 J, then 200, 300, and 360 J. In pulseless VT, begin at 200 J. In critical situations with delay in synchronization, use unsynchronized shocks.

G. Ventricular Fibrillation (VF) is found in patients who are hemodynamically unstable or unresponsive. Its rhythm is totally disorganized and has an undulating and unpredictable baseline. If a patient is in VF, call for the defibrillator immediately. Until it arrives, begin basic cardiopulmonary resuscitation (CPR). Upon arrival, up to three shocks (360 J) may be given before any other therapy is attempted. The pulse and rhythm should be assessed after each shock is delivered. If VF persists, proceed with the secondary survey of the ACLS protocol including airway management and intravenous access. The treatment pattern after the secondary survey is continued CPR and drug-shock-drug-shock. A single dose of vasopressin 40 U IV or epinephrine 1 mg IV push every 3–5 min may be used. One shock at 360 J may be delivered within 30–60 s of drug administration. Antiarrhythmics (amiodarone, lidocaine, magnesium, and procainamide) should be considered if VF continues.

H. Bradycardia (<60 bpm) may be the result of decreased discharge from the sinoatrial node because of cholinergic effects or an inherent block in the normal conduction between the SA node and ventricles.

I. Sinus Bradycardia (SB) is a regular rhythm in which P-waves originate in the SA node and precede narrow QRS complexes. This rhythm is normal in the well-conditioned athlete. It is pathologic in other conditions, including intrinsic nodal disease (sick sinus syndrome), increased vagal tone (inferior wall MI, vasovagal response, and systemic or intracranial hypertension), and drug toxicity (β-blockers, calcium-channel blockers, and adenosine).

In addition to treatment of the underlying condition, treatment for SB is necessary only when it causes hypoperfusion. Symptoms of hypoperfusion are syncope, altered mental status, extreme fatigue, pulmonary edema, and systemic hypotension. In addition to the stabilizing the patient and monitoring, atropine 0.5–1.0 mg IV should be administered until vital signs normalize. The maximum dose is 0.04 mg/kg. If this is unsuccessful, transcutaneous pacing should be used if available. If unavailable, isoproterenol 2–10 µg/min should be titrated to effect until transcutaneous or transvenous pacing is available.

J. In 1st degree AV Block, P-waves are normal and precede every QRS complex, but the PR interval is prolonged and constant. Patients with this rhythm are asymptomatic and only require monitoring for development of other conduction disturbances and development of symptoms.

K. Mobitz Type I (Wenkebach) 2nd degree AV Block results from a progressive decrease in conduction velocity at the AV node. This is manifested by progressive prolongation of the PR interval until a P-wave is completely blocked at the AV node and not followed by a QRS complex. The conduction ratio is repetitive and constant (i.e., 3:2 block implies 3 P-waves and 2 QRS complexes). This rhythm may be the result of an inferior wall MI, drugs that prolong AV node conduction (digoxin, β-blockers, calcium-channel blockers), and increased parasympathetic tone. Treatment and indications are similar to that described for SB, but pacing must take place at the ventricular level (below the AV block).

L. Mobitz Type II 2nd degree AV Block results from structural damage (due to an anterior wall MI, myocarditis, or cardiac surgery) to the ventricular conduction system below the AV node. It is manifested by a relatively constant conduction block (i.e., 2:1, 3:1, etc.) with a constant PR interval and a wide QRS complex. Treatment is always necessary as this rhythm is usually persistent or recurrent and may deteriorate into complete heart block (CHB). Transvenous pacing should be placed as soon as possible. Until it is available, isoproterenol...
or transcutaneous pacing should be used. Atropine should not be used as it may worsen the degree of block.

