THIRD EDITION

A survival manual for the surgery clerkship

Tips on what you must know to excel on the shelf exam and impress on rounds

Updated to reflect the latest protocols and guidelines

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Third Edition

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To my dad, Dr. Ganti L. Rao for constantly encouraging me to achieve; and to my children, Thor, Tej, Trilok, Karthik & Vaishnavi who put up with their mother’s very busy career demands.

—Latha Ganti, MD, MS, MBA, FACEP

For Jenny G—New York’s best

—Matthew S. Kaufman, MD

My work in this book is dedicated to my parents, Mr. Rajeshwar Mishra and Mrs. Indu Mishra.

—Nitin Mishra, MBBS, MS, MRC S (UK)
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introduction

This book is designed in the tradition of the First Aid series of books. It is formatted in the same way as the other books in the series. You will find that, apart from preparing you for success on the clerkship exam, this resource will also help guide you in the clinical diagnosis and treatment of common surgical conditions.

The content of the book is based on the objectives for medical students as determined by the Association for Surgical Education (ASE). Each chapter contains the major topics central to the practice of general surgery and has been specifically designed for the medical student. The book is divided into general surgery, which contains topics that comprise the core of the surgery rotation, and subspecialty surgery, which may be of interest but is generally considered less high yield for the clerkship. Knowledge of a subspecialty topic may be useful if observing a related surgery or if requesting a letter from a surgeon in that field.

The content of the text is organized in the format similar to other texts in the First Aid series. Topics are listed with bold headings, and the “body” of the topic provides essential information. The outside margins contain mnemonics, diagrams, summary or warning statements, and tips. Tips are categorized into Exam Tip 📚, Ward Tip ⌌, and OR tip ⬂.
How to contribute

To continue to produce a high-yield review source for the surgery clerkship, you are invited to submit any suggestions or correction. Please send us your suggestions for:

- New facts, mnemonics, diagrams, and illustrations
- Low-yield facts to remove

For each entry incorporated into the next edition, you will receive personal acknowledgment. Diagrams, tables, partial entries, updates, corrections, and study hints are also appreciated, and significant contributions will be compensated at the discretion of the authors. Also let us know about material in this edition that you feel is low yield and should be deleted. You are also welcome to send general comments and feedback, although due to the volume of e-mails, we may not be able to respond to each of these.

The preferred way to submit entries, suggestions, or corrections is via email. Please include name, address, school affiliation, phone number, and e-mail address (if different from the address of origin). If there are multiple entries, please consolidate into a single e-mail or file attachment. Please send submissions to:

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Otherwise, please send entries, neatly written or typed to:

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All entries become property of the authors and are subject to editing and reviewing. Please verify all data and spellings carefully. In the event that similar or duplicate entries are received, only the first entry received will be used. Include a reference to a standard textbook to facilitate verification of the fact. Please follow the style, punctuation, and format of this edition if possible.

Note to contributors

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# How to Succeed in the Surgery Clerkship

In the Operating Room...  

On the Wards...  

- Be on Time  
- Dress in a Professional Manner  
- Be Respectful  
- Be Aware of the Hierarchy  
- Address Patients and Staff in a Respectful Way  
- Take Responsibility for Your Patients  
- Respect Patients’ Rights  
- Volunteer  
- Be a Team Player  
- Be Honest  
- Keep Patient Information Handy  
- Present Patient Information in an Organized Manner  
- Presenting the Chest X-ray (CXR)  
- Types of Notes  
- Your Rotation Grade  
- How to Study  
  - Make a List of Core Material to Learn  
  - As You See Patients, Note Their Major Symptoms and Diagnosis for Review  
  - Select Your Study Material  
  - Prepare a Talk on a Topic  
  - Procedures  
- How to Prepare for the Clinical Clerkship Examination  
  - Study with Friends  
  - Study in a Bright Room  
  - Eat Light, Balanced Meals  
  - Take Practice Exams  
  - Tips for Answering Questions  
- Pocket Cards for the Wards
The surgery clerkship is unique among all the medical school rotations. Even if you are dead sure you do not want to be a surgeon, it can be a very fun and rewarding experience if you approach it prepared. There are three key components to the rotation: (1) what to do in the OR, (2) what to do on the wards, and (3) how to study for the exam.

**in the operating Room . . .**

One of the most fun things on the surgery rotation is the opportunity to scrub in on surgical cases. The number and types of cases you will scrub in on depends on the number of residents and students on that service and how busy the service is that month. At some places, being able to go to the OR is considered a privilege rather than a routine part of the rotation. A few tips:

- **Eat before you begin the case.** Some cases can go on for longer than planned and it isn't cool to leave early because you are hungry (read unprepared!) or, worse, to pass out from exhaustion. As a student, your function in the OR will most likely be to hold retraction. This can be tedious, but it is important to pay attention and do a good job. Not pulling in the right direction obscures the view for your attending, and pulling too hard can destroy tissue. Many students get light-headed standing in one position for an extended period of time, especially when they are not used to it. Make sure you shift your weight and bend your knees once in a while so you don't faint. If you feel you are going to faint, then say something—ask one of the surgical techs to take over or state discreetly that you need relief. Do not hold on to the bitter end, pass out, and take the surgical field with you (believe it or not, this has actually happened; we print this advice from real experience).

- **Find out about the case as much as possible beforehand.** Usually, the OR schedule is posted the night before, so you should be able to tell. Read up on the procedure as well as the pathophysiology of the underlying condition. Know the important anatomic landmarks. Read up on the patient’s H&P, with attention paid to PMH, PSH, medications, allergies, and relevant laboratory and radiology results.

- **Find out who you are working with.** If you can, do a quick bibliography search on the surgeon you will be working with. It can never hurt to know which papers (s)he has written, and this may help to spark conversation and distinguish you among the many other students they will have met.

- **Assess the mood in the OR.** The amount of conversation in the OR directed to you varies by attending. Some are very into teaching and will engage you during most of the surgery. Many others act as if you aren’t even there. Some will interact if you make the first move; others nuke all efforts at interaction. You’ll have to figure it out based on the situation. Generally, if your questions and comments reflect that you have read about the procedure and disease, things will go well.

- **Keep a log of all surgeries** you have attended, scrubbed on, or assisted with (see Table I-1). If you are planning to go into general surgery or a surgical subspecialty, it can be useful during residency interviews for conveying how much exposure/experience you have had. This is particularly true if your school’s strength is clinical experience. The log can also be useful if you are requesting a letter from the chairman of surgery whom you have never worked with. It gives her/him an idea of what you have been doing with your rotation. Many rotations will set a minimum number of surgeries you are to attend. Try to attend as many as possible, and document them. This serves both to increase your exposure, and confirm your interest.
on the Wards...

Be on Time

Most surgical ward teams begin rounding between 6 and 7 a.m. If you are expected to “pre-round,” you should give yourself at least 10 minutes per patient that you are following to see the patient and learn about the events that occurred overnight. Like all working professionals, you will face occasional obstacles to punctuality, but make sure this is occasional. When you first start a rotation, try to show up at least 15 minutes early until you get the routine figured out.

Dress in a Professional manner

Even if the resident wears scrubs and the attending wears stiletto heels, you must dress in a professional, conservative manner. Wear a short white coat over your clothes unless discouraged.

Men should wear long pants, with cuffs covering the ankle, a long collared shirt, and a tie. No jeans, no sneakers, no short-sleeved shirts.

Women should wear long pants or knee-length skirt, blouse, or dressy sweater. No jeans, no sneakers, no heels greater than 1½ inches, no open-toed shoes.

Both men and women may wear scrubs occasionally, especially during overnight call or in the operating room. Do not make this your uniform.

Be Pleasant

The surgical rotation is often difficult, stressful, and tiring. Smooth out your experience by being nice to be around. Smile a lot and learn everyone’s name. If you do not understand or disagree with a treatment plan or diagnosis, do not “challenge.” Instead, say “I’m sorry, I don’t quite understand, could you please explain . . .” Be empathetic toward patients.

Be aware of The hierarchy

The way in which this will affect you will vary from hospital to hospital and team to team, but it is always present to some degree. In general, address your questions regarding ward functioning to interns or residents. Address your medical questions to attendings; make an effort to be somewhat informed on your subject prior to asking attendings medical questions.
Address Patients and Staff in a Respectful Way

Address patients as Sir, Ma’am, or Mr., Mrs., or Miss. Do not address patients as “honey,” “sweetie,” and the like. Although you may feel that these names are friendly, patients will think you have forgotten their name, that you are being inappropriately familiar, or both. Address attending physicians as “doctor” unless told otherwise.

Take Responsibility for Your Patients

Know everything there is to know about your patients, their history, test results, details about their medical problem, and prognosis. Keep your intern or resident informed of new developments that he or she might not be aware of, and ask for any updates you might not be aware of. Assist the team in developing a plan and speaking to radiology, consultants, and family. Never give bad news to patients or family members without the assistance of your supervising resident or attending.

Respect Patients’ Rights

1. All patients have the right to have their personal medical information kept private. This means do not discuss the patient’s information with family members without the patient’s consent, and do not discuss any patient in hallways, elevators, or cafeterias.

2. All patients have the right to refuse treatment. This means they can refuse treatment by a specific individual (you, the medical student) or of a specific type (no nasogastric tube). Patients can even refuse life-saving treatment. The only exceptions to this rule are if the patient is deemed to not have the capacity to make decisions or understand situations (in which case a health care proxy should be sought) and if the patient is suicidal or homicidal.

3. All patients should be informed of the right to seek advanced directives on admission. Often, this is done by the admissions staff, in a booklet. If your patient is chronically ill or has a life-threatening illness, address the subject of advanced directives with the assistance of your attending.

Volunteer

Be self-propelled, self-motivated. Volunteer to help with a procedure or a difficult task. Volunteer to give a 20-minute talk on a topic of your choice. Volunteer to take additional patients. Volunteer to stay late.

Be a Team Player

Help other medical students with their tasks; teach them information you have learned. Support your supervising intern or resident whenever possible. Never steal the spotlight, steal a procedure, or make a fellow medical student look bad.

Be Honest

If you don’t understand, don’t know, or didn’t do it, make sure you always say that. Never say or document information that is false (a common example: “bowel sounds normal” when you did not listen).
keeP PaTienT infor maTion hanDy

Use a clipboard, notebook, or index cards to keep patient information, including a miniature history and physical, lab, and test results at hand.

PresenT PaTienT infor maTion in an or ganizeDmanner

Here is a template for the “bullet” presentation:

This is a [age] year old [gender] with a history of [major history such as HTN, DM, coronary artery disease, CA, etc.] who presented on [date] with [major symptoms, such as cough, fever and chills], and was found to have [working diagnosis], [Tests done] showed [results]. Yesterday the patient [state important changes, new plan, new tests, new medications]. This morning the patient feels [state the patient’s words], and the physical exam is significant for [state major findings]. Plan is [state plan].

The newly admitted patient generally deserves a longer presentation following the complete history and physical format.

Some patients have extensive histories. The whole history can and probably should be present in the admission note, but in ward presentation it is often too much to absorb. In these cases it will be very much appreciated by your team if you can generate a good summary that maintains an accurate picture of the patient. This usually takes some thought, but it’s worth it.

PresenTing The chesTraDiograPh (cXR)

3 “shuns” + “ABCDEFGHI”

1. Identification (identificay-SHUN)
   – correct patient, medical record number, date, and time.
2. Rotation (rotay-SHUN)
   – is this a posteroanterior (PA) or anteroposterior (AP) film?
   – are the clavicles at an equal height and can you see both medial convexities?
3. Penetration (penetray-SHUN)
   – the spinal column should become obscured halfway down the mediastinal silhouette.
   A. Airway
      – trachea midline, without deviation
      – carina and bronchi visible, without distraction
      – look for presence and position of ETT—should be 2 cm above the carina
      – look for pneumomediastinum (perforation)
   B. Bones
      – good inspiratory effort? (7–8 ribs visible)
      – look for rib/clavicular/humeral/ternal/scapular fractures or joint dislocations
   C. Costophrenic (angle)
      – look for blunting (hemothorax/pleural effusion)
   D. Diaphragm
      – look for flattening (COPD), hemidiaphragm
      – look for free air under the diaphragm (pneumoperitoneum)
E. Esophagus
   - can’t really see the esophagus, but can see presence of an NG or OG tube. Make sure that it’s uncoiled, in the stomach, and not in the lung.
   - make sure the stomach is in the abdomen, not the chest (diaphragmatic hernia vs. rupture)
   - look for pneumomediastinum (perforation)
F. Lung Fields
   - look for airspace disease, opacities, pneumothorax, venous congestion, hilar lymphadenopathy
   - look for presence of chest tubes
G. Great vessels
   - look for widening of mediastinum or mediastinal shift
   - look for presence of central venous catheters
H. Heart
   - look for cardiomegaly
   - look for retrocardiac opacities
I. Interval
   - compare this film to the patient’s previous films to see changes.

A sample CXR presentation may sound like:

This is the CXR of Mr. Jones. The film is an AP view with good inspiratory effort. There is an isolated fracture of the 8th rib on the right. There is no tracheal deviation or mediastinal shift. There is no pneumo- or hemothorax. The cardiac silhouette appears to be of normal size. The diaphragm and heart borders on both sides are clear; no infiltrates are noted. There is a central venous catheter present, the tip of which is in the superior vena cava.

The key elements of presenting a CXR are summarized in Table I-2.

### Table I-2. How to Present a Chest Radiograph (CXR)

- First, confirm that the CXR belongs to your patient.
- If possible, compare to a previous film.

Then, present in a systematic manner:

1. **Technique**
   - Rotation, anteroposterior (AP) or posteroanterior (PA), penetration, inspiratory effort.

2. **Bony structures**
   - Look for rib, clavicle, scapula, and sternum fractures.

3. **Airway**
   - Look for tracheal deviation, pneumothorax, pneumomediastinum.

4. **Pleural space**
   - Look for fluid collections, which can represent hemothorax, chylothorax, pleural effusion.

5. **Lung parenchyma**
   - Look for infiltrates and consolidations: These can represent pneumonia, pulmonary contusions, hematoma, or aspiration. The location of an infiltrate can provide a clue to the location of a pneumonia:
     - Obscured right (R) costophrenic angle = right lower lobe
     - Obscured left (L) costophrenic angle = left lower lobe

(continues)
In addition to the admission H&P and the daily progress note, there are a few other types of notes you will write on the surgery clerkship. These include the preoperative, operative, postoperative, and procedure notes. Samples of these are depicted in Tables 1-3 through 1-6.

**Table 1-2. How to Present a Chest Radiograph (CXR) (continued)**

- Obscured R heart border = right middle lobe
- Obscured L heart border = left upper lobe

6. Mediastinum
- Look at size of mediastinum—a widened one (>8 cm) goes with aortic rupture.
- Look for enlarged cardiac silhouette (>1/2 thoracic width at base of heart), which may represent congestive heart failure (CHF), cardiomyopathy, hemopericardium, or pneumopericardium.

7. Diaphragm
- Look for free air under the diaphragm (suggests perforation).
- Look for stomach, bowel, or NGT tube above diaphragm (suggests diaphragmatic rupture).

8. Tubes and lines
- Identify all tubes and lines.
- An endotracheal tube should be 2 cm above the carina. A common mistake is right mainstem bronchus intubation.
- A chest tube (including the most proximal hole) should be in the pleural space (not in the lung parenchyma).
- An NGT tube should be in the stomach, and uncoiled.
- The tip of a central venous catheter (central line) should be in the superior vena cava (not in the right atrium).
- The tip of a Swan–Ganz catheter should be in the pulmonary artery.
- The tip of a transvenous pacemaker should be in the right atrium.

**Types of Notes**

In addition to the admission H&P and the daily progress note, there are a few other types of notes you will write on the surgery clerkship. These include the preoperative, operative, postoperative, and procedure notes. Samples of these are depicted in Tables 1-3 through 1-6.

**Table 1-3. Sample Procedure Note (for Wound Repair)**

Under sterile conditions following anesthesia with 5 cc of 2% lidocaine with epinephrine and negative wound exploration for foreign body, the laceration was closed with 3-0 Bihon sutures. Wound edges were well approximated and no complications occurred. Wound was dressed with sterile gauze and triple antibiotic ointment.

**Table 1-4. Sample Preoperative Note**

<table>
<thead>
<tr>
<th>Pre-op diagnosis:</th>
<th>Abdominal pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedure:</td>
<td>Exploratory laparotomy</td>
</tr>
<tr>
<td>Pre-op tests:</td>
<td>List results of labs (CBC, electrolytes, PT, aPTT, urinalysis), ECG, CXR</td>
</tr>
<tr>
<td></td>
<td>(Most adult patients require coagulation studies; patients over 40 usually need ECG and CXR—these are institution specific.)</td>
</tr>
<tr>
<td>Blood:</td>
<td>How many units of what type were crossmatched and available; or, &quot;none&quot; if no blood needed</td>
</tr>
<tr>
<td>Orders:</td>
<td>For example, colon prep, NPO after midnight, preoperative antibiotics</td>
</tr>
</tbody>
</table>
Many students worry about their grade in this rotation. There is the perception that not getting honors in surgery pretty much closes the door to obtaining a residency spot in general or subspecialty surgery (ophthalmology, otorhinolaryngology, neurosurgery, plastic surgery, urology). While this is not necessarily true, the medicine and surgery clerkships are considered to be among the most important in medical school, so doing well in these is handy for all students. Usually, the clerkship grade is broken down into three or four components.

- Inpatient evaluation. This includes evaluation of your ward time by residents and attendings and is based on your performance on the ward. Usually, this makes up about half your grade and can be largely subjective.
- Ambulatory evaluation. This includes your performance in clinic, including clinic notes and any procedures performed in the outpatient setting.
Written examination. Most schools use the NBME or “Shelf” examination. Some schools have their own homemade version, very similar to the NBME’s. The test is multiple choice. This portion of the grade is anywhere from 20% to 40%, so performance on this multiple-choice test is vital to achieving honors in the clerkship. More on this below.

Objective Structured Clinical Examination (OSCE). Some schools now include an OSCE as part of their clerkship evaluation. This is basically an exam that involves a standardized patient and allows assessment of a student’s bedside manner and physical examination skills. This may comprise up to one-fourth of a student’s grade. It is a tool that will probably become more and more popular over the next few years.

How to Study

Make a List of Core Material to Learn

This list should reflect common symptoms, illnesses, and areas in which you have particular interest, or in which you feel particularly weak. Do not try to learn every possible topic. The Association for Surgical Education (www.surgicaleducation.com) has put forth a manual of surgical objectives for the medical student surgery clerkship, on which this book is based. The ASE emphasizes:

Symptoms and Lab Tests
- Abdominal masses
- Abdominal pain
- Altered mental status
- Breast mass
- Jaundice
- Lung nodule
- Scrotal pain and swelling
- Thyroid mass
- Fluid, electrolyte, and acid–base disorders
- Multi-injured trauma patient

Common Surgeries
- Appendectomy
- Coronary artery bypass grafting (CABG)
- Cholecystectomy
- Exploratory laparotomy
- Breast surgery
- Herniorrhaphy
- Peptic ulcer disease (PUD) surgery
- Bariatric surgery

We also recommend:
- Preoperative care
- Postoperative care
- Wound infection
- Shock

The core of the general surgery rotation consists of the following chapters:
1. The Surgical Patient
2. Wounds
3. Acute Abdomen
4. Trauma
5. Critical Care
6. Fluids, Electrolytes, and Nutrition
7. The Esophagus
8. The Stomach
9. Small Bowel
10. Colon, Rectum, and Anal Canal
11. The Appendix
12. Hernia and Abdominal Wall Problems
13. The Hepatobiliary System
14. The Pancreas
15. Endocrine System
16. The Spleen
17. The Breast
18. Burns
19. Vascular Surgery
20. Cardiothoracic Surgery

The other chapters are somewhat less important, as they focus on subspecialty surgery. The subspecialty chapters are comprehensive and less “high yield” than the abdominal chapters, but they are an excellent primer for anyone considering going into subspecialty surgery. We kept the detail in these chapters due to feedback from several students who wanted a concise but comprehensive overview of surgical subspecialties.

You will notice that the chapters discuss pathophysiology and in general a lot of things that seem like they belong in a medicine book. The reason for this is that the NBME clerkship exam covers the medicine behind surgical disease. The exam does not ask specifics of operative technique. So, in a way, you are studying for three distinct purposes. The knowledge you need on the wards is the day-to-day management know-how. The knowledge you want in the OR involves surgical knowledge of anatomy and operative technique (see OR TIPs). The knowledge you want on the end of rotation examination is the epidemiology, risk factors, pathophysiology, diagnosis, and treatment of major diseases seen on a general surgery service.

**as you see Patients, note Their major symptoms and Diagnosis for review**

Your reading on the symptom-based topics above should be done with a specific patient in mind. For example, if a patient comes to the office with a thyroid mass, read about Graves’ disease, Hashimoto’s, thyroid cancer, and the technique of needle aspiration in the review book that night.

**select your study material**

We recommend:
- This review book, First Aid for the Surgery Clerkship
- A major surgery textbook such as Schwartz’s Principles of General Surgery (costs about $140), or Lawrence’s Essentials of General Surgery.
- A full-text online journal database, such as [www.mdconsult.com](http://www.mdconsult.com) (subscription is $99/year for students)
A small pocket reference book to look up lab values, clinical pathways, and the like, such as Maxwell Quick Medical Reference (ISBN 0964519119, costs $7)

Free smartphone apps to look up drugs, such as Epocrates (Athenahealth, free)

Prepare a Talk on a Topic

You may be asked to give a small talk once or twice during your rotation. If not, you should volunteer! Feel free to choose a topic that is on your list; however, realize that this may be considered dull by the people who hear the lecture. The ideal topic is slightly uncommon but not rare, for example: bariatric surgery. To prepare a talk on a topic, read about it in a major textbook or a review article not more than 2 years old, and then search online or in the library for recent developments or changes in treatment.

Procedures

During the course of the surgery clerkship, there is a set of procedures you are expected to learn or at least observe. The common ones are:

- Intravenous line placement
- Nasogastric tube placement
- Venipuncture (blood draw)
- Foley (urinary) catheter placement
- Wound closure with sutures/staples
- Suture/staple removal
- Surgical knots (hand and instrument ties)
  - Ethicon Endosurgery (ethicon.com) provides free knot-tying training kits with instruction booklets for students. Simply go to their website, click “surgeons/clinicians” to contact a representative, and simply ask them to send one to your house.
- Dressing changes (wet to dry, saline, Vaseline gauze)
- Incision and drainage of abscesses
- Technique of needle aspiration (observe)
- Ankle–brachial index (ABI) measurement
- Evaluation of pulses with Doppler
- Skin biopsy (punch and excisional)
- Removal of surgical drains
- Transillumination of scrotum

How to Prepare for the Clinical Clerkship Examination

If you have read about your core illnesses and core symptoms, you will know a great deal about the medicine of surgery. To study for the clerkship exam, we recommend:

2–3 weeks before exam: Read this entire review book, taking notes.
10 days before exam: Read the notes you took during the rotation on your core content list, and the corresponding review book sections.
5 days before exam: Read this entire review book, concentrating on lists and mnemonics.
2 days before exam: Exercise, eat well, skim the book, and go to bed early.
1 day before exam: Exercise, eat well, review your notes and the mnemonics, and go to bed on time. Do not have any caffeine after 2 p.m.
Other helpful studying strategies include:

**Study with friends**

Group studying can be very helpful. Other people may point out areas that you have not studied enough, and may help you focus on the goal. If you tend to get distracted by other people in the room, limit this to less than half of your study time.

**Study in a bright room**

Find the room in your house or in your library that has the best, brightest light. This will help prevent you from falling asleep. If you don’t have a bright light, get a halogen desk lamp or a light that simulates sunlight (not a tanning lamp).

**Eat light, balanced meals**

Make sure your meals are balanced, with lean protein, fruits and vegetables, and fiber. A high-sugar, high-carbohydrate meal will give you an initial burst of energy for 1–2 hours, but then you’ll drop.

**Take practice exams**

The point of practice exams is not so much the content that is contained in the questions, but the training of sitting still for 3 hours and trying to pick the best answer for each and every question.

**Tips for answering questions**

All questions are intended to have one best answer. When answering questions, follow these guidelines:

Read the actual question, then glance at the answers. For all questions longer than two sentences, reading the actual question stem (the last sentence) and then the answer choices first can help you sift through for key information.

“Look for the words EXCEPT, MOST, LEAST, NOT, BEST, WORST, TRUE, FALSE, CORRECT, INCORRECT, ALWAYS, and NEVER. If you find one of these words, circle or underline it for later comparison with the answer.

**Evaluate each answer as being either true or false. Example:**

Which of the following is least likely to be associated with pulmonary embolism?

A. Tachycardia **T**
B. Tachypnea **T**
C. Chest pain? **F not always**
D. Deep venous thrombosis? **T not always**
E. Back pain **F? aortic dissection**

By comparing the question, noting LEAST, to the answers, “E” is the best answer.

Finally, as the boy scouts say, “BE PREPARED.”
Pocket cards for the Wards

The following “cards” contain information that is often helpful during the surgery rotation. We advise that you make a copy of these cards, cut them out, and carry them in your coat pocket when you are on the wards.

**SUTURE TECHNIQUE**

**I. SIMPLE INTERRUPTED**

- Used to close most simple wounds
- Edges should always be everted to prevent depression of scar. Do this by entering needle at 90 degrees to skin surface and follow curve of needle through skin.
- Entrance and exit point of needle should be equidistant from laceration.
- Do not place suture too shallow, as this will cause dead space.
- Use instrument tie or surgeons knot and place knot to one side of laceration not directly over laceration.

**II. RUNNING**

- Not commonly used in the ED
- Disadvantage: One nicked stitch or knot means the entire suture is out.
- Advantage: Done well with sturdy knots, it provides even tension across wound.

**III. HORIZONTAL MATTRESS**

- This suture also assists in wound edge eversion and helps to spread tension over a greater area.
- This stitch starts out like a simple interrupted suture; however, after the needle exits, it then enters again on the same side that it exited from only a few millimeters lateral to the stitch and equidistant from the wound edge and exits on the opposite side.

**IV. VERTICAL MATTRESS**

- This suture helps in reducing dead space and in eversion of wound edges.
- It does not significantly reduce tension on wound.
- The needle enters the skin further away (more lateral) from the laceration than the simple interrupted and also exits further away on the opposite side.
- It then enters again on the same side that it just exited from but more proximal to the laceration and exits on the opposite side (where it originally entered) proximally.
### V. DEEP SUTURES

- **Absorbable**: Used for delayed closure. *Note: The wound is placed deep inside wound and exits superficially in the subcutaneous layer of the opposite side and exits deep.*
- **Deep sutures are absorbable because you will not be removing them.**
- Use a proper suture size and type to close the skin from the inside. The suture close to the skin will appear as a simple interrupted suture.

### Location | Suture Size & Type | Suture Technique | Removal
--- | --- | --- | ---
Scalp | 3-0 or 4-0 nylon or polypropylene | Interrupted in galea, single tight layer in scalp, horizontal mattress if bleeding not well controlled | 7-12 days
Pinna | 5-0 Vicryl/Dexon in perichondrium | Close perichondrium with interrupted Vicryl and close skin with interrupted nylon | 3-5 days
Eyebrow | 4-0 or 5-0 Vicryl (SQ) | Layered closure | 3-5 days
Eyelid | 6-0 nylon | Single-layer horizontal mattress or simple interrupted | 3-5 days
Lip | 4-0 Vicryl (mucosa) | If wound through lip, close three layers (mucosa, muscle, skin); otherwise do two-layer closure | 3-5 days
Oral cavity | 4-0 Vicryl | Simple interrupted or horizontal mattress if muscularis of tongue involved | N/A
Face | 6-0 nylon (skin) | Simple interrupted for single layer, layered closure for full-thickness laceration | 3-5 days
Trunk | 4-0 Vicryl (SQ, fat) | Single or layered closure | 7-12 days
Extremity | 3-0 or 4-0 Vicryl (SQ, fat, muscle) | Single-layer interrupted or vertical mattress; apply splint if over a joint | 10-14 days
Hands and feet | 4-0 or 5-0 nylon | Single-layer closure with simple interrupted or horizontal mattress; apply splint if over a joint | 7-12 days
Nail bed | 5-0 Vicryl | Meticulous placement to obtain even edges, allow to dissolve | N/A
Hig H-Yiel d F a c t s F o r t H e  s u r g e r Y c l e r k s Hip

The Surgical Patient
Wounds
Acute Abdomen
Trauma
Critical Care
Fluids, Electrolytes, and Nutrition
The Esophagus
The Stomach
Small Bowel

17  Colon, Rectum, and Anal Canal
25  The Appendix
35  Hernia and Abdominal Wall Problems
43  The Hepatobiliary System
61  The Pancreas
73  The Endocrine System
91  The Spleen
107 The Breast
123 Burns

143
167
175
185
215
229
257
265
283
**Preoperative Evaluation**
- Anesthetic History
- The ASA Physical Status Classification System
- Evaluate Airway

**Cardiac Risk Assessment**

**Pulmonary Risk Assessment**
- Risk Factors for Pulmonary Complications
- Goals to Reduce Risks
- Ways to Decrease Complications

**Hepatic Risk Assessment**

**Renal Risk Assessment**
- Preoperative Evaluation
- Dialysis

**Hematological Assessment**
- Preoperative Labs
- Anemia

**Cardiac Risk Assessment**

**Pulmonary Risk Assessment**

**Hepatic Risk Assessment**

**Renal Risk Assessment**

**Hematological Assessment**

**Nutritional Assessment**

**Antibiotic Prophylaxis**

**General Postoperative Complications**

**Common Complications**
- Ileus
- Clostridium difficile colitis
- dvt/Pe
- Wound Infection

**Instructions to Patient**
- nPo
- bowel Preparation
- Usual Medications
Preoperative Evaluation

- Thorough history and physical exam.
- Optimization of any medical problems (i.e., cardiac or pulmonary diseases).

Anesthetic History

Note any prior anesthesics and associated complications.

The ASA Physical Status Classification System

See Anesthesia chapter.

Evaluate Airway

Mallampati Classification (Figure 1-1) predicts difficulty of intubation. Test is performed with the patient in the sitting position, the head held in a neutral position, the mouth wide open, and the tongue protruding to the maximum.

- Class I: Visualization of soft palate, fauces, uvula, anterior and posterior tonsillar pillars.
- Class II: Visualization of soft palate, fauces, uvula.
- Class III: Visualization of soft palate, base of uvula.
- Class IV: Nonvisualization of soft palate.

Cardiac Risk Assessment

- For a patient greater than 40 years old with no cardiac history: Obtain electrocardiogram (ECG); if normal, no further workup required.
- For a patient of any age with cardiac history, or for an older patient: Obtain ECG; consider stress test and echocardiogram.

Goldman’s risk assessment for noncardiac surgery (see Figure 1-2).

Pulmonary Risk Assessment

Risk Factors for Pulmonary Complications

- Known pulmonary disease.
- Abnormal pulmonary function tests (PFTs) (FEV < 11, maximum breathing capacity < 50% predicted).
- Smoking.
- Age > 60.
- Obesity.

Figure 1-1. Mallampati classification of ease of intubation.
WARD TIP

Echocardiography:
- Sensitivity 90–100%
- Specificity 50–80%
- Fixed defects, or defects that persist with time, indicate infarcted or scarred tissue.
- Reversible defects are more concerning: Normal and fixed defects have similar negative predictive values for cardiac events.

WARD TIP

Major abdominal surgery decreases vital capacity by 50% and functional residual capacity by 30%.

WARD TIP

Heparinduced thrombocytopenia (HIT).

WARD TIP

Because of the method by which they work (accentuate systemic thrombolysis) one SCD should work as well as two if one leg is injured.
cAutions

- Watch for prolonged elevations in drug levels in patients with preoperative liver dysfunction.
- Acute hepatitis is relative contraindication to surgery.
- Attempt to control ascites prior to elective surgery, with fluid restriction, diuretics, and nutritional therapy.

Renal Risk Assessment

Preoperative evaluation

- Check blood urea nitrogen (BUN) and creatinine.
- Estimate preoperative creatinine clearance (Cockcroft Gault equation):
  \[
  \left(\frac{140 - \text{age}}{\text{Ideal body weight in kilograms}}\right) \times \frac{72}{\text{Plasma creatinine (mg/dL)}}
  \]
- Maintain intravascular volume.
- Ensure electrolytes are repleted; correct acidosis.
- Dialysis patients should be dialyzed within 24 hours prior to surgery to best control creatinine, electrolytes, and uremic platelet dysfunction.

Diagnosis

- Overall mortality for dialysis-dependent patients: 5% (even when dialyzed within 24 hours of surgery).
- Acute renal failure that develops in perioperative period requiring dialysis is associated with a mortality of approximately 50–80%.
- Morbidity: Shunt thrombosis, pneumonia, wound infection, hemorrhage.

Hematological Assessment

Preoperative labs

- Check complete blood count (CBC).
- Blood should be typed and crossed.

Anemia

- Determine cause.
- Postpone elective operations.
- Patients with chronic hypoxia, ischemic heart disease, or cerebral ischemia do not tolerate anemia well.
- Sickle cell patients have increased risk of vaso-occlusive crises with operations. (This increased risk does not include patients with sickle cell trait.)

Thrombocytopenia

See Table 1-2.
**Table 1-2.** risk of postoperative Bleeding by platelet count

<table>
<thead>
<tr>
<th>Platelets</th>
<th>Likelihood of Bleed Perioperatively</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 150,000</td>
<td>Normal</td>
</tr>
<tr>
<td>100,000–150,000</td>
<td>Unlikely</td>
</tr>
<tr>
<td>50,000–100,000</td>
<td>Unlikely with adequate hemostasis</td>
</tr>
<tr>
<td>20,000–50,000</td>
<td>Possible excessive surgical bleeding</td>
</tr>
<tr>
<td>10,000–20,000</td>
<td>Spontaneous mucosal and cutaneous bleeding</td>
</tr>
<tr>
<td>&lt; 10,000</td>
<td>Major spontaneous mucosal bleed, including GI tract</td>
</tr>
</tbody>
</table>

**CoA GAL oPA t hy**

- Check prothrombin time (PT) and partial thromboplastin time (PTT) preoperatively.
- Note that elevated values should be expected in patients with liver disease.
- Factor abnormalities should be addressed (e.g., with hemophilia).

**Nutritional Assessment**

- Ideal body weight (IBW) = 50 kg + 2.3 kg/inch over 5 ft (male) or 45.5 kg + 2.3 kg/inch over 5 ft (female).  
  - Body mass index (BMI) = kg/m².  
  - Loss of > 10% body weight in 6 months or a serum albumin level of < 3 is a poor prognostic indicator.

**Antibiotic Prophylaxis**

**by type of surgery**

1. In general: Cefazolin.  
2. Gastrointestinal (GI) surgery: Cefazolin and metronidazole.  
3. Urologic procedures: Ciprofloxacin.  
4. Head and neck: Cefazolin or clindamycin and gentamicin.

**General Postoperative Complications**

- Immediate (0–24 hours):  
  - Primary hemorrhage—either starting during surgery or following postoperative increase in blood pressure: Replace blood loss, and may require return to OR to reexplore.  
  - Basal atelectasis.  
  - Shock: Blood loss, acute MI, arrhythmias, pulmonary embolism (PE).  
  - Oliguria: (hypotension intra- or postoperatively).

---

**ExAm TIP**

The risk of bleeding increases in patients with BUN > 100 due to platelet dysfunction, which can be corrected by desmopressin (DDAVP).

---

**WARD TIP**

To determine source of a renal problem:  
- $\text{FE}_\text{Na} > 1 =$ intrinsic damage  
- Specific gravity = 1.010 in ATN  
- $U_{\text{Na}} < 20$ in prerenal.

---

**WARD TIP**

Risk of bleeding is further increased at any platelet level if patient is septic or has a functional platelet deficit.  
- One unit of platelets raises platelet count by 5,000–10,000.
Early (2 days–3 weeks):
- Mental status changes due to electrolyte imbalance, dehydration, and sepsis.
- Nausea and vomiting: Analgesia or anesthesia related; paralytic ileus.
- Fever.
- Secondary hemorrhage (infection).
- Pneumonia.
- Wound infection.
- Wound or anastomotic dehiscence.
- DVT, PE.
- Acute urinary retention.
- Urinary tract infection.
- Postoperative wound infection.
- Bowel obstruction due to fibrinous adhesions.
- Paralytic ileus.

Late (weeks to months):
- Bowel obstruction due to fibrous adhesions.
- Incisional hernia.
- Nonhealing wound.
- Recurrence of reason for surgery (e.g., malignancy).

### Common Complications

#### ileus
- Incidence after GI surgery: 5%.
- Resolves with bowel rest and time in most patients.
- Check electrolytes! Hypokalemia can be a cause.

#### Cl o s t r i d i u m d i f f i C i l e c o l i t i s
- Variable presentation, from mild diarrhea and discomfort to severe pain, tenderness, fever, and elevated white blood count (WBC).
- Associated with age, prior residence in nursing home, renal failure, immunocompromised state, antibiotic use, GI surgery, nasogastric tube (NGT) for > 48 hours.
- Treatment: Metronidazole or PO vancomycin.

1. Atelectasis
   - Most common cause of fever within 24 hours of surgery.
     - Reduced by early ambulation.
     - Physical exam shows reduced breath sounds in bilateral lung bases.
     - Treated by incentive spirometry and chest PT; no role of antibiotics.

2. Pneumonia
   - Characterized by fever, cough, dyspnea, pleuritic chest pain, purulent sputum.
   - Exam shows bronchial breath sounds, dullness, rales.
   - Chest x-ray shows infiltrates; sputum culture demonstrates causative organism.
   - Treated by organism-specific antibiotics.

#### dvt / Pe
- PE presents with tachycardia, tachypnea, and hypoxia when symptomatic (see Table 1-3).
Prevented by SCDs, subcutaneous heparin, and ambulation.

PE diagnosed by ventilation/perfusion (V/Q) scan or chest computed tomography (CT) angiography.

Treated by heparin infusion for segmental, sub-segmental PE. Massive PE is treated by surgery (embolectomy).

**Wound Infection**

See Wounds chapter.

**Instructions to Patient**

**nPo**

- To decrease the risk of aspiration with intubation, patients should refrain from solids 6–8 hours prior and from liquids 2–3 hours prior to surgery.
- In bowel surgery, when patients require bowel preps, the duration of NPO may be preceded by a day of clear liquids only with the prep to clear the bowel of stool and facilitate the operation.

WARD TIP

To simplify instructions, we tell patients to be NPO after midnight the night prior to elective surgery.

WARD TIP

Remember that the bowel prep is a source of iatrogenic fluid loss. Elderly or chronically ill patients may not tolerate this loss without IV fluid replacement.
**bowel Preparation**

- **Types:**
  - Mechanical prep: Facilitates operation.
  - Oral antibiotics (neomycin, erythromycin base, metronidazole): Nadir bacterial count at commencement of operation if doses given at 1 p.m., 2 p.m., 11 p.m., the day prior (for a 7 a.m. case).

**usual medications**

- **Aspirin:** Avoid for 10 days preoperatively to allow platelets to regenerate.
- **Hold clopidogrel (Plavix) for 7 days.**
- **Warfarin (Coumadin):**
  - Three options: Avoid 3 days prior to operation and resume postoperative day 2; admit preoperatively and change to heparin, which can be held 2–4 hours ahead; operate through Coumadin.
- **Antihypertensives:** Continue, especially β-blockers; hold diuretics the morning of surgery.
- **Antithyroid medications:** Hold on morning of surgery.
- **Thyroid replacement:** Give on morning of surgery.
- **Oral hypoglycemics:** Avoid on day of surgery.
- **Insulin:** Give half usual dose on morning of surgery.

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**WARD TIP**

Aspirin and NSAIDs inhibit platelet activity and exacerbate bleeding. Holding Plavix in patients with drug-eluting cardiac stents may cause acute MI.

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**WARD TIP**

1/2 thyroxine = 7 days so it can be held for several postoperative days without much effect.
# High-Yield Facts in Wounds

## Introduction

## Steps of Wound Healing
- Coagulation
- Inflammation
- Collagen Synthesis
- Angiogenesis (granulation)
- Epithelialization
- Contraction and Remodeling

## Surgical Wound Classification
- Clean
- Clean-Contaminated
- Contaminated
- Dirty/Infected

## Types of Wound Healing
- Primary (first) Intention
- Second Intention
- Third (delayed primary) Intention

## Factors Affecting Wound Healing

## Wound Infections
- Classification
- Pathophysiology

## Common Surgical Pathogens
- Signs and Symptoms of Wound Infections

## Antimicrobial Prophylaxis
- General Principles

## Other Wound Complications
- Hematoma
- Seroma
- Wound Failure (dehiscence and incisional hernia)
- Complications of CesaSS Scar Formation
Introduction

By definition, the end result of any surgical case is wound healing. Surgeons define a successful surgical case as one in which the patient survives, the pathology is removed and/or corrected, and the patient's wound heals. To accomplish this, it is important to understand the processes involved in wound repair, and ways in which these processes can lead to complications.

Steps of Wound Healing

- Coagulation
- Inflammation
- Collagen synthesis
- Angiogenesis
- Epithelialization
- Contraction

Coagulation

- Begins following wound formation.
- Coagulation and complement cascades are activated, and platelets create a hemostatic plug.
- Release of various inflammatory mediators from activated platelets sets the stage for the steps that follow.
- Impaired by anticoagulants, antiplatelet agents, and coagulation factor deficiency. Also may see impaired coagulation in the setting of chronic liver disease and uremia.

Inflammation

- The signs of inflammation are pain, swelling, heat, erythema, and loss of function.
- Inflammation occurs as a result of the wound’s being invaded by polymorphonuclear neutrophils (PMNs, from the initial wound through the first 48 hours), and macrophages (peak numbers at 24 hours).
- Macrophages are essential for wound healing.
- Bacteria, cellular debris, and other foreign materials are also cleared from the wound site by the macrophages and PMNs.
- Impaired by steroids and other immunosuppressants, congenital, or acquired immune-deficient states.

Collagen Synthesis

- Occurs by fibroblasts in the vicinity of the wound, in response to various growth factor peptides.
- The amino acids hydroxyproline and hydroxylsine are components of collagen. Their synthesis and hydroxylation is dependent on Fe, α-ketoglutarate, and ascorbate (vitamin C).

Angiogenesis (Granulation)

- Occurs in response to peptide growth factors such as vascular endothelial growth factor (VEGF).
- Presence of these new vascular networks is what gives granulation tissue its characteristic beefy-red appearance.

WARD TIP

The presence of granulation tissue is reassuring evidence that the healing process is under way. At 1 month postinjury, the wound is at approximately 85% of its ultimate cohesive strength.

EXAM TIP

Phases of wound healing:
Hemostasis and Inflammation
Proliferation
Maturation
Remodeling

EXAM TIP

Macrophages are essential for wound healing.
**Epithelialization**

- Occurs with the migration of epithelial cells over the wound defect.
- Integrity of the basement membrane is restored as type IV collagen and other matrix components are deposited.
- Foreign bodies, such as suture material, and necrotic tissue remain separated from the wound by the migrating epithelial cells.
- Once this step has occurred, the wound is essentially waterproofed.

**Contraction and remodeling**

- Process by which the surrounding uninjured skin is pulled over the wound defect and the size of the scar is reduced.
- Made possible by the action of myofibroblasts, which possess a contraction mechanism similar to that seen in muscle cells.
- A long process that takes many months before it is complete.
- Do not confuse wound contraction with scar contracture, as the latter occurs after wound repair has ceased. Scar contracture can lead to undesirable effects since architecture of the surrounding tissue may become distorted.
- Maturation of the scar occurs over the next 9 months to 2 years, characterized by cross-linking of collagen and clinical flattening of the scar.

**Surgical Wound Classification**

**Clean**

For a wound to be considered “clean,” the following must be true:

- Wound created in a sterile and nontraumatic fashion, in an area that is free of preexisting inflammation.
- The respiratory, alimentary, genital, or urinary tract was not entered.
- All persons involved in the case maintained strict aseptic technique.

**Clean-Contaminated**

- The respiratory, alimentary, genital, or urinary tract was entered, but there was no significant spillage of its contents (e.g., feces), and there was no established local infection.
- There was only a minor break in aseptic technique.

**Contaminated**

- There was gross spillage from the gastrointestinal tract during the procedure.
- The genitourinary and biliary tracts were entered in the presence of local infection (e.g., cholangitis).
- The wound was the result of recent trauma.
- There was a major break in aseptic technique.