M. *Complete Heart Block/3rd degree AV Block (CHB)* implies a complete lack of conduction from the atria to the ventricles. The atria and ventricles are controlled by different pacemaker sites entirely. There is no meaningful relationship between P-waves and QRS complexes. CHB demands immediate treatment. Transvenous pacer should be placed as soon as possible. Until available, transcutaneous pacing, atropine, or isoproterenol should be administered to support the patient.
Management of Cardiac Arrhythmias

A. Initial Evaluation
   1.) Verify Arrhythmia
   2.) Ensure Patient Stability
   3.) History; Physical; ECG

B. Tachycardia
   C. Sinus Tachycardia
      Treat precipitating cause
      If no cause found, use β-blocker
   D. Narrow Complex Tachycardia
      Vagal Maneuvers, Adenosine
      Identify and treat rhythm
   E. Atrial Flutter
      Atrial Fibrillation
      DC Cardioversion, Digoxin, β-blocker or Calcium Channel Blocker
      Digoxin, β-blocker or Ca Channel Blocker Possible DC Cardioversion,

F. V. Tach
   Unstable
   DC Cardioversion
   Stable
   Monomorphic
   Procainamide, β-blocker
   Polymorphic
   Correct Electrolytes
   Normal Baseline
   8-blocker idio-caine
   Magnesium Isoproterenol Overdrive Pacing
   Prolonged
   Antiarrhythmics
   Defibrillate
   Secondary Survey
   Epinephrine or Vasopression
   Defibrillate x1 at 360
   Antiarrhythmics

G. V. Fibrillation
   Defibrillate
   Defibrillation
   Atropine, Isoproterenol, Transcutaneous Pacing

H. Bradycardia
   I. Symptomatic Sinus Bradycardia
      Atropine, Isoproterenol, Transcutaneous Pacing
   J. 1° Heart Block
      Observe
   K. 2° Mobitz I Heart Block
      Atropine
   L. 2° Mobitz II Heart Block
      Transcutaneous Pacing Isoproterenol
   M. Complete Heart Block
      Transcutaneous Pacing
Ventilator Management

Mitchell J. Cohen

A. Introduction. Understanding ventilator management begins with understanding the physiology of oxygenation and ventilation and the recognition of respiratory failure and its subsequent requirement for ventilatory support.

Breathing can be most simply broken up into two components: oxygenation and ventilation.

Oxygenation is simply the diffusion of breathed oxygen content into blood oxygen content where it can be delivered to tissues and consumed. Blood oxygen content is directly related to hemoglobin concentration and oxygen saturation with a minor component being supplied by oxygen dissolved in plasma. Useful clinical measures of oxygenation include PaO₂ and O₂ saturation. These are often used interchangeably although one must remember that their relationship is not direct but rather curvilinear as expressed by the oxygen dissociation curve.

Respiratory failure as a result of inadequate oxygenation is defined as an O₂ saturation of less than 90% or a PaO₂ of less than 60. Causes include inadequate systemic oxygenation, ventilation-perfusion mismatching, shunt, and hypoventilation.

Ventilation seeks to eliminate the product of oxygenation. CO₂ is produced as the body burns fuel in the presence of oxygen. Depending on the respiratory quotient of the fuel burned, more or less CO₂ is produced. Measures of ventilation include pH, PaCO₂, and end-tidal CO₂.

Respiratory failure as a result of inadequate ventilation is most often caused by a reduced ventilatory drive. This can be caused by neurologic impairment secondary to organic or nonorganic causes. Muscular failure is the other common cause especially in debilitated surgical patients. Other causes include airway obstruction and shunt.

B. Criteria for Initiation. Many criteria have been developed for intubation and the initiation of ventilatory support. These include failure to maintain a PaO₂ of <70 on 100% O₂, CO₂ retention with a CO₂ of >50, respiratory rate >35, use of accessory muscles, respiratory fatigue, central nervous system (CNS) impairment, or failure to protect the airway. The axiom “intubate early” or “if you think about intubation, intubate” serves to keep the airway protected while further diagnosis and treatment of the respiratory failure proceeds.

C. Ventilator Management. Once intubation has been accomplished most patients should be given total ventilatory support for at least 24–48 h. Initial settings should include a tidal volume (TV) of 7–12 cc/kg, FIO₂ of 100%, respiratory rate of 16–18, and a positive end-expiratory pressure (PEEP) of +5. Frequent blood gas measurements and constant O₂ saturation monitoring should then be used to maintain PaO₂ ~100 and PaCO₂ ~40 with minimal settings. Particular attention should be directed at minimizing the FIO₂ early in the ventilatory course to minimize the oxygen toxicity that takes place with FIO₂ of >60% for more than 48 h.

In its most simplistic form, the blood gas can be broken down into parameters that apply to ventilation and those that apply to oxygenation. Ventilatory parameters affect the pH and PaCO₂ and are controlled by adjusting the TV and rate. TV is usually set based upon the patient’s weight/mass and the rate is utilized to maintain a PaCO₂ around 40.