**Dirty/Infected**

- The wound was the result of remote trauma and contains devitalized tissue and/or purulent material.
- There is established infection or perforated viscera prior to the procedure.
Types of Wound Healing

**Primary (First) Intention**

- Type of healing seen following closure of clean surgical wounds, or traumatic lacerations in which there is minimal devitalized tissue, and minimal contamination.
- Edges of the incisional defect are approximated with the use of sutures or staples.
- Since the defect is very small, reepithelialization occurs rapidly, and overall healing time is short.
- Wounds closed primarily may have their dressing changed after 24–48 hours. By this time, epithelialization should be complete.
- Wound strength reaches 70–80% that of normal skin at 3 months.

**Secondary Intention**

- Type of healing seen following closure of wounds that are not approximated with sutures.
- Reason for not using sutures may be (1) that the wound edges cannot be apposed because the defect is very large (e.g., donor site of skin graft) or (2) that the surgeon chooses not to close the wound primarily because of the high risk of infection.

WARD TIP

Generally, clean traumatic lacerations are closed with sutures or staples (primary intention) if less than 6–8 hours old.

Figure 2-1. Different clinical approaches to the closure and healing of acute wounds.

Wounds healing by second intention should be packed loosely with moist gauze and covered with a sterile dressing. The wound should be assessed daily or the development of granulation tissue and the presence of infection.

Wound closes by contraction and then epithelialization. Contraction occurs from the edges of the wound inward, via contraction of myofibroblasts. Epithelialization then proceeds at 1 mm per day, also from the edges inward. Granulation tissue is generated at the center of the closing wound.

**third (delayed primary) intention**

- Type of healing seen following closure of wounds in which there is obvious gross contamination at the incisional site (i.e., the wound is classified as contaminated or dirty).
- An example of where delayed primary closure is often used is following removal of a ruptured appendix. In such cases, the parietal peritoneum and fascial layers are closed, and antibiotics are administered. The skin and subcutaneous tissue are not sutured until 3–5 days later after bacterial contamination has decreased.

### Factors Affecting Wound Healing

See Table 2-1.

### Wound Infections

**Classification**

Surgical site infections (SSIs) generally occur within 30 postoperative days. Depending on their location, they may be classified as superficial incisional, deep incisional, or organ/space infections.

- **Superficial incisional SSIs** exist when there is involvement by infection of the skin and subcutaneous tissue in the vicinity of the incision.
- **Deep incisional SSIs** exist when there is involvement by infection of deeper soft tissues, such as fascia or muscle, that were divided by the incision.
Organ/space SSIs exist when there is infection of any anatomical structure remote from the incisional site but manipulated during the procedure.

Early SSIs that occur in the first 24 hours postoperatively are most commonly due to Streptococcus or Clostridium. These bacteria grow very fast because they excrete enzymes that digest local tissue and impair host defenses. Infections due to other bacteria generally become apparent later (4–5 days postoperatively) because they lack such virulence factors.

Pathophysiology

Depends on:
- Factors relating to microorganism:
  - The critical number is $10^5$ (dose of contaminating microorganisms required to define it as a wound infection).
  - Factors relating to the patient—increased risk with:
    - Infection that is remote from the surgical site.
    - Diabetes.
    - Smokers.
    - Immunosuppressive agents, such as corticosteroids.
    - Severe protein–calorie malnutrition.
    - AIDS, disseminated malignancy, and any other immunocompromised state.
- Factors relating to surgical technique:
  - Preoperative considerations:
    - Patient skin preparation and the administration of antimicrobial prophylaxis.
  - Intraoperative considerations:
    - Strict attention to aseptic technique.
    - OR should be well ventilated and maintained under positive pressure to prevent the entrance of pathogens from the corridor.
  - Postoperative considerations:
    - Proper wound management and discharge instructions are key.
    - Wound management will differ depending on whether the wound is closed by primary, secondary, or delayed primary intention (see above).

Common Surgical Pathogens

- The organism responsible for causing an SSI is best identified by culturing the involved region.
- The most likely causative organism can be predicted based on the site of the operation:
  - *Staphylococcus aureus* and coagulase-negative staphylococci are commonly isolated from wounds that follow thoracic (cardiac and noncardiac), neurological, breast, ophthalmic, vascular, and orthopedic surgery.
  - *Gram-negative bacilli and anaerobes* are commonly isolated from wound infections that develop following appendectomy, colorectal, biliary tract, OB/GYN, and urological cases.
  - *Streptococci and oropharyngeal anaerobes* are commonly isolated from wounds that follow head and neck procedures in which the mucosa of the oral cavity is involved.
  - Animal bite infections: Pasteurella multocida; also Streptococcus and Staphylococcus.
**Signs and Symptoms of Wound Infection**

- Classic signs:
  - Calor (heat, warmth)
  - Rubor (redness)
  - Tumor (swelling)
  - Dolor (pain)
- More severe infections may produce systemic symptoms such as fever, chills, and rigors.
- Unusual for a wound infection to cause fever before postoperative day 3.
- Other causes of postoperative fever include urinary tract infection (UTI) (owing to prolonged placement of a Foley catheter), deep vein thrombosis (DVT)/thrombophlebitis, and certain medications.

**Diagnostic Approach**

- Physical exam should be directed at the surgical wound, looking for signs of infection such as induration, warmth, erythema, or frank purulent discharge.
- Any discharge present should be sent for microbiological culture.
- The febrile patient should be evaluated with an appropriate fever workup: complete blood count (CBC), blood and urine cultures, urinalysis, and chest x-ray should be sent.
- If an intra-abdominal abscess is suspected, a computed tomography (CT) scan of abdomen should be done.
- Lumbar puncture may be required for the patient with fever and altered mental status, especially in a patient that is status post craniotomy for a neurosurgical procedure.

**Treatment**

- Wound abscesses (superficial SSIs) require incision and drainage followed by thorough irrigation.
- Deeper SSIs may require surgical debridement.
- Systemic antibiotic therapy is required for deep SSIs; they may or may not be required for superficial SSIs, depending on the severity of the infection.
- Peritoneal abscesses (organ/space SSI) may be treated by CT-guided percutaneous drainage; those that cannot be drained percutaneously require open drainage.

**Antimicrobial Prophylaxis**

The benefits of surgical antimicrobial prophylaxis are maximized if the chosen antibiotic:

- Provides appropriate coverage against the most probable contaminating organisms.
- Is present in optimal concentrations in serum and tissues at the time of incision.
- Is maintained at therapeutic levels throughout the operation.

**General Principles**

- For gram-positive cocci: First- and second-generation cephalosporins.
- For gram-negative rods: Third-generation cephalosporins.
- For anaerobes: Metronidazole or clindamycin.
- For gram-negative rods: Aminoglycosides.
- For methicillin-resistant Staphylococcus aureus: Vancomycin.
- All choices of antibiotics should be tailored based on the resistance profile of the community your hospital serves.

### Other Wound Complications

#### Hematoma

- Collection of blood that may form in the vicinity of a surgical wound.
- Small hematomas may be left alone and allowed to reabsorb spontaneously, while larger hematomas may require drainage.

#### Seroma

- Collection of fluid in the vicinity of a wound that is not blood or pus.
- Due to creation of a potential space combined with disruption of local draining lymphatic channels (e.g., mastectomy).
- Small seromas may be left alone and allowed to reabsorb spontaneously, while larger ones may require aspiration.

### Wound Failure (Dehiscence and Incisional Hernia)

#### Definitions

- Occurs when there has been complete or partial disruption of one or more layers of the incisional site.
- Termed dehiscence if it occurs early in the postoperative course before all stages of wound healing have occurred (complete disruption).
- Termed incisional hernia when it occurs months or years after the surgical procedure (at least the skin is intact, i.e., partial disruption).

#### Causes

**Poor operative techniques** that may lead to wound failure include the following:

- Suture material with inadequate tensile strength. Since absorbable sutures lose their tensile strength rather quickly, nonabsorbable sutures should be used to close the fascia.
- Inadequate number of sutures. Sutures should be placed no greater than 1 cm apart; if placed greater than 1 cm apart, herniation of viscera may occur between sutures.
- Too small bite size. Sutures should be placed no less than 1 cm from the wound edge; if placed closer to the wound edge, the fascia may tear.
- Stitches tied too tight (ischemia).

**Patient factors** may be divided into:

1. Systemic illnesses that impair wound healing, such as malnutrition, corticosteroid therapy, sepsis, uremia, liver failure, or poorly controlled diabetes.
2. Physical factors that place stress on the incisional site, such as coughing/retching, obesity, and the presence of ascites.
Treatment

- Immediate treatment of wound dehiscence involves minimizing contamination of the operative site by the placement of sterile packing. The patient must then be brought back to the OR to reclose the incision.
- Incisional hernia must be treated promptly, especially if the patient is symptomatic (e.g., abdominal pain, nausea, vomiting). This is because strangulation of the bowel may occur, resulting in necrosis and increased morbidity. Incisional hernias are repaired by repairing the fascial defect, with or without the use of a synthetic mesh to reinforce the defect.

Complications of excess scar formation

Hypertrophic scar and keloid formation (both are raised above skin level):

- Keloids spread beyond the margins of the original wound and are painful.
- Common in African-Americans (genetic predisposition).
- Commonly seen around the earlobes and the deltoid, presternal, and upper back regions.
- Hypertrophic scars usually subside spontaneously, whereas keloids need treatment with intralesional corticosteroid injection, topical application of silicone sheets, or the use of radiation or pressure. Surgery is reserved for excision of large lesions or as second-line therapy when other modalities have failed.
Acute Abdomen

Definition

History

Physical Exam

Diagnosis

Management

Surgical Causes of Abdominal Pain

Important Nonsurgical Causes of Abdominal Pain

High-Yield Facts in Acute Abdomen

- Right Lower Quadrant (rLQ) 39
- Left Lower Quadrant (LLQ) 39
- Left Upper Quadrant (LUQ) 39
- Diffuse
- Periumbilical
- Suprapubic

- Right Upper Quadrant (rUQ) 38
- Important Nonsurgical Causes of Abdominal Pain 41

35
Acute Abdomen

**Definition**

Abrupt onset of abdominal pain usually accompanied by one or more peritoneal signs (i.e., rigidity, tenderness (with or without rebound), involuntary guarding). Most causes of acute abdomen are surgical.

**History**

Pain is the most common presenting feature of an acute abdomen. Special attention to the characteristics of the pain will aid in reaching the diagnosis.

**Location**

Visceral pain: Poorly localized, usually dull, achy pain arising from distention or spasm in hollow organs. Example: Crampy pain felt during early intestinal obstruction.

- Mid-epigastrium: Stomach, duodenum, hepatobiliary system, pancreas.
- Lower abdomen: Colon, internal reproductive organs.

Parietal pain: Sharp, well-localized, somatic pain arising from irritation (usually by pus, bile, urine, or gastrointestinal secretions) of the parietal peritoneum. Example: Infamed appendix causing sharp right lower quadrant (RLQ) pain due to irritation of nearby peritoneum.

**Quality of Pain**

Steady pain is most common, but differentiating character of pain is helpful:

<table>
<thead>
<tr>
<th>Gradual, steady pain</th>
<th>Intermittent, colicky pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute cholecystitis</td>
<td>Small bowel obstruction</td>
</tr>
<tr>
<td>Acute cholangitis</td>
<td>Inflammatory bowel disease</td>
</tr>
<tr>
<td>Hepatic abscess</td>
<td>Biliary colic</td>
</tr>
<tr>
<td>Diverticulitis</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Abrupt, excruciating pain</th>
<th>Rapid-onset, severe constant pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perforated ulcer</td>
<td>Acute pancreatitis</td>
</tr>
<tr>
<td>Ruptured aneurysm</td>
<td>Ectopic pregnancy</td>
</tr>
<tr>
<td>Ureteral colic</td>
<td>Mesenteric ischemia</td>
</tr>
<tr>
<td></td>
<td>Strangulated bowel</td>
</tr>
<tr>
<td></td>
<td>Acute appendicitis</td>
</tr>
</tbody>
</table>

**Precipitating or Palliative Factors**

May include:

- Change in position.
- Association with food (better, worse).
- Pain that wakes one from sleep (significant).

**Radiation**

- Biliary tract pain may radiate to the right shoulder or right scapula (due to right hemidiaphragmatic irritation).
- Splenic rupture pain may radiate to left shoulder.
- Kidney pain may radiate from flank to groin and genitalia (loin to groin).
- Pancreas pain may radiate to back.
**Associated Symptoms**
- Vomiting: Pain relieved by vomiting is supportive of small bowel obstruction (SBO), afferent loop syndrome.
- Bilious vomiting is a clue for proximal SBO.
- Bowel habits: Constipation or obstipation (no stool or flatus) is suggestive of bowel obstruction.
- Mucoïd diarrhea with blood (red currant jelly stool) is seen in intussusception.
- Anorexia: Very nonspecific symptom; however, most patients with acute appendicitis will have anorexia.
- Fever: Seen in appendicitis, acute cholecystitis, diverticulitis, acute pancreatitis, pyelonephritis.

**Medical and Surgical History**
- Past abdominal surgery? Any abdominal surgery increases the chance of SBO secondary to adhesions (even years later).
- Atrial fibrillation: Increased risk for mesenteric ischemia (embolism) because of emboli to mesenteric arteries.
- Menstrual and sexual history (acute salpingitis vs. pelvic inflammatory disease vs. ruptured ectopic).

**Physical Exam**

Things to look for:
- Vital signs: Most patients with a surgical abdomen will have vital sign abnormalities secondary to pain, inflammation, fluid and electrolyte derangements, and anxiety.
- General: Hydration status, mentation, nutritional status.
- Chest: Auscultation.
- Abdomen: See Tables 3-1 and 3-2.

**Diagnosis**

Initial laboratory evaluation should include:
- CBC.
- Electrolytes.
- Amylase, lipase.

---

**WARD TIP**
Serial abdominal exams and observation may be necessary in cases in which the etiology of abdominal pain is initially unclear.

---

**WARD TIP**
- Pain of perforated ulcer is severe and of sudden onset.
- Murphy's sign is seen in cholecystitis.

---

**WARD TIP**
Contrast for abdominal CT:
- For the most optimal imaging, both oral and IV contrast is used. In some cases (such as impaired renal function or allergy to IV contrast) this is not feasible. Noncontrast CT, although suboptimal for most cases (except nephrolithiasis), still provides lots of information.

---

**Table 3-1: Steps in Physical Examination of the Acute Abdomen**

<table>
<thead>
<tr>
<th>Step</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Inspection</td>
</tr>
<tr>
<td>2.</td>
<td>Auscultation</td>
</tr>
<tr>
<td>3.</td>
<td>Cough tenderness</td>
</tr>
<tr>
<td>4.</td>
<td>Percussion</td>
</tr>
<tr>
<td>5.</td>
<td>Guarding or rigidity</td>
</tr>
<tr>
<td>6.</td>
<td>Palpation</td>
</tr>
<tr>
<td></td>
<td>One finger</td>
</tr>
<tr>
<td></td>
<td>Rebound tenderness</td>
</tr>
<tr>
<td></td>
<td>Deep</td>
</tr>
<tr>
<td>7.</td>
<td>Punch tenderness</td>
</tr>
<tr>
<td>8.</td>
<td>Costal area</td>
</tr>
<tr>
<td>9.</td>
<td>Costovertebral area</td>
</tr>
<tr>
<td>10.</td>
<td>Special signs</td>
</tr>
<tr>
<td>11.</td>
<td>External hernias and male genitalia</td>
</tr>
<tr>
<td>12.</td>
<td>Rectal and pelvic examination</td>
</tr>
</tbody>
</table>

Table 3-2. Physical Findings in Various Causes of Acute Abdomen

<table>
<thead>
<tr>
<th>Condition</th>
<th>Helpful Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perforated viscus</td>
<td>Scaphoid, tense abdomen; diminished bowel sounds (late); loss</td>
</tr>
<tr>
<td></td>
<td>of liver dullness; guarding or rigidity.</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>Motionless; absent bowel sounds (late); cough and rebound</td>
</tr>
<tr>
<td></td>
<td>tenderness; guarding or rigidity.</td>
</tr>
<tr>
<td>Inflamed mass or</td>
<td>Tender mass (abdominal, rectal, or pelvic); punch tenderness;</td>
</tr>
<tr>
<td>abscess</td>
<td>special signs (Murphy’s, psoas, or obturator).</td>
</tr>
<tr>
<td>Intestinal obstruction</td>
<td>Distention; visible peristalsis (late); hyperperistalsis (early) or</td>
</tr>
<tr>
<td></td>
<td>quiet abdomen (late); diffuse pain without rebound tenderness;</td>
</tr>
<tr>
<td></td>
<td>hernia or rectal mass (some).</td>
</tr>
<tr>
<td>Paralytic ileus</td>
<td>Distention; minimal bowel sounds; no localized tenderness.</td>
</tr>
<tr>
<td>Ischemic or</td>
<td>Not distended (until late); bowel sounds variable; severe pain but</td>
</tr>
<tr>
<td>strangulated bowel</td>
<td>little tenderness; rectal bleeding (some).</td>
</tr>
<tr>
<td>Bleeding</td>
<td>Pallor, shock; distention; pulsatile (aneurysm) or tender (e.g.,</td>
</tr>
<tr>
<td></td>
<td>ectopic pregnancy) mass; rectal bleeding (some).</td>
</tr>
</tbody>
</table>


- Electrocardiogram (ECG) to rule out myocardial infarction (MI) and also as a preoperative baseline cardiac assessment.
- Liver function tests (LFTs) for right upper quadrant (RUQ) pain.
- β-hCG (human chorionic gonadotropin) for all women of childbearing age.
- Chest x-ray (CXR) and abdominal x-ray (AXR) to look for free air (can detect as little as 1–2 mL); easier to see under right hemidiaphragm. Presence of stomach bubble obscures it on the left (see Figure 3-1).
- Abdominal computed tomography (CT) should be used after the above assessment is complete and the diagnosis remains elusive (e.g., young male with clinical signs and symptoms of appendicitis should not undergo CT) (see Figure 3-2).

Management

- Early diagnosis improves outcome.
- Key is deciding whether surgical intervention is needed (see Table 3-3).

Surgical Causes of Abdominal Pain

Perforated duodenal ulcer
Acute cholecystitis
Hepatic abscess

WARD TIP

The pain of appendicitis localizes to McBurney's point.
Physical exam signs in appendicitis:
- Rovsing's, obturator, psoas (see Appendix chapter).

WARD TIP

Appendicitis is still the most common surgical emergency in the pregnant woman.
Retrocecal appendicitis
Appendicitis in a pregnant woman

**right Lower Quadrant (rLQ)**

Appendicitis
Cecal diverticulitis
Meckel’s diverticulitis
Intussusception
Ovarian pathology (torsion/ectopic pregnancy/tubo-ovarian abscess)

**Left Lower Quadrant (LLQ)**

Sigmoid diverticulitis
Volvulus
Ovarian pathology (torsion/ectopic pregnancy/tubo-ovarian abscess)

**Left Upper Quadrant (LUQ)**

Splenic rupture
Splenic abscess

**Diffuse**

Bowel obstruction
Leaking aneurysm
Mesenteric ischemia

---

**WARD TIP**

Patients with splenic rupture will have an elevated white count in the setting of trauma.

**WARD TIP**

Patients with bowel obstruction will initially be able to take in fluids by mouth, and vomit a short time afterward.

**EXAM TIP**

Abdominal aortic aneurysm (AAA): Pulsatile mass on physical exam.
**Figure 3-2. Abdominal anatomy.** Normal abdominopelvic CT scan of a 26-year-old man. Both oral and intravenous contrast was administered. A. Liver (right and left lobes), stomach, and spleen. B. Liver, gallbladder, kidneys, and pancreas. C. Kidneys, pancreas, and intestines. D. Small bowel, cecum, ascending colon, and normal appendix. E. Intestines and ureters at level of iliac wings. F. Bladder, distal ureters, and rectum at the level of the acetabular domes. (Reproduced, with permission, from Schwartz DT, Reisdorf EJ, eds. Emergency Radiology. New York: McGraw-Hill, 2000: 519.)
Early appendicitis
Pain from SBO

Ovarian pathology (torsion/ectopic pregnancy/tubo-ovarian abscess)
Psoas abscess
Incarcerated groin hernia
Sigmoid diverticulitis

Important Nonsurgical Causes of Abdominal Pain

Myocardial infarction
Mittelschmerz
Poisoning (lead, black widow spider)
Herpes zoster
Lower lobe (RLL) pneumonia
Endocrine (Addisonian crisis, diabetic ketoacidosis)
Sickle cell crisis
Hypercalcemia
Porphyrias
Psychological (hysteria)

WARD TIP

Fitz–Hugh–Curtis syndrome is perihepatitis associated with chlamydial infection of cervix.

WARD TIP

M/E: Do an ECG on all patients presenting with midepigastric pain.

WARD TIP

Pain of pancreatitis is described as boring radiating straight to the back.
Trauma

Advanced Trauma Life Support (ATLS)
“Golden Hour” of Trauma
Primary Survey
a BCs
Resuscitation
Intravenous Fluid
Shock
Radiologic and Diagnostic Studies
Secondary Survey
Tetanus Prophylaxis
Head Trauma
Neck Trauma
General
\[\text{ana}Tomy\]
Penetrating Injuries
\[\text{G}e\text{nu}HSG\]
\[\text{M}ana\ G\text{e}n\text{e}m\text{T}\]
Spinal Trauma
Thoracic Trauma
Pericardial Tamponade
\[\text{B}u\text{n}T\ Card\text{i}c\ T\text{rauma}\]
Pneumo Thorax
Tension Pneumo Thorax
Hemo Thorax
Abdominal Trauma
General

Physical Examination
Focused Abdominal Sonography for Trauma (FAST)
Diagnostic Peritoneal Lavage (d PL)
CT Scanning
\[\text{a}G\text{o}G\text{ra}Phy\]
Serial HemaTo CRITs
\[\text{l}a\text{P}aro\ SC\text{opy}\]
Indications for Exploratory Laparotomy
Types of Injury; General & Pneumothorax
Liver Injury
Splenic Trauma
Bowel Injury
Pancreatic Injury
General
\[\text{d}a\text{G}n\text{oo}SS\]
\[\text{T}rat\text{e}m\text{nT}\]
Genitourinary (GU) Trauma
General
SgS and Symptoms
\[\text{U}r\text{i}naly\text{SS}\]
\[\text{R}e\text{r}To\text{G}rade\ U\text{r}e\text{H}roG\text{ram}\]
\[\text{R}e\text{r}To\text{G}rade\ C\text{ys}toG\text{ram}\]
\[\text{Ba}dder\ Rup\text{h}ure\]
Ureteral Injury
\[\text{R}e\text{n}al\ Injury\]
Advanced Trauma Life Support (ATLS)

“Golden Hour” of Trauma

Period immediately following trauma in which rapid assessment, diagnosis, and stabilization must occur.

Primary Survey

Initial assessment and resuscitation of vital functions. Prioritization based on ABCs of trauma care.

aBCs

- Airway (with cervical spine precautions)
- Breathing and ventilation
- Circulation (and Control of hemorrhage)
- Disability (neurologic status)
- Exposure/Environment control
- Foley/Fingerstick/FAST exam

Airway and C-spine

- Assess patency of airway.
- Use jaw thrust or chin lift initially to open airway.
- Clear foreign bodies or suction airway if necessary.
- Insert oral or nasal airway when needed.
- Obtunded/unconscious patients should be intubated (GCS < 8).
- Surgical airway = cricothyroidotomy—used when unable to intubate.
- Expanding hematoma, emphysema in the neck, noisy breathing, and soot around nares (inhalation injury) all require considering intubation.

Breathing and Ventilation

- Inspect, auscultate, and palpate the chest.
- Ensure adequate ventilation and identify and treat injuries that may immediately impair ventilation:
  - Tension pneumothorax
  - Flail chest and pulmonary contusion
  - Massive hemothorax
  - Open pneumothorax

Control of Hemorrhage

- Place two large-bore peripheral (14- or 16-gauge) IVs.
- Consider intraosseous line placement or central line sheath introducers in patients with difficult access.
- Assess circulatory status (capillary refill, pulse, skin color) (see Shock section).
- Control life-threatening hemorrhage using direct pressure.

Disability

- Rapid neurologic exam.
- Establish pupillary size and reactivity and level of consciousness using the AVPU or Glasgow Coma Scale.
**Exposure/Environment/Extras**

Completely undress all trauma patients.

**Foley Catheter**

- Placement of a urinary catheter is considered part of the resuscitative phase that takes place during the primary survey.
- Foley is contraindicated when urethral transection is suspected, such as in the case of a pelvic fracture. If transection suspected, perform retrograde urethrogram before Foley.

**Signs of urethral Transection**

- Blood at the meatus.
- A “high-riding” prostate.
- Perineal or scrotal hematoma.
- Be suspicious with any pelvic fracture.

**Resuscitation**

- Begins during the primary survey.
- Life-threatening injuries are tended to as they are identified.

**inTravenous fluid**

- Fluid therapy should be initiated with 1 L of an isotonic (either lactated Ringer’s or normal saline) crystalloid solution.
- Consider early use of blood and blood products in patients with signs of shock.
- Pediatric patients should receive an IV bolus of 20 cc/kg.
- After 2 boluses, patients that continue to be hypotensive should receive blood products until stabilized.

**ShoCk**

- Inadequate delivery of oxygen on the cellular level secondary to decreased end-organ perfusion.
- In traumatic situations, shock is the result of hemorrhage until proven otherwise.

See Critical Care chapter.

**radiological diagnostic Studies: adjuncts to Primary Survey**

- X-rays of the chest and pelvis usually occur concurrently with early resuscitative efforts; however, this procedure should never interrupt the resuscitative process.
- Diagnostic peritoneal lavage (DPL) and focused abdominal sonogram for trauma (FAST) are also tools used for the rapid detection of intra-abdominal bleeding that often occurs early in the resuscitative process (see Abdominal Trauma).
- CT scans should be done only for patients who are hemodynamically stable.

**WARD TIP**

Don’t forget to keep your patients warm (cover them up again as soon as possible).

**WARD TIP**

Examine prostate and genitalia before placing a Foley.

**WARD TIP**

Trauma resuscitation requires teamwork with many different activities overlapping in both time and space.

**WARD TIP**

The antecubital fossae are a good place to find nice veins in which to place large-bore IVs.

**WARD TIP**

Use warmed fluids whenever possible.

**WARD TIP**

Stages of shock with % of blood loss

- Think of it as a tennis match:
  - Stage 1: Up to 15%
  - Stage 2: Up to 30%
  - Stage 3: Up to 40%
  - Stage 4: > 40% (game over)
Secondary Survey

**Trauma History**
- Begins once the primary survey is complete and resuscitative efforts are well under way. Whenever possible take an **AMPLE** history:
  - Allergies
  - Medications/Mechanism of injury
  - Past medical history/Pregnant?
  - Last meal
  - Events surrounding the mechanism of injury
- Head-to-toe evaluation of the trauma patient; frequent reassessment is key.
- Neurologic examination including the GCS (see Table 23-2), procedures, radiologic examination, and laboratory testing take place at this time if not already accomplished.

**TeTanus ProPHylaxis**
Immunize as needed.

**Head Trauma**
See Neurosurgery chapter.

**Neck Trauma**

**General**
- Described in broad terms as penetrating vs. blunt injuries even though considerable overlap exists between the management of the two.

**anatomy**
- The neck is divided into **zones** (see Figure 4-1):
  - Zone I spans the area from the thoracic inlet to the cricoid cartilage.
  - Zone II lies between cricoid and the angle of the mandible.
  - Zone III lies above the angle of the mandible to the base of the skull.
- These divisions help drive the diagnostic and therapeutic management decisions for penetrating neck injuries.

**Penetrating injuries**
- Any injury to the neck in which the platysma is violated.

**Vascular Injuries**
- Very common and often life threatening.
- Can lead to exsanguination, hematoma formation with compromise of the airway, and cerebral vascular accidents (e.g., from transection of the carotid artery or air embolus).
Nonvascular Injuries
- Injury to the larynx and trachea including fracture of the thyroid cartilage, dislocation of the tracheal cartilages and arytenoids, for example, leading to airway compromise and often a difficult intubation.
- Esophageal injury does occur and, as with penetrating neck injury, is not often manifest initially (very high morbidity/mortality if missed).

Resuscitation
- Obtain soft-tissue films or CT scan of the neck for clues to the presence of a soft-tissue hematoma and subcutaneous emphysema, and a chest x-ray (CXR) for possible hemopneumothorax.
- Surgical exploration is indicated for:
  - Expanding hematoma
  - Subcutaneous emphysema
  - Tracheal deviation
  - Change in voice quality
  - Air bubbling through the wound
- Pulses should be palpated to identify deficits and thrills, and auscultated for bruits.
- A neurologic exam should be performed to identify brachial plexus and/or central nervous system (CNS) deficits as well as Horner’s syndrome.

Management
- Most Zone II penetrating injuries require exploration in the OR.
- Injuries to Zones I and III may be taken to OR or managed conservatively using a combination of angiography, bronchoscopy, esophagoscopy, gastrografin or barium studies, and computed tomography (CT) scanning.

WARD TIP
- Avoid unnecessary manipulation of the neck as this may dislodge a clot.

WARD TIP
- Keep cervical in-line stabilization until C-spine fracture has been ruled out.

WARD TIP
- Tracheostomy is the procedure of choice in the patient with laryngotracheal separation.

WARD TIP
- Control of hemorrhage in the emergency department is via direct pressure (no blind clamping).
### Spinal Trauma

See Neurosurgery chapter.

### Thoracic Trauma

#### Pericardial Tamponade
- Life-threatening emergency usually seen with penetrating thoracic trauma, but may be seen with blunt thoracic trauma as well.
- Signs include tachycardia, muffled heart sounds, jugular venous distention (JVD), hypotension, and electrical alternans on electrocardiogram (ECG) (see Figure 4-2).
- Diagnosis may be confirmed with cardiac ultrasound (usually as part of focused assessment with sonography for trauma [FAST] exam).
- Requires immediate decompression via needle pericardiocentesis, pericardial window, or thoracotomy with manual decompression.

#### Blunt Cardiac Trauma
- Usually secondary to motor vehicle collision (MVC), fall from height, crush injury, blast injury, direct violent trauma.
- Screen for blunt cardiac injury with ECG.

#### Pneumothorax

**Definition**

Air in the pleural space.

**Signs and Symptoms**
- Usually asymptomatic.
- Chest pain
- Dyspnea.
- Hyper resonance of affected side.
- Decreased breath sounds on affected side.

**Diagnosis**
- Upright CXR is ~83% sensitive and demonstrates an absence of lung markings where the lung has collapsed (see Figure 4-3).
- Lung ultrasound, while operator dependent, is increasingly being used to detect pneumothorax.

---

**WARD TIP**

Never blindly probe a neck wound as this may lead to bleeding in a previously quiescent wound.

**ExAm TIP**

Beck’s tamponade triad:
1. Hypotension
2. JVD
3. Muffled heart sounds

**WARD TIP**

Clinically apparent tamponade may result from acute accumulation of as little as 60–100 mL of blood in the pericardial space.

**OR TIP**

1. Pericardium should be opened anterior and parallel to phrenic nerve (longitudinally).
2. Lacerations are repaired with 3-0 nonabsorbable suture and pledgets.
3. A Foley catheter with inflated balloon may be used as a temporizing measure to gain control for large defects.

---

*Figure 4-2.* ECG demonstrating electrical alternans. Note alternating heights of the R in the QRS complexes.
Tube thoracostomy: Chest tube placement should be confirmed by CXR.

**Te n Si o n  P n e u mo THo r a x**
- Life-threatening emergency caused by air entering the pleural space (most often via a hole in the lung tissue) and unable to escape.
- Causes total ipsilateral lung collapse and mediastinal shift (away from injured lung), impairing venous return and thus decreased cardiac output, eventually resulting in shock.

**Signs and Symptoms**
Same as for pneumothorax, plus tracheal deviation away from affected side (in tension pneumothorax).

**Treatment**
- Requires immediate needle decompression followed by tube thoracostomy.
- Needle decompression involves placing a needle or catheter over a needle into the second intercostal space, midclavicular line, over the rib on the side of the tension pneumothorax, followed by tube thoracostomy (chest tube).

**Hemo THo r a x**
The presence of blood in the chest.
- More than 200 cc of blood must be present before blunting of costophrenic angle will be seen on CXR.
- Treatment involves chest tube placement and drainage, and control of bleeding.

Figure 4-3.  

**F I G u r E  4 - 3 .  a. CXr demonstrating left-sided pneumothorax. (Note lack of lung markings.) B. Same patient, after tube thoracostomy and endotracheal intubation.**

**TRAUMA**

**WARD TIP**

The neurovascular bundle runs on the inferior margin of each rib.

**WARD TIP**

When the clamp enters the pleura, a rush of air or fluid should be obtained.
**Indications for Thoracotomy**
- 1,500 cc initial drainage from the chest tube.
- 200 cc/hr for 4 hours continued drainage:
  - Thoracic great vessel injury.
  - Esophageal injury.
  - Patients who decompensate after initial stabilization.

**Diagnostic Modalities**
- Angiography to localize injury and plan appropriate operation.
- CT scan for patients with normal initial CXR but suspicious mechanism and requiring CT for other reasons. If CT identifies injury, angiography still required for precise delineation of injury.
- Transesophageal echocardiogram (TEE):
  - Fast, no contrast required, concurrent evaluation of cardiac function, versatile in terms of location.
  - Contraindicated if potential airway problem or C-spine injury.
  - Not as sensitive or specific as angiography or CT scan.
  - User dependent.

**Definite Treatment**
- Surgical: Control bleeding, reconstruction, with graft if needed.
- Control hypertension pharmacologically.
- Endovascular stenting.
- Cardiac bypass as needed.
- Nonoperative: Close observation and pharmacologic treatment.

---

**Abdominal Trauma**

**General**
Penetrating abdominal injuries (PAIs; see Figure 4-4) resulting from a gunshot wound create damage via three mechanisms:
1. Direct injury by the bullet itself.
2. Injury from fragmentation of the bullet.
3. Indirect injury from the resultant “shock wave.”

PAIs resulting from a stabbing mechanism are limited to the direct damage of the object of impalement.
- Blunt abdominal injuries (BAIs) also have three general mechanisms of injury:
  1. Injury caused by the direct blow.
  2. Crush injury.

**Physical examination**

**Signs**
- **Seat-belt sign**—ecchymotic area found in the distribution of the lower anterior abdominal wall and can be associated with perforation of the bladder or bowel as well as a lumbar distraction fracture (Chance fracture).
- **Cullen’s sign** (periumbilical ecchymosis) is indicative of intraperitoneal hemorrhage.
- **Grey-Turner’s sign** (flank ecchymoses) is indicative of retroperitoneal hemorrhage.
- **Kehr’s sign**—left shoulder or neck pain secondary to splenic rupture. It increases when patient is in Trendelenburg position or with left upper quadrant (LUQ) palpation (caused by diaphragmatic irritation).

**General**
- Inspect the abdomen for evisceration, entry/exit wounds, impaled objects, and a gravid uterus.
- Check for tenderness, guarding, and rebound.

**Diagnosis**
- Perforation: AXR and CXR to look for free air.
- Diaphragmatic injury: CXR to look for blurring of the diaphragm, hemothorax, or bowel gas patterns above the diaphragm (at times with a gastric tube seen in the left chest).

**focused abdominal sonography for trauma (FAST)**
- Positive if free fluid is demonstrated in the abdomen.
- Has largely replaced DPL in most trauma centers.

**WARD TIP**
Bleeding usually stops spontaneously for low-velocity gunshot wounds and most stab wounds.

**WARD TIP**
Potential causes of iatrogenic great vessel injury:
- Central venous pressure (CVP) line or chest tube placement
- Intra-aortic balloon pump (IABP) placement
- Over inflation of Swan–Ganz balloon
Four views are utilized to search for free intraperitoneal fluid (presumed to be blood in the trauma victim) that collects in dependent areas and appears as hypoechoic areas on ultrasound (see Figures 4-5 and 4-6):
- Morrison’s pouch (RUQ): Free fluid can be visualized between the liver and kidney.
- Splenorenal recess (LUQ): Free fluid can be visualized between the spleen and kidney.
- Pouch of Douglas lies above the rectum (probe is placed in the suprapubic region).
- Subxiphoid and parasternal views to look for hemopericardium.

Advantages
- A rapid bedside screening study.
- Noninvasive.
- Not time consuming, can be repeated if necessary.
- Eighty to 95% sensitivity for intra-abdominal blood.

Disadvantages
- Operator dependent.
- Low specificity for individual organ injury.

WARD TIP
Traumatic aortic rupture is a high-mortality injury: Almost 90% die at the scene, and another 50% die within 24 hours.

ExAm TIP
Injury to innominate or subclavian arteries will result in absent or decreased upper extremity pulses and blood pressure (BP) with increased lower extremity BP.

![Imagery of ultrasound views](image-url)
Diagnosis Peritoneal Lavage (DPL)

Open DPL
Similar to open port placement in laparoscopic surgery (peritoneal cavity is entered under direct vision) using the Hassan port.

Closed DPL
Using the Seldinger technique, a catheter is placed through the needle and advanced into the peritoneum. The needle placement is similar to the closed technique of port placement in laparoscopy using the Veress needle.

Advantages
- Performed at bedside.
- Widely available.
- Highly sensitive for hemoperitoneum.
- Rapidly performed.

OR Tip
- Patient should be prepped and draped from sternum to knees to allow alternate access from the groin in the event of an emergency.
- Traditional approach (i.e., left anterolateral sternotomy) is used for the unstable patient with an undiagnosed injury.
- Angiography in the stable patient may dictate an alternate operative approach based on the location of injury.

Figure 4-6. abnormal FAST views. A. Liver–kidney view demonstrating presence of fluid (black stripe) in Morrison’s pouch compared to Figure 4-5A. B. Fluid in splenoral recess. C. Free fluid in the pelvis. D. Pericardial effusion (denoted as PE). (A, B, and D reproduced, with permission, from Jones RA, Welch R, Falcone R. Handbook of Trauma Ultrasonography. 1st ed. Bloomfield Hills, MI: Haines International; September 1999. C reproduced, with permission, from Melanson SW, Heller M. The emerging role of bedside ultrasonography in trauma care. Emerg Med Clin North Am 1998 Feb; 16(1):165-89.)
**Disadvantages**
- Invasive.
- Risk for iatrogenic injury (< 1%).
- Low specificity (many false positives).
- Does not evaluate the retroperitoneum.

**CT Scan**
- Useful for the hemodynamically stable patient.
- Has a greater specificity than DPL and ultrasound (US).
- Noninvasive.
- Relatively time consuming when compared with FAST.

**angiography**
- May be used to identify and embolize pelvic arterial bleeding secondary to pelvic fractures, or to assess blunt renal artery injuries diagnosed by CT scan.
- Otherwise limited use for abdominal trauma.

**Serial Hematocrits**
Serial hematocrits (every 4–6 hours) should be obtained during the observation period of the hemodynamically stable patient.

**Indications for Exploratory Laparotomy**
- Abdominal trauma and hemodynamic instability.
- Evisceration.
- Peritonitis.
- Diaphragmatic injury (see Figure 4-7).
- Hollow viscus perforation: Free intraperitoneal air.
- Intraperitoneal bladder rupture (diagnosed by cystography).
- Positive DPL.
- Surgically correctable injury diagnosed on CT scan.
- Removal of impaled weapon.
- Rectal perforation.
- Transabdominal missile (bullet) path (e.g., a gunshot wound to the buttoc with the bullet being found in the abdomen or thorax).

**Types of injury: General Approach**

**Liver Injury**
See Hepatobiliary System chapter.
Spleen Trauma

See Spleen chapter.

Bowel Injury

Stomach, Jejunum, and Ileum

- Isolated leaks from penetrating trauma lead to minimal contamination and patients usually do well if diagnosis is not delayed.
- Blunt injuries are “blowouts” resulting frequently from lap belts, and occur near the ligament of Treitz and the ileocecal valve.
- Mesentery can be significantly injured following blunt trauma.

Diagnosis:

- If the patient is awake and reliable, the exam is important to look for peritoneal irritation.
- If the exam is not reliable, DPL or laparoscopy may be required.
- CT scan has a high false-negative rate for small bowel injuries.
- Look for free air on upright CXR.
- Laparotomy for gastric or small bowel injury with primary repair and peritoneal lavage except in cases that have heavy soiling of the peritoneal cavity and present late, where intestinal diversion must be considered (e.g., ileostomy).
- Small bowel resection is needed where more than 50% of the bowel circumference is transected or several penetrating injuries are present within a very short segment of bowel where resecting is a better option than individually repairing each hole.

ExAm Tip

In a stable patient with neurologic dysfunction, whether from drugs, alcohol, head trauma, or baseline dementia, exam findings have a limited ability to direct care. These patients often require additional diagnostic tests.

WARD Tip

Serial abdominal examinations should be performed.

WARD Tip

DPL should be undertaken only after gastric and urinary decompression.
**Duodenum**
- **Mechanisms:** Three fourths of injuries result from penetrating trauma.
- **Diagnosis:**
  - Upper GI series with water-soluble contrast.
  - CT and DPL often miss duodenal injuries.
- **Treatment:**
  - Eighty percent of patients are able to undergo a primary repair.
  - Repair may be protected with an omental patch, jejunal serosal patch, and/or gastric diversion.
  - More complex injuries need pyloric exclusion or rarely pancreaticoduodenectomy (Whipple procedure).

**Large Bowel**
- Injuries generally occur via a penetrating mechanism (75% gunshot wound, 25% stab wound). Blunt injuries are rare but result from MVCs. Iatrogenic transanal injuries may also occur.
- **Signs and symptoms:** Abdominal distention, tenderness, guaiac-positive stool.
- **Diagnosis:**
  - In an awake and reliable patient, exam findings are consistent with peritonitis.
  - CXR may show free air.
  - In a patient with a flank injury but without clear peritoneal signs, consider a contrast enema.
- **Treatment:**
  - Primary repair: For small or medium-sized perforations, repair the perforation or, if needed, resect the affected segment and close with primary anastomosis. A proximal diverting stoma (e.g., ileostomy) is commonly placed.
  - Anastomosis is contraindicated in the setting of massive soiling.

**rectum**
- Two thirds are extraperitoneal.
- **Mechanism:** Majority by gunshot injury.
- **Diagnosis:**
  - DRE/guaiac: Suspicion increased by blood in stool or palpation of defect or foreign body on exam.
  - Rigid proctoscopy: May be done in OR if needed; mandatory for patients with known trajectory of knife or gunshot wound across pelvis or transanal; if patient is unstable, may be delayed until after resuscitation.
  - X-ray to look for missiles or foreign bodies.
- **Treatment:**
  - Diversion via colostomy is key.
  - Extraperitoneal injuries must be diverted via colostomy but may not need to be repaired (if not too big and not easily accessible).
  - Colostomy may be closed in 3–4 months.

**anus**
- Reconstruct sphincter as soon as patient is stabilized.
- Divert with sigmoid colostomy.
Pancreatic Injury

General
- **Mechanism:** Largely penetrating (gunshot wound > stab wound).
- Seventy-five percent of patients with penetrating injury to the pancreas will have associated injuries to the aorta, portal vein, or inferior vena cava.

Diagnosis
- Inspect pancreas during laparotomies performed for other indications.
- Check amylase and lipase.
- CT: Look for parenchymal fracture, intraparenchymal hematoma, lesser sac fluid, fluid between splenic vein and pancreatic body, retroperitoneal hematoma or fluid.
- Endoscopic retrograde cholangiopancreatography (ERCP): May be used in the stable patient if readily available or available intraoperatively; also may be used to evaluate missed injuries.

Treatment
- Nonoperative: May follow with serial labs and exam if patient can be reliably examined.
- Operative:
  - No ductal injury: Hemostasis and external drainage.
  - Distal transection, parenchymal injury with ductal injury: Distal pancreatectomy with duct ligation.
  - When duodenum or pancreatic head is devitalized, consider Whipple or total pancreatectomy.
  - Proximal transection/injury with probable ductal disruption:
    - If duct is spared, external drainage.
    - If duct is damaged, external drainage and pancreatic duct stenting may be considered.

Genitourinary (GU) Trauma

General
- Often overlooked in the initial evaluation of the multiply injured trauma patient.
- Diagnostic evaluation of the GU tract is performed in a “retrograde” fashion (i.e., work your way back from the urethra to the kidneys and renal vasculature).

Signs and Symptoms
- Flank or groin pain.
- Blood at the urethral meatus.
- Ecchymoses on perineum and/or genitalia.
- Evidence of pelvic fracture.
- Rectal bleeding.
- A “high-riding” or superiorly displaced prostate.