The PaO₂ and O₂ saturation are measurements of oxygenation and can be controlled by adjusting the FIO₂ and PEEP. Ideally the FIO₂ should be the minimum necessary to maintain PaO₂ around 100 with an O₂ sat above 90%. A PEEP of +5 is considered physiologic and is applied any time ventilatory support is initiated.

D. Discontinuing mechanical ventilation is a complex process. Patients who are ventilated because of neurologic impairment (iatrogenic or organic) are easily extubated when they awake. Patients ventilated for general anesthesia purposes fall into this group. Slightly more complicated are those patients who are ventilated for more mechanical reasons and need to be weaned from the vent.

Before extubation and discontinuation of ventilatory support can be considered, oxygenation requirements must be minimal. Patients should maintain adequate PaO₂ or O₂ saturation on FIO₂ of 40% and PEEP of 5 cm/H₂O. Once minimal FIO₂ is achieved, patients can either be weaned or undergo a spontaneous breathing trial. In a spontaneous breathing trial, patients are placed to continuous positive airway pressure (CPAP) for 30 min. Patients who can maintain a respiratory rate of <20 and maintain adequate oxygenation for 30 min are candidates for extubation.
Patients who fail these spontaneous breathing trials need further weaning and exercise to reduce their requirements for ventilatory support. Two popular methods are T piece weaning where patients are placed on a T piece for increasing periods until they are able to tolerate T piece for 24 h at which time extubation is attempted. Intermittent mandatory ventilation (IMV) weaning is similar to T piece weaning with patients undergoing a gradual decrease of the set IMV rate for increasing periods of time until they are placed on CPAP (essentially an IMV rate of zero) in preparation for extubation.

Many confounding factors exist that slow or prevent discontinuation of ventilatory support. These include electrolyte abnormalities, agitation leading to perceived dyspnea, increased CO₂ production caused by inadequate resuscitation, or overfeeding. Correction and optimization of each of these variables is necessary for weaning and ultimately extubation to be successful.
A. Introduction
1.) Breathing
2.) Oxygenation
3.) Ventilation

B. Criteria for Intubation
1.) $\text{PaO}_2 < 70$ on 100% $\text{O}_2$
2.) $\text{CO}_2 > 50$
3.) Respiratory rate > 35
4.) Use of accessory muscles
5.) Respiratory fatigue
6.) CNS impairment
7.) Failure to protect airway

C. Ventilator Management
1.) Initial settings
2.) Frequent ABG
3.) Maintain $\text{PaO}_2 \sim 100$ and $\text{PCO}_2 \sim 40$
4.) Minimize oxygen toxicity

D. Discontinuing Mechanical Ventilation
1.) Oxygen requirements minimal
2.) Maintain respiratory rate <20 over 30 minutes
3.) Weaning trials
Hypoxemia and Hypoxia

A. Although the terms hypoxia and hypoxemia are often used interchangeably, they are not synonymous. Hypoxemia is defined as a condition where arterial oxygen tension \( P_{\text{AO}_2} \) is below normal (normal \( P_{\text{AO}_2} = 80–100 \text{ mmHg} \)). Hypoxia is defined as the failure of oxygenation at the tissue level. It is not measured directly by a laboratory value (though an increased arterial lactate level usually accompanies tissue hypoxia). Hypoxia and hypoxemia may or may not occur together. Generally, the presence of hypoxemia suggests hypoxia. However, hypoxia may not be present in patients with hypoxemia if the patient compensates for a low \( P_{\text{AO}_2} \) by increasing oxygen delivery. This is typically achieved by increasing cardiac output or decreasing tissue oxygen consumption. Conversely, patients who are not hypoxemic may be hypoxic if oxygen delivery to tissues is impaired or if tissues are unable to use oxygen effectively. Nevertheless, hypoxemia is by far the most common cause of tissue hypoxia.