OR TIP
- Trauma laparotomy:
  - Midline incision.
  - Pack all four quadrants with laparotomy pads.
  - Evacuation of gross blood and clot.
  - Control bleeding.
  - Resuscitate as needed.
  - Systematically remove pads and inspect for source(s) of injury.
  - Definitive repair based on stability of patient and type of injury.

ExAm TIP
- Any wound from the nipple line to the gluteal crease can cause peritoneal or retroperitoneal injury.

OR TIP
- Have a low threshold for conversion to laparotomy during laparoscopy.

ExAm TIP
- Because of a common mechanism, Chance fractures and blunt small bowel injury are strongly associated. If one is present you should look for the other.

OR TIP
- To determine viability of the bowel in the OR, inject fluorescein dye IV and use a Wood’s lamp to inspect the bowel. Nonviable bowel will have patchy or no fluorescence.
Urinalysis
- The presence of gross hematuria indicates GU injury and often concomitant pelvic fracture.
- Urinalysis should be done to document presence or absence of microscopic hematuria.
- Microscopic hematuria is usually self-limited.

Retrograde Urethrogram
- Should be performed in any patient with suspected urethral disruption (before Foley placement).
- A preinjection KUB (kidneys, ureters, bladder) film should always be taken.

Retrograde Cystogram
- Should be performed on patients with gross hematuria or a pelvic fracture.
- Extravasation of contrast into the pouch of Douglas, paracolic gutters, and between loops of intestine is diagnostic for intraperitoneal rupture.
- Extravasation of contrast into the paravesicular tissue or behind the bladder is indicative of extraperitoneal bladder rupture.

Bladder Rupture

Intraperitoneal
- Usually occurs secondary to blunt trauma to a full bladder.
- Treatment is surgical repair.

Extrapitoneal
- Usually occurs secondary to pelvic fracture.
- Treatment is nonsurgical management by Foley drainage.

Ureteral Injury
- Least common GU injury (mostly iatrogenic).
- Must be surgically repaired.
- Diagnosed at the time of IVP or CT scan during the search for renal injury.

Renal Injury
- Commonly diagnosed by CT scan with contrast.
- Grade IV and V operative; the rest conservative (see Table 4-1 and Figure 4-8).
**TABLE 4-1.** Urologic Injury Scale of the American Association for the Surgery of Trauma

<table>
<thead>
<tr>
<th>Grade</th>
<th>Injury Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Contusion</td>
</tr>
<tr>
<td></td>
<td>Hematoma</td>
</tr>
<tr>
<td>II</td>
<td>Hematoma</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
</tr>
<tr>
<td>III</td>
<td>Laceration</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
</tr>
<tr>
<td>V</td>
<td>Laceration</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
</tr>
</tbody>
</table>


**FIGURE 4-8.** Organ injury scaling system for renal trauma. (Reproduced, with permission, from Feliciano DV, Mattox KL, Moore EE. Trauma. 6th ed. New York: McGraw-Hill; 2008: Fig. 39-8).

**WARD TIP**

Blood at the urethral meatus is virtually diagnostic for urethral injury and demands early retrograde urethrogram before Foley placement.

**WARD TIP**

Do not probe perineal lacerations as they are often a sign of an underlying pelvic fracture and disruption of a hematoma may occur.

**ExAm TIP**

Signs of arterial insufficiency: The 6Ps
- Pain
- Pullor
- Paresthesias
- Pulse deficit
- Poikilothermia
- Paralysis

**WARD TIP**

Rhabdomyolysis causes myoglobin release, which can cause renal failure. Maintaining a high urine output together with alkalization of the urine can help prevent the renal failure by reducing precipitation of myoglobin in the kidney.
High-Yield Facts in Critical Care

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**Shock**

**Definition**

Shock is defined as inadequate tissue perfusion. The delivery of oxygen and nutrients is not sufficient to maintain normal tissue and cellular function. Shock becomes irreversible if not treated early.

**Pathophysiology and Types of Shock**

Shock describes a state of imbalance between tissue substrate delivery (supply) and tissue substrate consumption (demand).

Tissue perfusion is determined by:

1. Cardiac Output (CO) = Stroke Volume (SV) × Heart Rate (HR)
2. Systemic Vascular Resistance (SVR) = [Mean Arterial Pressure (MAP) - Central Venous Pressure (CVP)] / [CO]
3. BP = CO × SVR

Deterioration in any one of these factors can cause hypotension and shock. Shock is classified into different types according to which of these factors are abnormal. The four major types of shock are hypovolemic, distributive (includes septic, anaphylactic, and neurogenic), cardiogenic, and obstructive.

**Note:** The first step in treatment of any type of shock is control of airway and breathing, next is restoration of circulation (ABC).

**Hypovolemic Shock**

- **Definition:** Decreased tissue perfusion secondary to rapid volume/blood loss (i.e., pre-load). CO is consequently decreased. The causes include bleeding, vomiting/diarrhea, and third spacing (e.g., from burns, bowel obstruction, pancreatitis, surgery).

**Signs and Symptoms**

Early on, patients will have tachycardia, orthostatic hypotension, and cool skin. As the condition progresses, they are hypotensive, have decreased pulse pressure (diastolic blood pressure becomes elevated), become confused, and have cold, clammy skin due to “clamping down” of peripheral vessels via increased sympathetic tone.

**Classification of Severity of Hypovolemic Shock**

- **Class I**—Compensated: Loss of < 15% of circulating blood volume. Little or no clinical manifestations.
- **Class II**—Partially compensated: Loss of 15–30% of blood volume. Manifestations include mild tachycardia, tachypnea, anxiety, orthostatic hypotension, decreased pulse pressure, and oliguria. Reduced splanchnic and renal blood flow.
- **Class III**—Uncompensated: Loss of 30–40% of blood volume. Hypotension, oliguria, marked tachycardia, and confusion.
- **Class IV**—Life threatening: Loss of > 40% of circulating blood volume. All of the above plus lethargy, mental status change, severe hypotension, and oliguria or anuria.

**WARD TIP**

Think ABCs with any patient in “shock”: Secure airway, breathing, and circulation.

**WARD TIP**

If the skin is warm, it is distributive shock. If the skin is cold and clammy, it is hypovolemic, obstructive, or cardiogenic shock. Shock with bradycardia is neurogenic unless proved otherwise.

**WARD TIP**

Shock in trauma or postop patient is assumed to be hypovolemic until proven otherwise.

**EXAM TIP**

- First sign of hypovolemic shock: tachycardia!

**WARD TIP**

Of the vital organs, the first “casualty” of hypovolemic or cardiogenic shock (both “cold shocks”) is the kidneys, as blood is shunted away from the constricted renal arteries. Therefore, it is crucial to monitor for renal failure. An adequate urine output is one of the crucial signs that the treatment is adequate.
treatment
- Rapid initial FLUID resuscitation! Crystalloids (normal saline [NS]/lactated Ringer’s [LR]) infusion via two large-bore peripheral IVs is best for volume repletion.
- Normal saline vs. lactated Ringer’s: Large-volume NS infusion may result in hyperchloremic acidosis. Therefore, LR (containing alternative anions to Cl-) is the preferred choice.
- Colloid vs. isotonic crystalloid: No evidence that colloid fluids improve mortality in critically ill patients.
- Class III and IV shock: Think about blood transfusion.
- Replacement of blood if hemorrhage is cause.
- Treat underlying cause (i.e., surgical correction if patient has ongoing hemorrhage). Failure to respond to fluid resuscitation is usually due to persistent massive hemorrhage, hence requiring emergent surgical procedure.

Distributive shock

Definition
A family of shock states that are caused by systemic vasodilation (i.e., severe decrease in SVR). They include septic shock (most common), neurogenic shock, and anaphylactic shock. These patients will have warm skin from vasodilation.

Septic Shock
Infection that causes vessels to dilate and become leaky, causing hypotension refractory to fluid resuscitation.

Lab/Physical Findings
- Physical exam findings—fever, tachypnea, warm skin and full peripheral pulses (from vasodilation), hypotension, poor urinary output.
- Positive blood cultures (negative about 50% of the time, particularly if drawn after antibiotics are started).

The Continuum
SIRS → Sepsis → Severe Sepsis → Septic Shock
Septic shock is the most severe manifestation of infection in a continuum. Milder manifestations of infection are classified as systemic inflammatory response syndrome (SIRS), sepsis, and severe sepsis (Figure 5-1 and Table 5-1).

Treatment
- Fluids!!
- Source control
- Antibiotics: Start broad-spectrum antibiotics early and empirically treat until blood cultures come back!!
  - Surgical drainage of abscess or focus of infection.
- If blood pressure is unresponsive to fluids, use pressors, classically norepinephrine (Levophed).
- Tight glycemic control (maintain blood glucose < 180).

Anaphylactic shock
Systemic type I hypersensitivity reaction causing chemically mediated edema and increased vascular permeability, resulting in hypotension and/or airway compromise.
Figure 5-1. Schematic depiction of physiologic consequences of septic insult.

Table 5-1. Clinical Spectrum of infection and Systemic inflammatory response Syndrome (SIRS)

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection</td>
<td>Identifiable source of microbial insult</td>
</tr>
<tr>
<td>SIRS</td>
<td>Two or more of following criteria</td>
</tr>
<tr>
<td></td>
<td>Temperature ≥ 38°C or ≤ 36°C</td>
</tr>
<tr>
<td></td>
<td>Heart rate ≥ 90 beats/min</td>
</tr>
<tr>
<td></td>
<td>Respiratory rate ≥ 20 breaths/min or Paco₂ ≤ 32 mmHg or mechanical ventilation</td>
</tr>
<tr>
<td></td>
<td>White blood cell count ≥ 12,000/µL or ≤ 4,000/µL or ≥ 10% band forms</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Identifiable source of infection + SIRS</td>
</tr>
<tr>
<td>Severe sepsis</td>
<td>Sepsis + organ dysfunction</td>
</tr>
<tr>
<td>Septic shock</td>
<td>Sepsis + cardiovascular collapse (requiring vasopressor support)</td>
</tr>
</tbody>
</table>

Salient Physical Findings
- Urticaria and anigœdema (especially around lips)
- Laryngeal edema (stridor), wheezing

Treatment
- Epinephrine
- Antihistamines (diphenhydramine)
- Steroids

NeuRogenic Shock
Central nervous system (CNS) injury causing disruption of the sympathetic system, resulting in unopposed vagal outflow and vasodilation. It is characterized by hypotension and bradycardia (absence of reflex sympathetic tachycardia and vasoconstriction). Usually secondary to spinal cord injury of cervical or high thoracic region.

Treatment
- IV fluids: Usually patients respond; helps to place patient in Trendelenburg position.
- Vasopressors: Used early if patient is unresponsive to fluids.

Cardiogenic Shock
Pump failure, resulting in decreased CO. This can be caused by myocardial infarction (MI), arrhythmias, valvular defects, heart failure, or cardiac contusion. Wedge pressure and SVR are elevated.

Findings
Patients will have cold, clammy skin from peripheral vasoconstriction. Additionally, they will have jugular venous distention (JVD), dyspnea, bilateral crackles, and S3/4 gallop. Chest x-ray (CXR) will show bilateral pulmonary congestion. Echocardiography demonstrates poorly contractile left ventricle, pulmonary capillary wedge pressure (PCWP) > 20 mmHg, cardiac index (CI) < 2.0.

Treatment
- Correct electrolyte abnormalities: Most commonly hypokalemia and hypomagnesemia.
- Pain control: IV morphine sulfate or fentanyl to minimize anxiety.
- Antiarrhythmics, cardiac pacing, or cardioversion for pathologic dysrhythmias or heart block.
- Treatment of cardiogenic shock is specific to cause:
  - Acute MI: Oxygen, nitroglycerin, aspirin, IV morphine, cardiology consultation.
  - Cardiac contusion: Infusion of inotropes. Intra-aortic balloon pump can provide temporary treatment.
- Left Heart Failure or Biventricular Failure:
  - Isolated left ventricular failure: Pressors, and reduce afterload.
  - Congestive heart failure: Diuretics and vasodilators (nitrates) to decrease preload. May or may not use pressors.
  - Classically, the following inotropes can be used:
    - Dobutamine: Used after dopamine to increase cardiac contractility.
    - Amrinone and milrinone (phosphodiesterase inhibitors): Used in patients unresponsive to dopamine or dobutamine.
  - Intra-aortic balloon pump (IABP): Used as mechanical support in patients who do not respond to pressors or inotropes. IABP increases...
Table 5-2: Types of Shock and their Hemodynamic Profiles

<table>
<thead>
<tr>
<th></th>
<th>PCWP</th>
<th>CO</th>
<th>SVr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemic shock</td>
<td>↓↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Cardiogenic/obstructive shock</td>
<td>↑</td>
<td>↓↓</td>
<td>↑</td>
</tr>
<tr>
<td>Neurogenic shock</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Distributive shock</td>
<td>↓ or normal</td>
<td>↑</td>
<td>↓↓</td>
</tr>
</tbody>
</table>

CO₂ and decreases work of heart by reducing systolic afterload and increasing diastolic perfusion to coronary arteries. Device can be inserted percutaneously via femoral artery at the ICU bedside.

- **Isolated right heart failure:**
  - Most commonly caused by right ventricular infarction or PE.
  - Give fluids (maintains preload).

See Table 5-2.

### Obstructive shock

Extracardiac causes of cardiac failure. They can be divided into pulmonary vascular etiologies (massive pulmonary embolism, severe pulmonary hypertension), or mechanical causes (tension pneumothorax, pericardial tamponade, constrictive pericarditis).

#### Findings

Patients will have cold clammy skin from peripheral vasoconstriction. They will have physical findings similar to patients with cardiogenic shock. CXR findings will vary depending on the reason for obstructive shock. Pulmonary embolism may not result in acute cardiopulmonary abnormalities. Pneumothorax will be visible on CXR. Pericardial effusion (if chronic) will present as an enlarged cardiac silhouette. Echocardiography would show pericardial effusions, and CT angiography of the chest will reveal the PEs Swan–Ganz catheter will be helpful to diagnose pulmonary hypertension.

#### Treatment

- Treatment of obstructive shock is specific to cause:
  - Pulmonary embolism: possible embolectomy, anticoagulation (heparin, Coumadin)
  - Pulmonary hypertension: pulmonary vasodilators (sildenafil, remodulin), afterload reduction
  - Cardiac tamponade: Percardiocentesis
  - Constrictive pericarditis: NSAID, steroids

### Swan–Ganz Catheter

The Swan–Ganz catheter (pulmonary artery catheter) is used with ICU and shock patients in order to obtain information relevant to fluid and volume status (it has never been shown to change mortality and its use is weaning). It is threaded via the subclavian or internal jugular into superior vena cava → right atrium → right ventricle → pulmonary artery.
The following are some of the measurements obtainable through the Swan–Ganz that will allow a better understanding of the different types of shock (Table 5-3):

- **Pulmonary artery occlusion pressure (PAOP or PCWP)** (normal 6–12 mmHg) This reflects the pressures of the left ventricle (end-diastolic pressure). It can be thought of as preload.
  
  **Clinical context:** If the pump fails, pressures in the left ventricle increase and you will have an increased wedge.

- **CO** (normal 4–8 mmHg) Remember, CO = SV × HR. The Swan–Ganz allows CO to be measured via the **thermodilutional technique:** The temperature change is measured at the distal end of the catheter when cold fluid is injected from the proximal port. The difference in temperature reflects CO, which can also be thought of as pump function.
  
  **Clinical context:** If you have an MI and lose wall motion, your SV will be decreased. Likewise, if you hemorrhage and have no preload, your SV will decrease as well.

- **SVR** (usually divided by body surface area to give systemic vascular resistance index [SVRI]): SVR reflects the vascular resistance across the systemic circulation (it can be thought of as afterload as well).
  
  **Clinical context:** Distributive shock causes vessels to dilate and leak, causing SVR to decrease. Cardiogenic, obstructive, and hypovolemic shock results in vasoconstriction, causing SVR to increase.

---

**Table 5-3. Normal ranges for Selected Hemodynamic Parameters in adults**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVP</td>
<td>0–6 mmHg</td>
</tr>
<tr>
<td>Right ventricular systolic pressure</td>
<td>20–30 mmHg</td>
</tr>
<tr>
<td>Right ventricular diastolic pressure</td>
<td>0–6 mmHg</td>
</tr>
<tr>
<td>PAOP</td>
<td>6–12 mmHg</td>
</tr>
<tr>
<td>Systolic arterial pressure</td>
<td>100–130 mmHg</td>
</tr>
<tr>
<td>Diastolic arterial pressure</td>
<td>60–90 mmHg</td>
</tr>
<tr>
<td>MAP</td>
<td>75–100 mmHg</td>
</tr>
<tr>
<td>Q₁</td>
<td>4–6 L/min</td>
</tr>
<tr>
<td>Q₇</td>
<td>2.5–3.5 L·min⁻¹·m⁻²</td>
</tr>
<tr>
<td>SV</td>
<td>40–80 mL</td>
</tr>
<tr>
<td>SVR</td>
<td>800–1,400 dyne·sec·cm⁻³</td>
</tr>
<tr>
<td>SVRI</td>
<td>1,500–2,400 dyne·sec·cm⁻³·m⁻²</td>
</tr>
<tr>
<td>PVR</td>
<td>100–150 dyne·sec·cm⁻³</td>
</tr>
<tr>
<td>PVRI</td>
<td>200–400 dyne·sec·cm⁻³·m⁻²</td>
</tr>
<tr>
<td>Caₒ₂</td>
<td>16–22 mL/dL</td>
</tr>
<tr>
<td>Cvo₂</td>
<td>~15 mL/dL blood</td>
</tr>
<tr>
<td>Do₂</td>
<td>400–660 mL·min⁻¹·m⁻²</td>
</tr>
<tr>
<td>Vo₂</td>
<td>115–165 mL·min⁻¹·m⁻²</td>
</tr>
</tbody>
</table>

Caₒ₂ = arterial oxygen content; Cvo₂ = central venous oxygen pressure; CVP = mean central venous pressure; Do₂ = systemic oxygen delivery; MAP = mean arterial pressure; PAOP = pulmonary artery occlusion (wedge) pressure; PVR = pulmonary vascular resistance; PVRI = pulmonary vascular resistance index; Q₁ = cardiac output; Q₇ = cardiac output indexed to body surface area (cardiac index); SV = stroke volume; SVI = stroke volume index; SVR = systemic vascular resistance; SVRI = systemic vascular resistance index; Vo₂ = systemic oxygen utilization.


---
Pressors and Inotropes

A group of vasoactive drugs that are the final line of defense in treating shock (Table 5-4).

effects and Side effects

Generally, pressors are used to increase CO or SVR. All of them have important side effects that can limit their use.

These side effects are easily predicted based on the drug’s action. For example, in addition to stimulating $\beta_1$ receptors, dobutamine stimulates $\beta_2$ (which causes vasodilation). The $\beta_2$ stimulation causes the side effect of hypotension.

Furthermore, remember that virtually any direct stimulation of the heart ($\beta_1$) can cause the side effect of arrhythmias.

Dobutamine

- **Action**: Strong stimulation $\beta_1$ receptors (ionotropic/chronotropic effects on the heart) with a mild stimulation of $\beta_2$ (vasodilation).
- **Result**: ↑ CO, ↓ SVR.
- **Typical use**: Cardiogenic shock.

isoPRoTereNol

Similar to dobutamine.

- **Action**: Strong stimulation of $\beta_1$ receptors (ionotropic/chronotropic effects on the heart) and $\beta_2$ (vasodilation).

### Table 5-4. Relative Selectivity of Adrenoceptor Agonists

<table>
<thead>
<tr>
<th><strong>alpha agonists</strong></th>
<th>$\alpha_1 &gt; \alpha_2 &gt;&gt;&gt; &gt;&gt;&gt; \beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenylephrine, methoxamine</td>
<td></td>
</tr>
<tr>
<td>Clonidine, methylnorepinephrine</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Mixed alpha and beta agonists</strong></th>
<th>$\alpha_1 = \alpha_2; \beta_1 &gt;&gt;&gt; \beta_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norepinephrine</td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Beta agonists</strong></th>
<th>$\beta_1 &gt; \beta_2 &gt;&gt;&gt; \alpha$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dobutamine</td>
<td></td>
</tr>
<tr>
<td>Isoproterenol</td>
<td></td>
</tr>
<tr>
<td>Terbutaline, metaproterenol, albuterol, ritodrine</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Dopamine agonists</strong></th>
<th>$D_1 = D_2 &gt;&gt; \beta &gt;&gt; \alpha$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td></td>
</tr>
<tr>
<td>Fenoldopam</td>
<td></td>
</tr>
</tbody>
</table>

Milrinone is technically not a pressor, but it is an important inotrope used in the ICU.

- **Action:** Phosphodiesterase inhibitor, which results in increased cyclic adenosine monophosphate (cAMP). This has positive ionotropic effects on the heart and also vasodilates.
- **Result:** ↑ CO, ↓ SVR.
- **Typical use:** Heart failure/cardiogenic shock.

**Dopamine**

Dopamine has different action depending on the dose.

**Low Dose (1–3 µg/kg/min): “Renal Dose”**

- **Action:** Stimulation of dopamine receptors (dilates renal vasculature) and mild β₁ stimulation.
- **Typical use:** None.

**Intermediate Dose (5–10 µg/kg/min): “Cardiac Dose”**

- **Action:** Stimulation of dopamine receptors, moderate stimulation of β₁ receptors (heart ionotropy/chronotropy), and mild stimulation of α₁ receptors (vasoconstriction).
- **Result:** ↑ CO.
- **Typical use:** Cardiogenic shock.

**High Dose (10–20 µg/kg/min)**

- **Action:** Stimulates dopamine receptors, β₁ receptors (heart ionotropy/chronotropy), and strong stimulation of α₁ receptors (vasoconstriction).
- **Result:** ↑↑ SVR.
- **Typical use:** Septic shock (replaced by norepinephrine).

**Norepinephrine**

- **Action:** Strong stimulation of α₁ receptors (vasoconstriction), moderate stimulation of β₁ receptors (heart ionotropy/chronotropy).
- **Result:** ↑↑ SVR, ↑ CO.
- **Typical use:** Septic shock.

**Epinephrine**

- **Action:** Strong stimulation of β₁ and β₂ receptors. Also α₁,2 stimulation (vasoconstriction).
- **Result:** ↑↑ SVR, + / - ↑ CO, bronchodilation.
- **Typical use:** Anaphylaxis, septic shock, cardiopulmonary arrest.
Phenylephrine

- **Action:** Strong stimulation of $\alpha_1$ receptors (vasoconstriction).
- **Result:** ↑↑ SVR.
- **Typical use:** Septic shock, neurogenic shock, anesthesia-induced hypotension.

### Mechanical Ventilation

The goals of mechanical ventilation are to:

1. Improve gas exchange.
2. Decrease the work of breathing.

**Indications for intubation:**

- Airway protection (patients with GCS < 8).
- Failure to oxygenate—hypoxia despite high oxygen delivery content and clinical signs of respiratory distress (excess work of breathing).
- Failure to ventilate leading to progressive hypercapnia with acidosis and signs of mental status change.

### Setting the Ventilator

See Table 5-5.

Several parameters have to be set for the ventilator. You need to define the mode, respiratory rate, tidal volume, and the FiO$_2$

- **Mode:** Choose assist control (AC), synchronized intermittent mandatory ventilation (SIMV), pressure support (PS), or continuous positive airway pressure (CPAP).
- **Respiratory rate** (for AC or SIMV only): Usually 10–20.
- **Tidal volume** (for AC or SIMV only): Usually 400–600 cc (6–8 cc/kg).
- **FiO$_2$:** Always start at 100% and titrate down, maintaining the pulse oximetry > 90%. Keep FiO$_2$ below 60% to minimize oxygen-induced free radical injury.

**Example**

**Initial AC mode setting used in the surgical patient:** AC 12–16, tidal volume 6–8 cc/kg (ideal body weight), FiO$_2$ = 100% (reduced after initial ABG), PEEP 5.

### Adjusting the Ventilator

1. Based on ABG (interpretation discussed in previous chapter).
2. Minute ventilation.

   \[
   \text{Minute Ventilation} = \text{Respiratory Rate} \times \text{Tidal Volume}
   \]

   You can therefore adjust either the rate or the tidal volume to change the minute ventilation.

   Know this simple rule: Increasing minute ventilation will decrease Pco$_2$ and increase pH. Decreasing minute ventilation will increase Pco$_2$ and decrease pH.

### Oxygenation

FiO$_2$ and PEEP are the ventilator parameters that adjust oxygenation.
### Various Modes of Mechanical Ventilation

| Continuous positive airway pressure (CPAP) | Noninvasive ventilation that applies constant positive pressure, with no variation in breathing cycle. Patient must breathe on his or her own. CPAP keeps inspiratory airway pressure above atmospheric pressure without increasing work of breathing; improves functional residual capacity and compliance. Can be used to help avoid intubation. Mode of choice for sleep apnea. |
| Synchronized intermittent mandatory ventilation (SiMV) | Patient breathes on his or her own, plus receives a preset rate of MV that is synchronized to and delivered with the patient’s breath. Pressure support is often added to spontaneous breathing (gives patient initial boost of pressure to overcome airway resistance). |
| Assist-control ventilation (aCV) (may be volume controlled or pressure controlled) | Each breath initiated by patient triggers the machine to deliver the set tidal volume (volume control) or set peak inspiratory pressure (pressure control). A set tidal volume/pressure is given a set number of times per minute, even if patient is breathing less than the preset respiratory rate. |
| Pressure support ventilation (PSV) | Each breath initiated by patient triggers the machine to deliver an initial “boost of pressure” with variable flow of air into lungs. The patient determines the rate, duration of inspiration, and tidal volume. This boost helps the patient overcome resistance of the endotracheal tube and reduces work of breathing. This mode can be used alone, as the only ventilator setting, or in conjunction with the IMV/SIMV modes. Pressure support is typically set at between 5 and 20. This mode is used in weaning of ventilator. |

### Acute Respiratory Distress Syndrome (ARDS)

Acute lung injury due to inflammatory process in both lungs causing increased permeability of the capillaries and severe ventilation/perfusion mismatch. ARDS is a disease of altered lung compliance. These patients are tachypneic and hypoxic and have bilateral crackles on lung exam.

The diagnostic criteria are:

1. Bilateral, fluffy infiltrates on CXR
2. PaO₂/FiO₂ ratio < 200.
3. No evidence of heart failure (PCWP ≤ 18 mmHg).
4. Acute onset.
5. Presence of an underlying cause.

---

**WARD TIP**

Pco₂ is the marker of ventilation. Po₂ is the marker of oxygenation.

**WARD TIP**

Try to titrate down the FiO₂ (< 50% is ideal) in order to avoid oxygen toxicity. Oxygen toxicity is thought to be caused by oxygen-free radicals damaging the lung interstitium.

**WARD TIP**

The CXR does not reliably distinguish ARDS from CHF. A Swan–Ganz catheter can be useful in this matter:
- PCWP < 18 = ARDS
- PCWP > 18 = CHF
There is a wide variety of causes of ARDS. Common causes include:

- Direct lung injury:
  - Pneumonia
  - Aspiration
  - Near drowning
- Indirect causes:
  - Sepsis (most common of all causes).
  - Massive transfusion.
  - Severe trauma, burns, toxins.
  - Pancreatitis.

**management**

1. It is necessary to intubate because of hypoxemia.
2. Treat underlying cause, especially infections.
3. **Low tidal volume ventilation (6–8 cc/kg).**
4. PEEP is often used to improve gas exchange and keep lungs open at relatively low lung volumes.
5. FiO₂ is kept at ≤ 60% to avoid free radical injury.
Anatomy of Body Fluids

ToTaL Body WateR

InTraCellular Fluid (ICF)

ExTraCellular Fluid (ECF)

Normal Fluid and Electrolyte Exchange

Water Movement Between ICF and ECF

Renal Control of Fluids/Electrolytes

Volumed eFlits (dehydration)

Volume Excess

Causes

Signs and Symptoms

TreatmenT

Ongoing Fluid Loss

Causes

Assessing Volume Status

Vital Signs

History and Physical Exam in HypovoleMic

History and Physical Exam in HypovoleMic

Input, Output, Weight

Urine Output (UO)

Lab

Sodium Balance

HyponatRemic

HypernatRemic

Potassium Balance

HyponatRemic

HypernatRemic

Calcium Balance

HyponatRemic

HypernatRemic

Indications for Nutritional Support

Total Parenteral Nutrition (TPN)

Nutrient Requirements

Caloric Requirements

Respiratory Quotient (RQ) of Individual Substrates

Protein Requirements

Enteral Nutrition

Complications

Acid–Base Disorders

Metabolic Acidosis

Metabolic Alkalosis

Respiratory Acidosis

Respiratory Alkalosis
Anatomy of Body Fluids

ToTal Body WaTer
- Fifty to 70% of total body weight.
- Greater in lean individuals because fat contains little water, average 60%.
- Greatest percentage in newborns 70%, then decreases with age to around 50%.
  - Example: Average 70-kg male would be 42 L water since 1 L of water = 1 kg.
- Made up of two compartments—ICF and ECF.

InTracelullar Fluid (ICF)
- Mostly in skeletal muscle mass, thus slightly lower in females (50%) than males (60%).
- Cell wall separates the ICF from the ECF and acts as a semipermeable membrane.

exTracelullar Fluid (ECF)
- Made up of plasma and interstitial (extravascular) fluid.
- Capillary membrane separates plasma and interstitial fluid and acts as a semipermeable membrane.

<table>
<thead>
<tr>
<th>Intracellular Compartment</th>
<th>Extracellular Compartment</th>
</tr>
</thead>
<tbody>
<tr>
<td>⅔ = 67% TBW</td>
<td>⅓ = 33% TBW</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Interstitial</td>
<td>Plasma</td>
</tr>
<tr>
<td>⅓ ECC = 25% TBW</td>
<td>⅓ ECC = 8% TBW</td>
</tr>
<tr>
<td>In 70-kg man (42 L TBW) = 28 L</td>
<td>11 L</td>
</tr>
<tr>
<td>In 45-kg woman (22.5 L TBW) = 15 L</td>
<td>5.6 L</td>
</tr>
</tbody>
</table>

Normal Fluid and Electrolyte Exchange

WaTer MoveMent BeTWeen IcF and eCF
Water flows freely between the three compartments, shifting compartments to maintain osmotic equilibrium between them (Figure 6-1). See Table 6-1.
### Table 6-1. Signs and Symptoms of Volume Disturbances

<table>
<thead>
<tr>
<th>System</th>
<th>Volume Deficit</th>
<th>Volume Excess</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generalized</td>
<td>Weight loss</td>
<td>Weight gain</td>
</tr>
<tr>
<td></td>
<td>Decreased skin turgor</td>
<td>Peripheral edema</td>
</tr>
<tr>
<td>Cardiac</td>
<td>Tachycardia</td>
<td>Increased cardiac output</td>
</tr>
<tr>
<td></td>
<td>Orthostasis/hypotension</td>
<td>Increased central venous pressure</td>
</tr>
<tr>
<td></td>
<td>Collapsed neck veins</td>
<td>Distended neck veins</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Murmur</td>
</tr>
<tr>
<td>Renal</td>
<td>Oliguria</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Azotemia</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Ileus</td>
<td>Bowel edema</td>
</tr>
<tr>
<td>Pulmonary</td>
<td></td>
<td>Pulmonary edema</td>
</tr>
</tbody>
</table>

Renal Control of Fluids/Electrolytes

See Figure 6-2.

- Distal tubules—reabsorption of Na in exchange for K and H secretion.
- Affected by adrenocorticotropic hormone (ACTH) and aldosterone.
- Aldosterone directly stimulates K secretion and Na reabsorption from the distal tubule.

**volume deficit (dehydration)**

Most common fluid disorder.

**causes**

**Losses that Mimic ECF**

- Hemorrhage.
- Loss of gastrointestinal (GI) fluid—vomiting, nasogastric (NG) suction, diarrhea, fistular drainage.
- Postoperative fluid sequestration (third spacing): Intestinal obstruction.
- Intra-abdominal and retroperitoneal inflammation (e.g., pancreatitis, peritonitis).
- Systemic inflammatory response syndrome (SIRS), burns, sepsis, pancreatitis.

**Losses that Are Principally Water**

- Fever.
- Osmotic diuresis.
- Diabetes insipidus.
- Prolonged water deprivation.
- Inadequate input during procedure.

**signs and symptoms**

See Table 6-1.

- Central nervous system (CNS) and cardiovascular (CV) signs occur early with acute loss.
- CV signs are secondary to a decrease in plasma volume.
- Tissue signs may be absent until the deficit has existed for 24 hours.

---

**Figure 6-2.** Renal mechanism of fluid and electrolyte balance.
Tissue signs may be difficult to assess in the elderly patient or patient with recent weight loss.

Body temperature varies with environment—cool room may mask fever.

After partial correction of volume deficit, the temperature will generally rise to the appropriate level.

Severe volume depletion depresses all body systems and interferes with the clinical evaluation of the patient.

Volume depleted patient with severe sepsis from peritonitis may be afebrile and have normal white blood count (WBC), complain of little pain, and have unremarkable findings on abdominal exam. This may change dramatically when the ECF is restored.

History items important for evaluating fluid deficits include:

- Weight change, intake (quantity and composition), output, general medical status.
- Degree of dehydration dependent on acute loss of body weight and is assessed clinically:
  - Mild—3% for adults, 5% for kids
  - Moderate—6% for adults, 10% for kids
  - Severe—9% for adults, 15% for kids

**Treatment**

- Goal is to replace this deficit in most patients over the next 24 hours.
- The amount of fluid the patient is missing needs to be combined with the expected maintenance fluid for the next 24 hours.
- Rehydration is done over this period of time to try to allow continual equilibration between the reexpanded intravascular space and the contracted ECF and ICF.
- The initial intervention is to give a large amount of fluid as a volume expander; 20 mL/kg of normal saline (NS) or Ringer’s lactate (LR) is given as a bolus. During the remaining 8 hours, the expected maintenance fluid is given plus about half of the remaining calculated loss. Over the remaining 16 hours, the other half of the remaining calculated loss is given along with the maintenance fluid.
- Volume expansion can be accomplished with crystalloid (NaCl, LR, etc.) or colloid (albumin, blood products).

**Crystalloid**

- Dextrose solutions are used to deliver free water to the body (dextrose is quickly metabolized).
- 0.9% NaCl quickly adds volume to the intravascular space.
- Goal is to expand the intravascular space.

**Colloids**

- Include packed RBCs, FFP, albumin.
- Stay mainly within intravascular space if the capillary membranes are intact.
- Expensive.
- Indications:
  - Hypovolemic patients with excess Na and water—such as ascites, congestive heart failure (CHF), postcardiac bypass patients.
  - Patients unable to synthesize enough albumin or other proteins to exert enough oncotic pressure—such as liver disease, transplant recipients, resections, malnutrition.
  - Severe hemorrhage or coagulopathy—packed red blood cells (PRBCs) and fresh frozen plasma (FFP) may increase hematocrit to help correct coagulopathy.

When replacing fluids, remember that:

- Large volumes may lead to peripheral and/or pulmonary edema.
- Large amounts of dextrose may cause hyperglycemia.
- Large amounts of NS may cause hyperchloremic metabolic acidosis.
- Ringer’s lactate given when patient is hypovolemic and in metabolic alkalosis (i.e., from NG tube, vomiting) may worsen the alkalosis when the lactate is metabolized.
Volume Excess

**causes**

**Isotonic**
- Iatrogenic—intravascular overload of IV fluids with electrolytes.
- Increased ECF without equilibration with ICF—especially postoperative or trauma when the hormonal responses to stress are to decrease Na and water excretion by kidney.
- Often secondary to renal insufficiency, cirrhosis, or CHF.

**Hypotonic**
- Inappropriate NaCl-poor solution as a replacement for GI losses (most common).
- Third spacing.
- Increased antidiuretic hormone (ADH) with surgical stress, inappropriate ADH (SIADH).

**Hypertonic**
- Most common cause: excessive Na load without adequate water intake:
  - Water moves out of the cells because of increased ECF osmolarity.
  - Causes an increase in intravascular and interstitial fluid.
  - Worse when renal tubular excretion of water and/or Na is poor.
  - Can also be caused by rapid infusion of nonelectrolyte osmotically active solutes such as glucose and mannitol.

**Signs and Symptoms**

See Table 6-2.

**Treatment**

- Restriction of Na and fluids for isotonic hypervolemia.
- Free water replacement for hypertonic hypervolemia.
- Saline for hypotonic hypervolemia.

**Table 6-2. Composition of Gastrointestinal Secretions**

<table>
<thead>
<tr>
<th>Type of Secretion</th>
<th>Volume (ml/24 h)</th>
<th>Na (meq/l)</th>
<th>K (meq/l)</th>
<th>Cl (meq/l)</th>
<th>HCO$_3^-$ (meq/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stomach</td>
<td>1,000–2,000</td>
<td>60–90</td>
<td>10–30</td>
<td>100–130</td>
<td>0</td>
</tr>
<tr>
<td>Small intestine</td>
<td>2,000–3,000</td>
<td>120–140</td>
<td>5–10</td>
<td>90–120</td>
<td>30–40</td>
</tr>
<tr>
<td>Colon</td>
<td></td>
<td>60</td>
<td>30</td>
<td>40</td>
<td>0</td>
</tr>
<tr>
<td>Pancreas</td>
<td>600–800</td>
<td>135–145</td>
<td>5–10</td>
<td>70–90</td>
<td>95–115</td>
</tr>
<tr>
<td>Bile</td>
<td>300–800</td>
<td>135–145</td>
<td>5–10</td>
<td>90–110</td>
<td>30–40</td>
</tr>
</tbody>
</table>

• Diuresis with furosemide 10–50 mg:
  - Be sure to replace K as needed.
  - Be careful not to over diurese—must maintain kidney and brain perfusion as well as appropriate cardiac output.

**Ongoing Fluid Loss**

• **Rule of thumb**: Replace one half of the “usual” ongoing losses along with the assumed maintenance and the rehydration replacement fluid.
• Electrolyte content of the ongoing loss can be either assumed based on serum electrolyte values or can be determined by direct electrolyte measurement of the fluid (see Table 6-2).

**Causes**

- Fever: Each °C above 37°C adds 2.0–2.5 mL/kg/day of insensible water loss.
- Loss of body fluids: From vomit, NG suction, fistulas.
- Third-space losses:
  - Adults—approximately 1 L of third-space fluid intra-abdominally for each quadrant of the abdomen that is traumatized, inflamed, or operated on.
  - Kids—approximately one fourth of calculated maintenance fluid per 24-hour period is sequestered for each quadrant of the abdomen that is traumatized, inflamed, or operated on.
- Burns:
  - See Burns chapter for estimating volume losses and replacement.
  - Osmotic diuresis:
    - Secondary to urea, mannitol, or glucose.
    - Urine electrolytes should be checked to determine the appropriate replacement fluid, if one is necessary.

**Assessing Volume Status**

**Vital signs**

- Early signs of hypovolemia: Tachycardia, decreased pulse pressure, orthostatic blood pressure (BP).
- BP is not persistently lowered until 20–30% of circulating volume is lost.

**History and Physical exam in Hyper Volemia**

- **Hx**: Weight gain, recent myocardial infarction (MI), shortness of breath, orthopnea.
- **PE**: Jugular venous distention (JVD), rales, S3, pitting edema, ascites.

**History and Physical exam in Hypovolemia**

- **Hx**: Weight loss, vomiting, diarrhea, burns.
- **PE**: Flat neck veins, poor tissue turgor, dry mucous membranes, cool extremities, slow capillary refill.
Daily weight is one of the best methods for assessing volume status.

**ur Ine ouTpuT (uo)**

- Normal UO: 0.5 cc/kg/hr for adults, 1 cc/kg/hr for kids.
- Low UO: Hypovolemia, renal failure, low flow states.
- High UO: Hypervolemia, diabetes insipidus, osmotic diuresis, post-obstructive diuresis.

**laB**

- Check daily serum electrolytes in intensive care unit (ICU) patients.
- Blood urea nitrogen (BUN)/creatinine (Cr) > 20 and FeNa < 1% indicates hypovolemia.
- BUN/Cr < 15 indicates adequate hydration.

**Sodium Balance**

Alterations in sodium balance cause fluid shifts between compartments. The clinical picture is most often neurologic, due to fluid shifts within the brain.

**hyponaTr eMla**

**definition**

- < 130 mEq/L.
- First steps in hyponatremia: Determine volume status clinically, then determine plasma osmolality!

**Step 1: Determine Plasma Osmolality**

- Normal osmolality—pseudohyponatremia: Lab artifact due to increased lipids or plasma proteins → next step; check lipid profile or possible multiple myeloma.
- High osmolality—pseudohyponatremia: Due to increase of osmotically active molecules—glucose or mannitol.
- Low osmolality—true hyponatremia.

**Step 2: Assess Volume Status**

- Hypovolemia
- Euvolemia
- Hypervolemia

See below for discussion of each.

**Hyponatremia with High Plasma Osmolality (Pseudohyponatremia)**

**Causes**

Hyperglycemia, either physiologic or due to rapid infusion of glucose or mannitol will cause increased osmotic pressure that shifts fluid from the ICF to the ECF. The total body sodium in this case is normal but has become diluted due to the fluid shift.

The expected Na concentration can be calculated as follows: For every 100 mg/dL that glucose is increased over 100 mg/dL, the Na concentration falls 1.6 mEq/L. Remember “sweet 16.”
For example, a patient with a glucose concentration of 500 mg/dL is expected to have a hyponatremia of around 133.6 mEq/L ($4 \times 1.6 = 6.4$, $140 - 6.4 = 133.6$).

**Hyponatremia with Hypotonicity (true Hyponatremia)**

True hyponatremia reflects excess ingestion of water that overwhelms the kidneys (either normal or diseased) or due to increased ADH. Hyponatremia is not due to increased excretion of sodium.

**Hypovolemia (dehydration)**
- Renal cause: Diuretics.
- Extrarenal cause: Vomiting, diarrhea, burns, pancreatitis.
- Differentiate using urine Na: Urine Na $< 20$ mEq/L indicates expected renal retention in the face of hypovolemia, suspect an extrarenal cause. Urine Na $> 20$ mEq/L indicates a renal cause.

**Hypervolemia**
- May be from CHF, cirrhosis, or nephrotic syndrome.
- Increased thirst and vasopressin.
- Edematous state.

**Euvolemia**
- SIADH: Most common cause of normovolemic hyponatremia.
- Increased vasopressin release from posterior pituitary or ectopic source causes decreased renal free water excretion.
- **Signs and symptoms:**
  - Hypo-osmotic hyponatremia (hyponatremia with hypotonicity).
  - Inappropriately concentrated urine (urine osmolality $> 100$ mOsm/kg).
  - Normal renal, adrenal, and thyroid function.
- **Causes:**
  - Neuropsychiatric disorders, malignancies (especially lung), and head trauma.
  - Glucocorticoid deficiency (Addison’s disease) — cortisol deficiency causes hypersecretion of vasopressin.
  - Hypothyroidism — causes decreased CO and glomerular filtration rate (GFR), which leads to increased vasopressin secretion.
  - Primary polydipsia — usually seen in psychiatric patients who compulsively drink massive volumes of water.

**Signs and Symptoms of (true) Hyponatremia**
- **Signs:** Decreased reflexes, respiratory depression, seizures, coma (see Table 6-3).
- **Symptoms:** Nausea/vomiting, headache, lethargy, muscle cramps.
- Hypovolemic hyponatremia: Give 0.9% NaCl. Na repletion with saline isotonic to the patient, in order to avoid rapid changes in ICF volume. Major complication from rapid correction of chronic hyponatremia is central pontine myelinolysis.
- Hypervolemic hyponatremia: Correct underlying disorder — CHF, liver or renal failure.
- Euvolemic hyponatremia: Raise plasma Na (lower ICF volume) — restrict water intake.

**Definition**

$> 145$ mEq/L.
Hypernatremia is always associated with hyperosmolarity. (Note that in the plasma osmolality equation, Na is the major factor).

**Causes**
- Loss of water (dehydration!): Diabetes insipidus, diuretics, sweating, GI loss, burns, fistulas.
- Gain of sodium due to excess mineralocorticoid activity: Primary hyperaldosteronism, Cushing’s, renal artery stenosis (hyperreninism), congenital adrenal hyperplasia (will cause concomitant hypokalemia).
- If thirst mechanism is intact and water is available, hypernatremia will not persist. Suspect hypernatremia in the young, elderly, and patients with altered mental status who may not have access to water.