The five causes of tissue hypoxia are listed below:

1. **Hypoxic hypoxia (disorders causing low arterial \( P_{\text{AO}_2} \)):**
   - Decreased \( P_{\text{AO}_2} \) (decreased \( F_{\text{IO}_2} \), low barometric pressure, or causes of elevated \( P_{\text{CO}_2} \)). Any cause of hypoventilation will cause hypoxemia if \( P_{\text{CO}_2} \) rises high enough.
   - V/Q (ventilation perfusion) mismatch in the lungs causing a widened A-a gradient. V/Q mismatch responds to oxygen therapy. Pneumonia, heart failure, and atelectasis are common causes of V/Q mismatch.
   - Increased pulmonary shunt (\( Q_s/Q_t \)), that is, perfusion without gas exchange. \( Q_s/Q_t \) does not respond to oxygen therapy. Adult respiratory distress syndrome (ARDS) is an example of severe \( Q_s/Q_t \).

2. **Stagnant or circulatory hypoxia:** Stagnant or circulatory hypoxia is caused by
   - Decreased cardiac output. Extremely low cardiac output (e.g., cardiogenic shock) causes a decreased mixed venous oxygen tension that does not permit complete oxygenation of the blood during pulmonary gas exchange.
   - Increased nonpulmonary shunting. In certain diseases (e.g., cirrhosis) large amounts of blood flow bypass the lungs entirely preventing gas exchange.

3. **Anemic hypoxia:** Anemic hypoxia is caused by reduced tissue oxygenation as a consequence of low hemoglobin or hemoglobin with abnormal oxygen carrying capacity.

4. **Histotoxic hypoxia:** Histotoxic hypoxia is caused by the inability of the tissues to use oxygen, even in the absence of hypoxemia. Cyanide poisoning, where cyanide interferes with aerobic cellular metabolism, is a classic example of histotoxic hypoxia.

5. **Oxygen affinity hypoxia:** When the oxygen dissociation curve is shifted to the left, there is an increased affinity and

Thus the normal \( P_{\text{AO}_2} \) is \( \sim 100 \text{ mmHg} \). Given that the A-a gradient is less than 20, normal \( P_{\text{AO}_2} \) is 80–100 mmHg when the patient is breathing room air. This corresponds to an oxygen saturation of greater than 95%.

Hypoxic hypoxia is caused by

(a) Decreased \( P_{\text{AO}_2} \) (decreased \( F_{\text{IO}_2} \), low barometric pressure, or causes of elevated \( P_{\text{CO}_2} \)). Any cause of hypoventilation will cause hypoxemia if \( P_{\text{CO}_2} \) rises high enough.

(b) V/Q (ventilation perfusion) mismatch in the lungs causing a widened A-a gradient. V/Q mismatch responds to oxygen therapy. Pneumonia, heart failure, and atelectasis are common causes of V/Q mismatch.

(c) Increased pulmonary shunt (\( Q_s/Q_t \)), that is, perfusion without gas exchange. \( Q_s/Q_t \) does not respond to oxygen therapy. Adult respiratory distress syndrome (ARDS) is an example of severe \( Q_s/Q_t \).

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\[
P_{\text{AO}_2} = F_{\text{IO}_2} (BP - P_{\text{H}_2O}) - \left( \frac{P_{\text{CO}_2}}{R} \right)
\]

where \( P_{\text{AO}_2} \) is the alveolar oxygen tension, \( F_{\text{IO}_2} \) is the fraction of inspired oxygen, \( BP \) is the barometric pressure (760 mmHg at sea level), \( P_{\text{H}_2O} \) is the pressure of water vapor in the inspired air (47 mmHg), \( P_{\text{CO}_2} \) is the arterial carbon dioxide tension, and \( R \) is the respiratory quotient (assumed to be 0.8 in most patients).

In the typical patient breathing room air at sea level (\( F_{\text{IO}_2} = 0.21 \)) the equation becomes:

\[
P_{\text{AO}_2} = 0.21(760 - 47) - \frac{40}{0.8} = 100
\]
consequent decreased oxygen delivery to the tissues. While this is usually not clinically significant, in conditions such as carbon monoxide poisoning and massive blood transfusions, the shift of the oxygen hemoglobin dissociation curve to the left can result in tissue hypoxia.