**Symptoms**
- Thirst.
- Restlessness, weakness, delirium.
- Hypotension and tachycardia.
- Decreased saliva and tears.
- Red, swollen tongue.
- Oliguria.
**Treatment**
- Calculate FWD.
- If euvolesmic: Replace water deficit with D5W.
- If hypovolemic: Use NS. Correct one half of water deficit in first 24 hours; remaining water deficit over next 1–2 days.
- After euvolesmic, slow D5W infusion to correct hypernatremia at rate between 8 and 12 mEq/dL/24 hours. Any faster than 12 mEq/dL/day, and there is risk of cerebral edema.

---

**Potassium Balance**
- Ninety-nine percent of K is in ICF. Therefore, small alterations in extracellular K balance can have significant clinical effects, particularly impaired electrical signaling in the heart, muscle, and nerve (see Table 6-4). Proper proportions of K⁺ and Ca²⁺ must exist for their exchange across membrane channels that allow electrical conduction to occur.
- Cells act as a rapid potassium buffer. Kidney regulates long-term potassium control.

**Hyponatremia**
- Definition
  - < 3.5 mEq/L.

---

**Clinical Manifestations of Abnormalities in Potassium, Magnesium, and Calcium**

**Increased Serum Levels**

<table>
<thead>
<tr>
<th>System</th>
<th>Potassium</th>
<th>Magnesium</th>
<th>Calcium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>Nausea/vomiting, colic, diarrhea</td>
<td>Nausea/vomiting</td>
<td>Anorexia, nausea/vomiting, abdominal pain</td>
</tr>
<tr>
<td>Neuromuscular</td>
<td>Weakness, paralysis, respiratory failure</td>
<td>Weakness, lethargy, decreased reflexes</td>
<td>Weakness, confusion, coma, bone pain</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Arrhythmia, arrest</td>
<td>Hypotension, arrest</td>
<td>Hypertension, arrhythmia</td>
</tr>
<tr>
<td>Renal</td>
<td></td>
<td></td>
<td>Polyuria, polydipsia</td>
</tr>
</tbody>
</table>

**Decreased Serum Levels**

<table>
<thead>
<tr>
<th>System</th>
<th>Potassium</th>
<th>Magnesium</th>
<th>Calcium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>Ileus, constipation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neuromuscular</td>
<td>Decreased reflexes, fatigue, weakness, paralysis</td>
<td>Hyperactive reflexes, muscle tremors, tetany, seizures</td>
<td>Hyperactive reflexes, paresthesias, carpopedal spasm, seizures</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Arrest</td>
<td>Arrhythmia</td>
<td>Heart failure</td>
</tr>
</tbody>
</table>

causes
- Most commonly due to excessive renal secretion.
- Loss of potassium due to excess mineralocorticoid activity: Primary hyperaldosteronism, Cushing’s, renal artery stenosis (hyperreninism), congenital adrenal hyperplasia (will cause concomitant hypernatremia).
- Movement of K into cells due to insulin, catecholamines, alkalemia.
- Prolonged administration of K-free parenteral fluids.
- Total parenteral hyperalimentation with inadequate K replacement.
- Loss in excessive lower GI secretions such as diarrhea, colonic fistulas, VIPoma.
- Diuretics.

signs and symptoms
- Electrocardiogram (ECG) (see Figure 6-3): Flattened T waves, ST depression, U wave.
- Arrhythmias, signs of low voltage.

Treatment
- Check Mg level first as hypomagnesemia is commonly associated with hypokalemia and must be corrected before/along with hypokalemia.
- Amount of K to be replaced can be conservatively estimated as: (4.0 – current K) × 100, in mEq.
  - Example: if current K is 3.1, give 90 mEq (total, not all at once!!!)
- In asymptomatic patient with K > 3.0 mEq/L, oral K replacement may be sufficient.
- No more than 40 mEq should be added to a liter of IV fluid since rapid K administration can cause fatal arrhythmias.
- Rate should not exceed 40 mEq/hr.
- May cause a burning sensation if given in peripheral IV. Using low flow rate of 10 mEq/hr or adding a small amount of lidocaine to the solution can decrease discomfort.

hyperkalemia

definition
> 5 mEq/L.

causes
- Commonly due to renal failure.
- Rarely found when renal function is normal and usually causes a transient hyperkalemia, due to cellular shifts: Potassium spillage from cells in severe injury; cells take up hydrogen ions in exchange for intracellular potassium, acting as a buffer in states of acidosis.
- Drugs: Angiotensin-converting enzyme (ACE) inhibitors, potassium-sparing diuretics.
- Iatrogenic causes: Penicillin G contains 1.7 mEq K per 1 million units, KCl added to maintenance fluids, blood transfusion with old batch of packed red blood cells (PRBCs) where K may have leaked out of cells, overtreatment of hypokalemia.
- Digoxin toxicity can cause severe hyperkalemia by blocking the sodium–potassium–adenosine triphosphatase (ATPase) pump.
- Hypoaldosteronism.
- Pseudohyperkalemia: Can result when RBCs lyse in the test tube and release potassium. This is a lab error: Repeat test before treating!
Cardiac effects are most significant. Confirm hyperkalemia and obtain an ECG.

- **ECG:**
  - Early: Peaked T waves (see Figure 6-4), wide QRS, ST depression.
  - Late: Disappearance of T waves, heart block, sine wave—ominous for impending fatal arrhythmia, cardiac arrest.
- **GI:** Nausea, vomiting, intermittent intestinal colic, diarrhea.

**Treatment (in order of importance)**

- Ten percent calcium gluconate 1 g IV—monitor ECG. Calcium temporarily suppresses cardiac arrhythmias by stabilizing the cardiac membrane and should be administered first. Does not affect potassium load.
- Lower extracellular K\(^+\) (acute treatment): Albuterol, insulin with glucose or sodium bicarbonate promote cellular reuptake of K—transient relief of hyperkalemia.
- Kayexalate—cation exchange resin. As opposed to above measures, which immediately protect against dangers of high potassium, this actually removes the potassium from the body.
- Dialysis (last resort).

---

**Calcium Balance**

- Normal: 1,000–1,200 mg—most is in the bone in the form of phosphate and carbonate.
- Normal daily intake: 1–3 g.
- Most excreted via stool (~200 mg via urine).
- Normal serum level: 8.5–10.5 mg/dL (total calcium).
- Half of this is nonionized and bound to plasma protein.

If hypocalcemia is seen on laboratory report, first correct for low albumin:

Corrected Calcium = 0.8 \((\text{Normal Albumin} - \text{Observed Albumin}) + \text{Observed Calcium}\)
I corrected calcium alls within normal range, no action is required.

- Ionized calcium is the most accurate measure of calcium, but labs report total calcium.
- An additional nonionized fraction (5%) is bound to other substances in the ECF.
- Ratio of ionized to nonionized Ca is related to pH (see Figure 6-5):
  - Acidosis causes increase in ionized fraction.
  - Alkalosis causes decrease in ionized fraction.

**Hypocalcemia**

**Definition**

< 8 mg/dL.

**Causes**

- Acute pancreatitis.
- Massive soft-tissue infections (necrotizing fasciitis).
- Acute/chronic renal failure.
- Pancreatic/small bowel fistulas.
- Hypoparathyroidism (common after parathyroid or thyroid surgery).
- Hypoproteinemia (often asymptomatic, corrected calcium will fall within normal range).
- Severe depletion of magnesium.
- Severe alkalosis may elicit symptoms in patient with normal serum levels because there is a decrease in the ionized fraction of total serum calcium.
- Recent massive pRBC transfusion (citrate in packed RBCs binds serum calcium)

**Signs and Symptoms**

- Numbness and tingling of fingers, toes, and around mouth.
- Increased reflexes.
- Chvostek's sign: Tapping over the facial nerve in front of the tragus of the ear causes ipsilateral twitching.
- Trousseau's sign: Carpopedal spasm following inflation of sphygmomanometer cuff to above systolic blood pressure for several minutes.
- Muscle and abdominal cramps.
- Convulsions.
- ECG—prolonged QT interval.
**Hypercalcemia**

**Definition**

> 15 mg/dL.

**Causes**

- Hyperparathyroidism.
- Cancer (especially breast, multiple myeloma).
- Drugs (e.g., thiazides).

**Signs and Symptoms**

- Fatigue, weakness, anorexia, weight loss, nausea, vomiting.
- Somnambulism, stupor, coma.
- Severe headache, pain in the back and extremities, thirst, polydipsia, polyuria.
- Death.

**Treatment**

- Vigorous volume repletion—dilutes Ca and increases urinary Ca excretion:
  - May be augmented with furosemide.
  - Definitive treatment of acute hypercalcemic crisis in patients with hyperparathyroidism is immediate surgery.
  - Treat underlying cause.

**Indications for Nutritional Support**

**Enteral**

- Gut works but oral intake not possible—altered mental state, ventilator, oral/pharyngeal/esophageal disorders.
- Oral intake not sufficient for metabolic requirements.
- Presence of malnutrition and wasting.

**Total Parenteral Nutrition (TPN)**

- Enteral feeding not possible—GI obstruction, ileus.
- Enteral intake not sufficient for metabolic requirements—chronic diarrhea/emasium, malabsorption, fistulas, chemotherapy, irradiation therapy.
- The biggest danger of TPN is infection (the organic products in TPN can become infiltrated with bacteria and sent directly into the bloodstream).
- Adjunctive support necessary for managing disease—pancreatitis, hepatic failure, renal failure, chylothorax.
- Increased risks of acalculous cholecystitis, liver dysfunction.
Nutrient Requirements

**Caloric Requirements**

- Harris–Benedict equation and Fick equation (in patients with Swan–Ganz catheters) used to estimate basal energy expenditure (BEE).
- General estimation of BEE:
  - Males: BEE = 25 kcal/kg/day
  - Females: BEE = 22 kcal/kg/day
  - Multiply this by the desired goal:
    - Nonstressed patient: BEE × 1.2
    - Postsurgery: BEE × 1.3 to 1.5
    - Trauma/sepsis/burns: BEE × 1.6 to 2.0
    - Fever: 12% increase per °C
- The caloric needs must be met by nonprotein calories (i.e., fat and carbohydrate). Usually, carbs provide 70% of calories and fat provides 30%.

**Respiratory Quotient (RQ) of Individual Substrates**

RQ is defined as the ratio of carbon dioxide released to oxygen consumed per unit metabolism of a substrate (i.e., VCO₂/VO₂).

- RQ for lipid: 0.7
- RQ for protein: 0.8
- RQ for carbohydrates: 1.0
- RQ for balanced diet: 0.83
- RQ for overfeeding: > 1

**Protein Requirement**

- Usual protein requirement is 0.8–1 g/kg body weight.
- Requirement increases in illness and is maximal in burn patients.
- The average surgical patient needs 1.2–1.6 g/kg protein intake.
- Each gram of urinary nitrogen is equivalent to 6.25 g of degraded protein.
- Nitrogen balance:
  \[ \text{N Balance (g)} = \frac{\text{Protein Intake (g)}}{6.25} - (\text{UUN} + 4) \]
  - UUN = Urinary urea nitrogen, 4 represents daily nitrogen loss other than UUN.
  - The goal of nitrogen balance is to maintain positive balance of 4–6 g.
- Vitamins and trace elements must be incorporated in all feeding regimens.

**Enteral Nutrition**

- Preferred over parenteral nutrition.
- Reduces incidence of infection.
- May be continuous or intermittent.
- Routes for GI feeding: PO; NG feeding, gastrostomy, jejunostomy.
- Glutamine is the fuel for enterocytes.
- Short-chain fatty acids serve as fuel for the colonocytes.
- Omega-3 fatty acids and arginine serve as immune-modulating agents.

EXAM TIP

Causes of elevated anion gap metabolic acidosis:
- *Mud Pile*
  - Methanol/Metabolism (inborn errors)
  - Uremia
  - Diabetic ketoacidosis
  - Paraldehyde
  - Iron/isoniazid
  - Lactic acidosis
  - Ethylene glycol
  - Salicylates/strychnine
- Narrow down to four basic processes:
  - Ketoacidosis
  - Lactic acidosis
  - Renal failure
  - Intoxication
Complications

- Diarrhea.
- Aspiration.
- Obstruction of feeding tube.

Acid–Base Disorders

Assess the acid–base disorder step by step (Table 6-5):

- Is the primary disorder an acidosis (pH < 7.40) or alkalosis (pH > 7.40)?
- Is the disorder respiratory (pH and Pco₂ move in opposite directions)?
- Is the disorder metabolic (pH and Pco₂ move in same direction)?
- Is the disorder a simple or mixed disorder?

Use the following general rules of thumb for acute disorders:

- Metabolic acidosis: Pco₂ drops ~ 1.5 (drop in HCO₃⁻).
- Metabolic alkalosis: Pco₂ rises ~ 1.0 (rise in HCO₃⁻).
- Respiratory acidosis: HCO₃⁻ rises ~ 0.1 (rise in Pco₂).
- Respiratory alkalosis: HCO₃⁻ drops ~ 0.3 (drop in Pco₂).

Compensation beyond above parameters suggests mixed disorder.

metabolic acidosis

- Two varieties:
  - **Anion gap acidosis**—due to addition of unmeasured acid.
  - **Nonanion gap acidosis**—due to HCO₃⁻ loss.

**TABLE 6-5. Respiratory and Metabolic Components of Acid–Base Disorders**

<table>
<thead>
<tr>
<th>Type of Acid–Base Disorder</th>
<th>Acute (Uncompensated)</th>
<th>Chronic (Partially Compensated)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>p&lt;sub&gt;co₂&lt;/sub&gt;</td>
<td>p&lt;sub&gt;Asma&lt;/sub&gt;</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>↓↓</td>
<td>↑↑</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>↑↑</td>
<td>↓↓</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>↓↓</td>
<td>N</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑↑</td>
<td>N</td>
</tr>
</tbody>
</table>

*Measured as standard bicarbonate, whole blood buffer base, CO₂ content, or CO₂ combining power. The base excess value is positive when the standard bicarbonate is above normal and negative when the standard bicarbonate is below normal.

Calculating the anion gap:

\[ \text{AG} = \text{Na} - [\text{Cl} + \text{HCO}_3^-] \]

Normal AG = 10

**Metabolic Alkalosis**

Two mechanisms:

- Loss of H\(^+\) from kidneys or GI tract:
  - Renal: Mineralocorticoid excess, diuretics, potassium-losing nephropathy.
  - GI: Vomiting, gastric drainage, villous adenoma of colon.
- Gain HCO\(_3^-\): Milk-alkali syndrome, exogenous NaHCO\(_3\), lactated Ringer’s, packed RBC’s, TPN all contain substrates that metabolize to bicarbonate.

**Respiratory Acidsosls**

Hypercapnia secondary to one of two mechanisms:

- Hypoventilation (brain stem injury, neuromuscular disease, ventilator malfunction, opiates).
- Ventilation-perfusion (V/Q) mismatch (chronic obstructive pulmonary disease, pneumonia, pulmonary embolism, foreign body, pulmonary edema).

**Respiratory Alkalosis**

Hyperventilation secondary to anemia, anxiety, increased intracranial pressure (ICP), salicylates, fever, hypoxemia, systemic disease (sepsis), pain, pregnancy, CHF, pneumonia, asthma, liver disease.

Alkalosis causes decrease in serum K and ionized Ca, resulting in paresthesias, carpopedal spasm, and tetany.
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- AchAL AsIA
- Diffuse Esophageal Spasm (Des)
- Esophageal Diverticula
- Zenker’s Diverticulum

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Anatomy and Physiology

The esophagus is a 25-cm-long muscular tube that begins at the pharynx (begins at lower border of C6), travels through the thorax in the posterior mediastinum, and empties into the cardia of the stomach.

- Superior third: Striated muscle only.
- Middle third: Both striated and smooth muscle.
- Inferior third: Smooth muscle only.

Three areas of narrowing (evident on barium swallow; see Figure 7-1):

1. At the beginning of the esophagus, caused by the cricopharyngeus muscle.
2. Where the left mainstem bronchus and aortic arch cross.
3. At the hiatus of the diaphragm.

Two sphincters are present, which function as control points:

- Upper esophageal sphincter (UES, cricopharyngeus muscle) prevents the passage of excess air into the stomach during breathing.
- Lower esophageal sphincter (LES) is a physiological sphincter; it relaxes with initiation of the pharyngeal swallow and prevents the reflux of gastric contents when swallowing is not occurring.

Esophageal peristalsis accompanying swallowing is termed primary peristalsis.

Secondary peristalsis can be initiated by the esophageal musculature without the pharyngeal phase to clear the esophagus of any substance left behind from primary peristalsis.

![Figure 7-1](image.png)

**WARD TIP**

- The esophagus does not have the serosal layer, so esophageal anastomoses are prone to leaks.

**WARD TIP**

- The distance of the gastroesophageal (GE) junction from the incisor teeth is 40 cm and serves as an important landmark in upper gastrointestinal (GI) endoscopy.

**WARD TIP**

- Clinically significant motility disorders involve smooth muscle in the lower two thirds only.

**EXAM TIP**

Recall the vertebral levels at which the following traverse the diaphragm:

- T8 = inferior vena cava (IVC).
- T10 = esophagus.
- T12 = aorta.

“I (IVC) ate (T8) ten (T10) eggs (esophagus) at (aorta) noon (T12).”
Tools for the Assessment of Esophageal Functions

- Barium swallow: The patient ingests a radiopaque substance; swallowing and the esophagus are analyzed fluoroscopically.
- Flexible endoscopy: A camera is passed through the oral cavity into the esophagus, with the patient under sedation. There are multiple ports in the scope to allow for biopsies, irrigation, and suction.
- Manometry: Examines the motor function.
- Twenty-four-hour ambulatory pH monitoring.

Esophageal Motor Disorders

Result from abnormalities in the propulsive pump action of the esophagus or relaxation of the LES. Can be primary (achalasia, diffuse esophageal spasm [DES], nutcracker esophagus, hypertensive LES) or secondary (result of another systemic disease, including collagen vascular diseases like scleroderma and mixed connective tissue disease, neuromuscular disease, endocrine and metastatic disorders).

Definition

- Dysphagia: Difficult swallowing.
- Odynophagia: Pain on swallowing. May or may not accompany dysphagia.

Achalasia

Definition

Failure of the lower portion of the esophagus to relax during swallowing is defined as achalasia. The resulting dysphagia is due to three mechanisms:

1. Complete absence of peristalsis in the esophageal body.
2. Incomplete/impaired relaxation of the LES after swallowing.
3. Increased resting tone of the LES.

These result in elevation of intraluminal esophageal pressure, esophageal dilatation, and subsequent progressive loss of normal swallowing mechanisms—a functional holdup of ingested material.

Signs and Symptoms

- Dysphagia for both solids and liquids.
- Regurgitation of food.
- Severe halitosis (due to the decomposition of stagnant food within the esophagus).

Diagnosis

- Lateral upright chest x-ray (CXR) may reveal a dilated esophagus and the presence of air-fluid levels in the posterior mediastinum.
- Barium swallow will reveal the characteristic distal bird’s beak sign due to the collection of contrast material in the proximal dilated segment and the passage of a small amount of contrast through the narrowed LES (Figure 7-2).
- Esophageal motility study will confirm nonperistaltic contractions, incomplete LES relaxation, and increased LES tone.
- Esophagoscopy is indicated to rule out mass lesions or strictures, and to obtain specimens for biopsy.

WARD TIP

- A complaint of dysphagia must elicit a full dietary history from a patient; what they experience, when they eat, what types of food they eat that cause it.
- If the patient is immunocompromised, have a high index of suspicion for candidal esophagitis if human immunodeficiency virus (HIV)-positive, and for cytomegalovirus (CMV)/herpes simplex virus (HSV) esophagitis if non-HIV.

EXAM TIP

Achalasia: Gr. Failure to relax.

Classic triad of achalasia: Dysphagia to solids and liquids, regurgitation, and weight loss.

WARD TIP

Barium swallow:
- Bird’s beak or steeple sign: Achalasia.
- Corkscrew-shaped: Diff use esophageal spasm.
Medical management: Drugs that relax the LES—nitrates, calcium channel blockers, and antispasmodics.

- Botox injection: Helps relax the LES.
- Endoscopic dilatation: Has a lower success rate and a higher complication rate. It involves inserting a balloon or progressively larger sized dilators through the narrowed lumen, which causes tearing of the esophageal smooth muscle and decreases the competency of the LES.

Surgical management: Esophagomyotomy (Heller’s myotomy) with or without fundoplication is the treatment of choice for achalasia.

- Esophagomyotomy: Esophagus is exposed via a transthoracic (left thoracotomy), transabdominal, thorascopic, or laparoscopic technique. The tunica muscularis of the esophagus is incised distally, with extension to the LES. Complete division of the LES necessitates the addition of an antireflux procedure a (partial fundoplication).

Complications

- Risk of squamous cell carcinoma (SCC) is as high as 10% in patients with long-standing achalasia (duration 15–25 years).
- Patients may also develop pulmonary complications such as aspiration pneumonia, bronchiectasis, and asthma due to reflux and aspiration.

**Diffuse esophageal spasm (Des)**

**Definition**

DES is a disorder of unknown etiology that is primarily a disease of the esophageal body. It may be a primary disease process of the muscle, or may occur in association with reflux esophagitis, esophageal obstruction, collagen vascular disease, or diabetic neuropathy. Spasm occurs in the distal two thirds of the esophagus and is caused by uncoordinated large-amplitude rapid contractions of smooth muscle. By definition, the LES tone is normal.
**Signs and Symptoms**
- Dysphagia for both solids and liquids.
- Substernal chest pain, similar to that seen in a myocardial infarction: Acute onset of severe retrosternal pain that may radiate to the arms, jaw, or back. The chest pain may occur at rest, or it may follow swallowing. The degree of chest pain depends on the duration and severity of the contractions.
- No regurgitation (unlike achalasia); no water brash (unlike gastroesophageal reflux disease).

**Diagnosis**
- Barium swallow may reveal the characteristic “corkscrew” appearance of the esophagus, due to the ripples and sacculations that are visible as a result of uncoordinated esophageal contraction. Barium swallow may be entirely normal, however, because the esophagus may not be in spasm at the time of the study. In contrast to achalasia, the LES appears to be a normal diameter.
- Esophageal manometry studies will reveal the presence of large, uncoordinated, and repetitive contractions in the lower esophagus. Alternatively, manometry may appear normal when the patient is asymptomatic. LES manometry will show normal resting pressure with LES relaxation upon swallowing (again, unlike achalasia).
- Esophagoscopy should be performed to rule out mass lesions, strictures, or esophagitis.