B. Clinical Assessment. Clinical assessment of adequate oxygenation can be difficult. Taking a good history is the first step. Patients most susceptible to hypoxemia are those with a history of either cardiac or pulmonary disease. Smoking history and a history of respiratory illness are crucial. In the postoperative patient (especially those with upper abdominal incisions) atelectasis causing V/Q mismatch is common in the immediate postoperative period, while pneumonia is often seen later in the postoperative course. Atelectasis and pneumonia are more common in patients who are elderly, obese, or have neurologic diseases and those treated with large doses of sedatives or opiates.

Most, but not all, hypoxemic patients will complain of shortness of breath. Dyspnea indicates that the patient is experiencing an increase in the work of breathing. Normally, breathing accounts for a very small amount of the patient’s total oxygen consumption. When there is dyspnea, the work of breathing is significant enough to be noticed by the patient. Mental confusion, while nonspecific, is often seen in severe hypoxemia. In any patient with an acute change in mental status, the first condition that must be excluded is hypoxemia.

The most common vital sign abnormalities in hypoxemia are tachypnea (respiratory rate >24/min) and tachycardia (heart rate >100/min). Patients with hypoxemia typically increase their rate of respiration while decreasing their tidal volume (in the attempt to decrease the work of breathing). This is not an invariable finding, especially in patients with central nervous system disease where central breathing patterns may predominate. While hypoxemia is a strong sympathetic stimulus causing tachycardia, patients with limited cardiac reserve may not manifest this finding. In severe hypoxemia, vagal stimulation and the direct effect of hypoxemia on the heart may result in bradycardia. The absence of tachycardia and/or tachypnea should never be taken as a sign that the patient is not hypoxemic.

In the absence of arterial hypoxemia, cyanosis can be found in patients who are severely vasoconstricted or hypothermic where intense local venous desaturation occurs. This generally indicates some degree of tissue hypoxia. Owing to the patient’s skin pigment and the light source there is a degree of observer variability in the diagnosis of cyanosis. Occasionally, other changes in skin coloration (e.g., cherry red in carbon monoxide poisoning or dull red in cyanide poisoning) indicate tissue hypoxia.

With hypoxia, aerobic metabolism is replaced by anaerobic tissue metabolism. This leads to reduced cellular function, lactic acidosis, cessation of the ATP-dependent ion pumps within cell membranes, and subsequently, uncontrolled cell death. Loss of function of cerebral tissue occurs after less than 1 min of hypoxia. The myocardium can survive up to 4 min and skeletal muscles up to 2 h without oxygen. Irreversible damage occurs if the period of anoxia that results in loss of function is prolonged by a factor of 4. Partial recovery is expected with lesser periods of hypoxia. When hypoxia is suspected in a patient, the most important condition to diagnose is hypoxemia because it is the most common cause of tissue hypoxia and the most readily treatable.

C. The physician’s first priority is to determine whether the patient is stable, or in an imminently life-threatening situation. This is usually evident based on the presence of severe deviations from normal in heart rate, respiratory rate, blood pressure, and mental status.

In the approach to hypoxia the physician immediately looks to diagnose and treat hypoxemia. If the patient is unstable, start the patient on oxygen, draw an arterial blood gas and arterial lactate, obtain a chest x-ray, and then determine whether the patient can be stabilized with high-flow oxygen (Venturi Mask or continuous airway pressure) alone or whether intubation will be necessary. The indications for intubation are (1) the need for positive pressure ventilation, (2) obstruction of the airway, (3) protection of the airway, for example, neurologic impairment, (4) provision of tracheobronchial toilet when the patient is unable to generate an effective cough. If any one of these indications is present, the physician should undertake intubation before instituting a more precise workup for the etiology of the problem. The most common etiologies of hypoxic hypoxia are sepsis, cardiac abnormalities, and pulmonary embolism. Workup should include culture of suspected sites of infection, appropriate radiographic examinations (e.g., V/Q scan and computerized axial tomography), and/or cardiac studies (electrocardiogram, echocardiogram, and pulmonary artery catheterization).

If the patient is stable, the physician should order pulse oximetry for the patient. If the saturation is normal (exceeds 95%), hypoxemia is unlikely, but the patient may still have a condition where tissue hypoxia exists in the absence of hypoxemia. The patient’s venous serum bicarbonate should be checked to see if it is grossly abnormal, less than 18 mEq/L, or greater than 30 mEq/L, either of which is suggestive of a serious acid–base disorder. If the serum bicarbonate is normal in the face of normal oxygen saturation, in all likelihood the patient can be observed. If the serum bicarbonate is grossly abnormal, a chest x-ray, arterial blood gas, and arterial lactate should be obtained. Further diagnostic measures should proceed based on the results of these studies.

If the initial saturation reading is between 90 and 95%, a trial of low-flow oxygen (by nasal cannula) is warranted. The patient should receive a chest x-ray and should be observed for the response to oxygen. If the saturation after oxygen therapy rises consistently above 98%, the patient should be observed with any diagnostic tests obtained as clinically indicated. If the saturation does not rise consistently above 98%, an arterial...
blood gas and arterial lactate should be obtained. On the basis of these results, the decision for high-flow oxygen or intubation should be made and a careful workup should ensue.

In the case where the patient’s initial oxygen saturation is below 90%, the approach should be similar to that for the unstable patient—immediate high-flow oxygen therapy, an arterial blood gas and arterial lactate, and chest x-ray. On the basis of patient’s response to high-flow oxygen, the physician should then determine whether intubation is warranted. Once the patient’s oxygen saturation has increased to a level greater than 95%, appropriate diagnostic workup can be instituted.
Hypoxemia and Hypoxia

A. Tissue Hypoxia
1.) Hypoxemic hypoxia
2.) Stagnant or circulatory hypoxia
3.) Anemic hypoxia
4.) Histotoxic hypoxia
5.) Oxygen affinity hypoxia

B. Clinical Assessment
1.) History
   A. cardiac/pulmonary disease
   B. smoking
   C. postoperative
   D. infection
   E. sedatives
   F. age
2.) Physical Exam
   A. mental status
   B. vital signs
   C. cyanosis

C. Stability Assessment

Unstable Patient
a.) oxygen supplement
b.) ABG, lactate
c.) CXR
d.) Intubate
e.) Blood culture
f.) V/Q scan or CAT scan
g.) EKG
h.) Echocardiogram
i.) PA catheter

Stable Patient

A.) Sat >95%
   1.) HCO₃ normal – observe
   2.) HCO₃ abnormal – CXR, ABG, Lactate
B.) Sat 90-95%
   1.) Oxygen supplement – if not improved ABG, lactate, intubate or high flow O₂
C.) Sat <90%
   1.) Similar approach as unstable patient.
Hypotension in the Postoperative Patient

Anthony W. Kim and Justin B. Maxhimer

A. Differential Diagnosis. Postoperative hypotension can have several causes; however, hemorrhage is the most common. By the time patients display physical signs of hemorrhage, such as hypotension, they have lost at least 30% or ~1,500 ml of their blood volume. The goal is to identify potential causes at an early stage and to prevent overt clinical shock. Shock is not the equivalent of hypotension, but episodes of hypotension can be an indicator of a pathologic condition that can ultimately lead to shock. In the case of bleeding, expedient identification and correction of hemorrhage are critical in the prevention of complete exsanguination.

B. Evaluation. The appropriate management of postoperative hypotension should consist of parallel reasoning and action. A quick yet adequate history must be obtained; it is important to identify any preoperative conditions such as anemia or clotting disorders that could predispose the patient to bleeding. Additional information regarding both a history of drug allergies and preoperative medications is necessary. Knowledge of the recent postoperative events such as the administration of certain medications (e.g., ampicillin in a true penicillin allergic patient) or the omission of essential drugs (e.g., stress dose hydrocortisone in a steroid-dependent patient) is extremely important as these can be managed and reversed quickly. History of significant intraoperative bleeding or certain anesthetics used during surgery can be of significant importance.

Physical examination is extremely important. Postoperative hypotension should never be managed from a distance; it requires a bedside evaluation to determine the degree to which the patient is affected. In addition to the blood pressure, the pulse rate (>100 beats/min), respiratory rate (>20 breaths/min), temperature (>100.5 or <96.0 °F), and urine output (<0.5 cc/kg/h) can indicate the degree of duress the patient is experiencing. A quick assessment of the mental status and a rapid yet thorough evaluation of the integument, neck, lungs, chest, abdomen, back, rectum, and extremities can provide extremely useful clues to the etiology of the hypotension. The extremity exam can also indicate the degree to which the patients are perfusing their organs. Palpable carotid, femoral, and radial pulses indicate a systemic systolic blood pressure of at least 60, 70, and 80 mmHg, respectively.

C. Diagnostic Tests. Diagnostic tests should include an arterial blood gas, complete blood count, chemistry profile, coagulation profile, electrocardiogram, and chest radiograph. The hemoglobin or hematocrit may or may not reflect the extent of blood loss, depending on how much time has elapsed, whether equilibration has occurred, and whether large volumes of crystalloid have been given. The coagulation profile, particularly if abnormal, may support a hemorrhagic etiology. In the presence of overt hypotension and a hemoglobin of less than 8.0 g/dl, typed and crossmatched blood should be given (O-negative in an unmatched patient). An exception to this may be the hypotensive patient with cardiac risk factors in which case, the threshold for transfusion is lower. Patients with a hemoglobin level of less than 10.0 g/dl are treated with transfusion. These patients should be given blood to maintain a hemoglobin level of greater than 10.0 g/dl to optimize myocardial O2 delivery. An arterial blood gas, electrocardiogram, and chest radiograph can demonstrate a pulmonary or cardiac etiology of hypotension. The arterial blood gas, in concert with a chemistry profile, can also support a metabolic etiology of hypotension (e.g., Addisonian crisis).

D. Initial Management. Temporizing measures must be instituted while information gathering and workup are ongoing. Immediately ensuring that the Airway, Breathing, and Circulation (ABCs) are intact is of greatest importance. Oxygen by nasal cannula, face mask, or endotracheal intubation must be administered to maximize oxygenation. Crystalloid (0.9 NaCl, 0.45 NaCl, or lactated ringers) or colloid (blood, clotting factors, or albumin) should be given intravenously to support and resuscitate the patient. The advantages of additional fluid in a hypovolemic patient outweigh the disadvantages of additional fluid in a hypervolemic patient, that is, one with congestive heart failure. Therefore, one should be aggressive with fluid administration. Vasodilating agents, hypotensive medication, sedatives, and opioid analgesics should be discontinued.
E. Hemorrhage. If clinical assessment suggests that an acute hemorrhagic process is occurring from an identifiable source of external bleeding, simple pressure or ligation of the bleeding site may be sufficient. This should be followed up with vigilant monitoring of vital signs, urine output, and serial hemoglobin measurements to confirm adequate hemostasis. If it is apparent that the bleeding is coming from the operative site and there is no coagulopathy, then reoperation is indicated without delay. The blood bank should be notified to type and crossmatch the necessary blood products. Blood products, in addition to continued crystalloid and colloid resuscitation, should be maintained until the patient is returned to the operating room. The decision to reoperate must frequently be made based on clinical judgment rather than wait for confirmatory studies which would needlessly jeopardize the patient’s well-being and outcome. If the clinical assessment is consistent with a coagulopathic bleed, the patient should be resuscitated with blood products in addition to crystalloid and colloid fluids. However, if the patient does not respond, then reexploration is mandated to rule out a discrete bleeding source. Even in the face of coagulopathy, postoperative bleeding is most likely secondary to a surgical cause.

F. Nonhemorrhagic Hypovolemia. Hypotension secondary to hypovolemia from preoperative or intraoperative under-resuscitation mandates immediate and aggressive resuscitation with either crystalloid or colloid products. Resuscitative intravenous fluids should be continued at a rate greater than the maintenance rate to ensure an adequate systolic and mean blood pressure of at least 90 and 60 mmHg, respectively, and a urine output of greater than 0.5 cc/kg/h in adults. A severe hypovolemic state needing a significant amount of resuscitation may require central venous pressure catheter or Swan Ganz catheter monitoring to guide resuscitation.

G. Cardiogenic Causes. An electrocardiogram, cardiac monitor, and chest x-ray can help delineate an intrinsic or extrinsic cardiogenic cause of postoperative hypotension. An electrocardiogram can identify a myocardial infarction. Cardiac enzymes will help confirm ECG findings. If a myocardial infarction is definitively diagnosed, then supportive management in concert with the assistance of the cardiologist is required. Congestive heart failure secondary to iatrogenic hypervolemia may require diuresis and supportive management to optimize the patient’s cardiac status along the starling curve. Placement of a central venous or Swan Ganz catheter may guide management. Pulmonary embolism, cardiac tamponade, profound valvular disease, or tension pneumothorax need immediate and etiology-specific treatment. If a pulse is palpable, an irregular rate and rhythm should be treated by advanced cardiac life-support protocol. In the absence of a pulse, cardiopulmonary resuscitation and advance cardiac life-support protocols should be followed.

H. Sepsis. In many general surgery patients, postoperative sepsis from a preoperative disease process or an intraoperative complication can occur. Physical examination findings include a fever or hypothermia and evidence of shunting of the peripheral circulation. An elevated white blood cell count or positive cultures can support this diagnosis. Confirmation can also be achieved with imaging studies such as CT scans. In these circumstances, treatment should begin with resuscitative efforts including intravenous crystalloid or colloid boluses and increased maintenance intravenous fluids. Culture-directed antibiotic therapy is necessary to control the septic condition. Central venous pressure catheter or Swan Ganz catheter monitoring can confirm hemodynamic changes consistent with sepsis and can guide resuscitative efforts. Pressor support can then be administered as needed with monitoring of the hemodynamic parameters. If an abscess is present, either ultrasound or CT-guided external drainage must be weighed against operative drainage.

I. Neurogenic Shock. Neurogenic shock is a rare cause of hypotension in the general surgery patient. It must be considered if the etiology remains unclear after ruling out a hypovolemic, cardiogenic, or septic cause. The loss of vasomotor control typically associated with this condition can manifest with good skin perfusion, low or normal urine output, and a slowed heart rate in addition to hypotension. If a neurologic cause of hypotension is identified, immediate resuscitation with 0.9 normal saline or 0.45 normal saline or lactated ringer is required. Cessation of reversible causes of neurologic hypotension such as epidural pain catheters or other anesthetic agents should be done. This will serve both a diagnostic and therapeutic purpose. Central venous pressure or Swan Ganz catheter monitoring with the addition of pressor support may be needed until the neurogenic cause is reversed.
Hypotension in the Postoperative Patient

A. Differential Diagnosis
   hemorrhage most common
   hypovolemia, cardiogenic,
   sepsis, neurogenic

B. Evaluation
   history and physical examination

C. Diagnostic Tests
   ABG, CBC, serum chemistries,
   EKG, CXR

D. Initial Management
   ABC's of resuscitation
   IV fluids

E. Hemorrhage
   re-operation usually required

F. Non-hemorrhagic Hypovolemia
   resuscitation

G. Cardiogenic
   myocardial infarction
   congestive failure
   pulmonary embolism
   tamponade
   tension pneumothorax

H. Sepsis
   resuscitate
   treat underlying cause

I. Neurogenic
## Appendix

### Cancer Screening Guidelines

Shahnaz Chowdhry

An adaptation of 2005 American Cancer Society Recommendations for the Early Detection of Cancer in Average-risk Asymptomatic People

<table>
<thead>
<tr>
<th>Cancer Site</th>
<th>Population</th>
<th>Test/Procedure &amp; Frequency</th>
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<tbody>
<tr>
<td>Breast</td>
<td>Women 20-39</td>
<td>Clinical breast exam (CBE) at least every 3 years</td>
</tr>
<tr>
<td></td>
<td>Women ≥ 40</td>
<td>Annual CBE and annual mammogram</td>
</tr>
<tr>
<td>Colorectal</td>
<td>Men/Women ≥ 50</td>
<td>Fecal occult blood test (FOBT) annually</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>AND</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Colonoscopy every 10 years</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>OR</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Double contrast barium enema with flexible sigmoidoscopy every 5 years</td>
</tr>
<tr>
<td>Prostate</td>
<td>Men ≥ 50</td>
<td>Digital rectal exam (DRE) and prostate-specific antigen test (PSA) annually</td>
</tr>
<tr>
<td>Cervix</td>
<td>Women age ≥ 18</td>
<td>Pap test beginning 3 years after starting vaginal intercourse, but no later than 21, then annually</td>
</tr>
<tr>
<td></td>
<td>Women ≥ 30-69</td>
<td>Pap test every 2-3 years (if 3 normal test results in a row)</td>
</tr>
<tr>
<td></td>
<td>Women ≥ 70</td>
<td>No screening if 3 or more normal tests in last 10 years or total hysterectomy</td>
</tr>
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