**Treatment**
- Nitrates or calcium channel blockers to relax smooth muscle.
- Surgical treatment via an esophageal myotomy is not as successful in relieving symptoms as it is for achalasia, and is therefore not recommended unless dysphagia is severe and incapacitating (see Table 7-1).

**Esophageal Diverticula**

**Definition**
- Outpouching of the esophageal mucosa that protrudes through a defect in the muscle layer. Often occur when there are coexistent motility disorders.

---

**WARD TIP**
Due to the fact that DES produces cardiac-like complaints, the diagnosis is often delayed until an extensive cardiologic workup is performed.

**WARD TIP**
Patients with DES often have other functional intestinal disorders such as irritable bowel syndrome and spastic colon.

**WARD TIP**
*Nutcracker esophagus*, another hypermotility disorder, involves more focal segments of the esophagus.

**EXAM TIP**
*Epiphrenic and pharyngeal esophageal spasm* is caused by elevated pressure (pulsion) and are pseudo (false).

---

**Table 7-1. Achalasia vs. Diffuse Esophageal Spasm**

<table>
<thead>
<tr>
<th></th>
<th>Achalasia (Achalasia)</th>
<th>Diffuse Esophageal Spasm</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Signs and symptoms</strong></td>
<td>Dysphagia, regurgitation of undigested food, severe halitosis, weight loss, cough, diffuse chest pain.</td>
<td>Dysphagia, diffuse chest pain.</td>
</tr>
<tr>
<td><strong>Pattern of contraction</strong></td>
<td>Failure of LES to relax on swallowing.</td>
<td>Swallow-induced large wave of esophageal contraction, normal LES pressure.</td>
</tr>
<tr>
<td><strong>Barium swallow findings</strong></td>
<td>Absence of gastric bubble, narrowing of terminal esophagus that looks like a bird’s beak.</td>
<td>Corkscrew appearance.</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Nitroglycerin, local botulinum toxin, balloon dilatation, sphincter myotomy (Heller’s myotomy)</td>
<td>Nitroglycerin, nifedipine; surgery only if symptoms are severe and persistent.</td>
</tr>
</tbody>
</table>
■ May be either a true diverticulum, which involves all three layers of the esophagus (e.g., midesophageal diverticulum), or a false diverticulum, involving only the mucosa and submucosa (e.g., Zenker’s diverticulum).

■ Characterized by its location: Pharyngoesophageal (Zenker’s diverticulum), midesophageal, or epiphrenic (terminal third of the esophagus).

■ Pharyngoesophageal/Zenker’s and epiphrenic diverticula are called pulsion diverticula, since they are caused by increased esophageal pressure; they are false diverticula.

**Zenker’s Diverticulum**

**Signs and Symptoms**

Pharyngoesophageal (Zenker’s) type is the most likely to be symptomatic. Typical symptoms include dysphagia along with spontaneous regurgitation of undigested food, halitosis, choking, aspiration, repetitive respiratory infections, and, eventually, debilitation and weight loss.

**Diagnosis**

A barium swallow will reveal the presence of all types of diverticula. Endoscopy is difficult and potentially dangerous due to the risk of perforation through the diverticulum.

**Treatment**

Treatment of Zenker’s diverticulum is recommended to relieve symptoms and to prevent complications.

■ The most common procedure is a cervical pharyngocricoesophageal myotomy (incising the cricopharyngeus) and is done in all cases needing operative intervention.

■ Diverticulopexy (suturing the diverticulum in the inverted position to the prevertebral fascia) is added to myotomy for larger diverticula.

■ Diverticulectomy (endoscopic stapling of the diverticulum), along with myotomy, is performed for the largest diverticulae.

**Esophageal Varices**

**Pathophysiology**

■ Occur as a result of portal hypertension, most commonly a result of alcoholic cirrhosis.

■ As elevated portal system pressure impedes the flow of blood through the liver (increased intrahepatic resistance), various sites of venous anastomosis become dilated secondary to retrograde flow from the portal to systemic circulations. Varices are portosystemic collaterals.

■ Clinically significant portal-systemic sites:
  ■ Cardio-esophageal junction—dilatation leads to esophageal varices.
  ■ Periumbilical region—dilatation leads to caput medusae.
  ■ Rectum—dilatation leads to hemorrhoids.
  ■ Accounts for 10–30% of upper GI bleeds; up to 30% are fatal, 70% rebleed.

**Signs and Symptoms**

■ Painless hematemesis.
■ Unprovoked (i.e., not postemetic).
Hemodynamic instability is common.
- Risk for rebleeding is high.
- Peripheral stigmata of liver disease.

**Treatment**

- Identifying high-risk patients and preventing the first bleeding episode are critical (i.e., screening endoscopy to determine varices in cirrhotic patients). This includes pharmacological therapy to reduce portal pressure and consequently intravariceal pressure—reduce collateral portal venous flow with vasoconstrictors (somatostatin, vasopressin and octreotide decrease portal flow) and intrahepatic resistance with vasodilators (beta blockers, especially propranolol and nitrates, decrease portal pressure).
- Variceal bleeding ceases spontaneously in ~50% of cases.
- Management of ruptured varices causing an acute bleed:
  1. Stabilization of hemodynamics: Volume replacement with normal saline (NS) or lactated Ringer's and packed red blood cells (PRBCs), nasogastric (NG) suction, and lavage.
  2. Continuous vasopressin/somatostatin/octreotide to reduce splanchnic blood flow and portal pressure.
  3. Urgent endoscopic therapy: Endoscopic sclerotherapy (injection of the bleeding vessel(s) with a sclerosing agent via a catheter that is passed through the endoscope) stops bleeding in 80–90%; endoscopic band ligation (small elastic band is placed around the bleeding varix resulting in hemostasis) is equivalent to sclerotherapy initially, with fewer complications.
  4. Balloon tamponade (Sengstaken–Blakemore tube) to apply direct pressure and hemostasis to the varix with an inflatable balloon.
  5. For refractory acute bleeding, TIPSS procedure (transjugular intrahepatic portosystemic shunt).
  6. Intraoperative placement of a portocaval shunt. Surgical therapy is considered when there is continued hemorrhage or recurrent rebleeding with poor control.
  7. Liver transplant.

**Esophageal Perforation or Rupture**

**Definition**

- Trauma to the esophagus that may result in leakage of air and esophageal contents into the mediastinum.
- A surgical emergency.
- Carries a 50% mortality.

**Etiology**

- The most common cause of esophageal perforation is iatrogenic. Occurs following endoscopy, dilatation, tamponade tubes (Blakemore, Minnesota).
- Boerhaave syndrome (15% of cases): A spontaneous perforation and full-thickness tear. Usually occurs in the area of the left pleural cavity or just above the GE junction, due to transmission of abdominal pressure to the esophagus. Can result from forceful vomiting, retching coughing, labor, lifting, or trauma.

**WARD TIP**

Endoscopic sclerotherapy or band ligation for control of ruptured esophageal varices has a 90% success rate. Patients are usually intubated prior to the procedure to prevent aspiration of blood.

**EXAM TIP**

- Medical management in esophageal varices: Beta-blockers (propranolol, nadolol) and nitrates in asymptomatic patients; octreotide in active bleed.

**EXAM TIP**

The most serious complication of balloon tamponade for esophageal varices is esophageal perforation.

**EXAM TIP**

- TIPS can worsen hepatic encephalopathy—treat with lactulose, neomycin, or rifaximin.

**EXAM TIP**

**Typical scenario:** A man presents severe retrosternal and upper abdominal pain after an episode of retching. Think: Boerhaave syndrome (full-thickness) or Mallory–Weiss syndrome (partial-thickness) tears in the esophagus. A patient who recently underwent an endoscopic procedure develops fever and chest pain. Think: Iatrogenic esophageal rupture.
Mallory–Weiss syndrome: A partial-thickness mucosal tear. Usually occurs in the right posterolateral wall of the distal esophagus and results in bleeding that generally resolves spontaneously. Due to forceful vomiting.

Foreign body ingestions (14% of cases): Objects usually lodge near anatomic narrowings and then perforate through:
- Above the UES.
- Near the aortic arch.
- Above the LES.

**signs And symptoms**
- Severe, constant cervical, substernal, or back pain (depending on the location of the perforation).
- Dysphagia.
- Dypsnea.
- Subcutaneous emphysema.
- Mediastinal emphysema heard as a “crunching” sound (Hamman’s sign).
  - Sepsis/fever.
  - Pneumothorax.

**Diagnosis**
- CXR: Left-sided pleural effusion; mediastinal, cervical, or subcutaneous emphysema; mediastinal widening.
- Esophagogram with water-soluble contrast (gastrografin): Shows extravasation of contrast in 90% of patients.
- If water-soluble (gastrografin) study is negative, the patient must then get a barium swallow before a perforation can be ruled out.
- Other studies: Endoscopy, computed tomography (CT), and thoracentesis (check fluid for low pH and high amylase).

**treatment**
- Resuscitation and stabilization of patient.
- Primary surgical closure of full-thickness tears within 24 hours: 80–90% survival rate.
- Drain the contaminated mediastinum.
- Monitor for recovery from sepsis.
- Conservative nonoperative management: If the perforation is well contained in the mediastinum (with the barium draining back into the esophagus) and the patient has mild symptoms with minimal signs of sepsis, then the perforation can be managed with hyperalimentation, antibiotics, and gastric acid inhibition. Oral intake can be resumed within 1–2 weeks.

**Caustic Injury**
- Caused by acid (in household cleaning agents) or alkali (lye, sodium hydroxide tablets).
- Alkali burns are worse than acidic, as acid substances usually burn the mouth immediately and are less frequently swallowed; alkaline substances are more frequently ingested. Acidic substances also cause coagulative tissue necrosis, which limits their penetration, whereas alkaline substances cause injury deep into the tissue as they dissolve the tissue.

WARD TIP

In Boerhaave syndrome, the most common site of rupture is the left lateral wall of the esophagus, just above the esophageal hiatus. Iatrogenic perforation occurs most commonly following esophagogastroduodenoscopy (EGD) in the cervical esophagus near the cricopharyngeus muscle.

WARD TIP

Subcutaneous and mediastinal emphysema signify a full-thickness tear.
Caustic injury has both an acute phase—controlling immediate tissue injury and perforation potential—and a chronic phase—managing structures and swallowing disturbances that have developed. Acute damage is dependent on nature of substance, quantity, and time in contact with tissue.

**Signs and Symptoms**

- Oral and substernal pain in the initial phase; pain on swallowing and dysphagia.
- Hypersalivation.
- Fever—strongly correlated with an esophageal lesion.
- Vomiting and hemoptysis.
- Systemic hypovolemia and acidosis.
- Laryngospasm, laryngedema.
- Dysphagia reappears in the chronic phase due to fibrosis, retraction, and narrowing of the esophagus. Strictures develop in 80% of patients within 2 months.

**Treatment**

- Careful inspection of both the oral cavity and esophagus; however, there is poor correlation between the appearance of one and damage to the other.
- Early esophagoscopy (< 24 hours) to establish the presence of esophageal injury with exquisite care not to perforate the already damaged esophagus.
- Limiting the burn by administering neutralizing agents, preferably within the first hour. Lye/alkali can be neutralized with half-strength vinegar, lemon juice, or orange juice; acid with milk, egg white, or antacid.
- Broad-spectrum antibiotics to prevent infectious complications.
- Dilatations are controversial as they can traumatize the esophagus, but some start them early after injury to preserve the esophageal lumen and remove adhesions. They are done with a bougie in order to prevent and manage strictures.
- Extensive necrosis leading to perforation is best managed by resection; if the esophagus is viable, it can be managed with an intraluminal esophageal stent.
- Surgical intervention is indicated if there is complete stenosis with failure to establish a lumen, marked irregularity on barium swallow, development of severe mediastinitis with dilatation, fistula formation, or if the patient is unable to undergo prolonged periods of dilatation.
- Currently, the stomach, jejunum, and colon are organs used to replace the esophagus.

**Gastroesophageal Reflux Disease (GERD)**

**Definition**

- Defined by symptoms, presence of endoscopic esophagitis, or by measuring the increased exposure of the esophagus to gastric juice.
- Common disease, accounts for ~75% of esophageal pathology.

**Pathophysiology**

- Loss of the normal gastroesophageal barriers results in reflux. The primary barrier is the LES, and it is usually secondary to low or reduced LES resistance with reflux of acidic gastric contents into the esophagus.
Causes include a structurally defective sphincter; hiatal hernia; transient loss of the GE barrier (with a structurally normal LES) secondary to gastric abnormalities such as distention with air or food; delayed gastric emptying; and increased intra-abdominal pressure.

Prolonged exposure to a low pH from gastric contents (acid, pepsin, and duodenal contents, including biliary and pancreatic secretions) will cause irritation of the esophageal mucosa (as well as the respiratory epithelium) and the development of complications including esophagitis, stricture, Barrett’s esophagus (see next section) and risk of aspiration.

**Signs And Symptoms**

- Patients with GERD may report a range of symptoms from heartburn to angina-like chest pain.
  - Atypical symptoms include nausea, vomiting, postprandial fullness, choking, chronic cough, wheezing, and hoarseness.
  - Minimal or transient reflux may cause asymptomatic esophagitis, while severe reflux may cause severe esophagitis accompanied by laryngitis, aspiration pneumonitis/recurrent pneumonia, idiopathic pulmonary fibrosis, or asthma.
  - The presence of dysphagia may indicate peptic stricture formation.

**Diagnosis**

- Patients presenting with vague symptoms of chest pain must be evaluated for cardiac and pulmonary disease as deemed necessary (thorough physical exam, electrocardiogram [ECG], cardiac enzymes, and admission as appropriate).
- A barium study is useful to look for an anatomical cause for reflux, such as a hiatal hernia; can also elucidate pathology resulting from long-standing reflux, such as a stricture or ulcer formation.
- Twenty-four-hour ambulatory pH monitoring of the esophagus: A probe with pH electrodes is inserted into the patient’s esophagus for 24 hours. The probe continuously records the esophageal pH; useul in determining the severity of reflux (gold standard for diagnosing GERD).
- Esophageal manometry is useful in evaluating the competence of the LES.
- Esophagoscopy should be performed to evaluate the esophageal mucosa to rule out Barrett’s esophagus, and to obtain specimens for biopsy and Helicobacter pylori testing.

**Treatment**

Initial treatment of GERD involves medications that decrease gastric acid production along with lifestyle modification, including:

- Elevation of head end of bed.
- Antacids (symptomatic relief).
- H$_2$ antagonist (e.g., ranitidine).
- Proton pump inhibitors (PPIs; e.g., omeprazole).
- Education to avoid alcohol, coffee, chocolate, and peppermint, as they may aggravate symptoms; avoid nicotine, as it decreases LES tone.
- Instruct and educate the patient to eat small, frequent meals, elevate the head of the bed, avoid tight clothing, and to not go to sleep within 3–4 hours of a meal.
- Medications that promote gastric emptying (e.g., metoclopramide) may be beneficial early in the disease.
- The patient undergoes a trial of medical therapy for 6–12 weeks before further investigations.
- If medical management fails and the patient develops complications like chronic esophagitis or stricture, surgical intervention should be considered.
Surgery should be limited to those patients who have persistent or progressive disease despite maximal medical therapy or with a structurally defective LES. The primary goal of surgery is to return normal sphincter length and function and return the physiologic swallowing functions of the esophagus. The procedure of choice is a fundoplication (Figure 7-3)—wrapping

**Figure 7-3. Fundoplication procedure.** A. A large gastric tube or rubber esophageal dilator is inserted into the esophagus to prevent undue compression of the esophageal lumen. The right hand is introduced behind the fundus of the stomach to test the adequacy of the gastric mobilization. B. One or more long Babcock forceps are applied to the gastric wall on either side of the esophagus. The anterior and posterior gastric walls are approximated with interrupted silk sutures. C. After the traction drain and esophageal dilator are removed, the surgeon introduces the index finger or thumb upward under the plicated gastric wall. (Reproduced, with permission, from Zollinger RM Jr, Zollinger RM Sr. Zollinger's Atlas of Surgical Operations. 8th ed. New York: McGraw-Hill; 2003:97.)
the fundus of the stomach around the distal portion of the esophagus to create a sphincter, called the Nissen procedure, can be open or laparoscopic, transabdominal or transthoracic.

- Treatment of GERD is important in order to prevent the progression to Barrett’s esophagus. In addition, chronic reflux predisposes the patient to pulmonary complications as mentioned.

### Barrett’s Esophagus

#### Definition

A condition in which the distal portion of the tubular esophagus becomes lined by columnar epithelium as opposed to the normal squamous epithelium—histological appearance of intestinal metaplasia (the appearance of goblet cells).

This new region is susceptible to ulceration, bleeding, stricture, and adenocarcinoma formation.

#### Signs and Symptoms

- Usually similar to patients with GERD.
- Bleeding, hematemesis.
- Signs of esophageal perforation (see previous discussion).

#### Diagnosis

- Endoscopy for evaluation. Suspect when there is difficulty visualizing the squamocolumnar junction in the lower esophagus or an appearance of a redder mucosa.
- Multiple biopsies should be taken for a definitive or histologic diagnosis.

#### Management

- The same as those patients with GERD—require long-term PPI therapy for symptom relief and management of esophageal mucosal injury.
- Monitor and prevent disease progression to malignancy (risk 1% per year).
- Antireflux surgery when there are associated complications (stricture, ulceration, metaplastic progression).
- Surgical resection for refractory cases with high-grade dysplasia.

### Esophageal Carcinoma

#### Epidemiology

- The increasing prevalence of adenocarcinomas (due to Barrett’s) as compared to what was mostly SCC is shifting the epidemiology of esophageal cancer. Adenocarcinomas have increased in prevalence to more than 50% of all esophageal cancer in the western world. Other tumors of the esophagus are less common (including leiomyomas, melanomas, carcinoids, lymphomas).
Most cases occur in patients over the age of 50, but there is an increase in cases in younger patients with disease detected at an earlier stage.

Males are affected more frequently than females.

Over 50% of patients have unresectable or metastatic disease at the time of presentation.

Five-year survival rate is poor, but has increased slightly to 15%.

**Risk Factors**

- Environmental:
  - Tobacco.
  - Alcohol.
  - Food additives (nitrates in smoked and pickled meats).
- Esophageal disorders:
  - GERD/Barrett’s esophagus.
  - Achalasia.
  - Damage from caustic ingestion/strictures:
    - Chronic esophagitis.
    - Plummer–Vinson syndrome.
- History of radiation therapy to the mediastinum.

**Signs and Symptoms**

- Physical exam is usually entirely normal.
  - Patients may present with nonspecific GI complaints.
  - Gradual development of dysphagia (74% of patients) due to invasion of serosal layer, first for solids and later for both solids and liquids (mechanical dysphagia), may be present as well.
- Decreased PO intake and pain on swallowing result in profound weight loss, easy fatigability, and weakness.
- With advanced disease, the patient will appear cachectic; supraclavicular lymphadenopathy may be present, as may signs of distant metastasis. May develop symptoms depending on local invasion (stridor, coughing, aspiration pneumonia, hemoptysis, vocal cord/recurrent laryngeal nerve paralysis).

**Diagnosis**

- Population screening is untenable due to relatively low incidence, often absent early symptoms, rarity of a genetic cause.
- Asymptomatic patients are occasionally identified by surveillance endoscopy, especially patients with Barrett’s esophagus.
- Barium esophagram is the initial diagnostic test—may show stricture, ulceration, or mass.
- EGD is useful to both visualize the mass and to retrieve specimens for biopsy.
  - Majority of adenocarcinoma is found in the distal esophagus; most SCCs are found in the middle and lower third. The cervical esophagus is an uncommon site for disease.
- CT scan of the thorax, abdomen, and pelvis is useful to define the extent of disease and thereby determine appropriate treatment.
- Endoscopic ultrasound (EUS) is useful to measure the depth of tumor invasion and presence of lymphadenopathy for preoperative staging and surgical planning.
- Positron emission tomographic (PET) scan for lymphatic spread.
- Staging according to TNM (tumor, node, metastasis) classification.

---

**Exam Tip**

Dysphagia does not usually develop until > 60% of the esophageal lumen is obstructed.
Surgical therapy vs. palliative surgical therapy vs. nonsurgical palliation (see Figure 7-4).

- Management of disease limited to the esophagus:
  - Surgical resection (right thoracic or transhiatal approach, gastric pull-up, or colonic interposition can be used to reconstruct the GI tract).
  - Radiotherapy for avoidance of perioperative morbidity and mortality (can shrink tumor but may predispose to local complications and not palliate dysphagia and odynophagia).
  - Pre/postoperative chemotherapy.
  - Combination therapy of these three modalities is becoming increasingly common.
- Treatment for advanced stage IV disease: Chemotherapy to promote tumor shrinkage and palliate symptoms; poor survival rate nonetheless.

**WARD TIP**

The 5-year survival rate for esophageal carcinoma is ~5%.

---

**Figure 7-4.** Suggested global algorithm for the management of carcinoma of the esophagus. CT = computed tomography.

Postoperative complications are common and include fistulae or abscesses and respiratory complications. Other options include endoscopic laser therapy, endoscopic dilatation and stent placement, or placement of a gastrostomy or jejunostomy.

**Miscellaneous Esophageal Disorders**

- **Schatzki’s ring**: A thin, submucosal circumferential ring in the lower esophagus often associated with a hiatal hernia. Some believe it to be congenital, others due to infolding of redundant esophageal mucosa, and others due to stricture result from inflammation from chronic reflux.
  - Symptoms include brief episodes of dysphagia during hurried ingestion of solid foods.
  - Treatment ranges from dilatation +/- antireflux measures to incision of the ring and excision.
- **Plummer–Vinson syndrome** (Patterson–Kelly syndrome): An uncommon clinical syndrome characterized by dysphagia, atrophic oral mucosa, spoon-shaped and brittle fingernails, and chronic iron deficiency anemia. More common in perimenopausal women of Scandinavian origin.
  - An esophageal web, which is usually the cause of dysphagia was often thought to be a main component of the syndrome, but evidence has shown that it develops as a response to ingesting ferrous sulfate for the treatment of the anemia. Ferrous sulfate has been known to cause esophageal injury.
  - The web is usually below the cricopharyngeus muscle. Treatment consists of dilatation and iron therapy.
- **“Differentiating Esophageal Ulcers”**
  - CMV ulcers are typically linear, while HSV ulcers are typically punched out and deep

See Pediatric Surgery chapter for discussion of esophageal embryology and tracheoesophageal fistulas.
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**Anatomy**

See Figure 8-1.

**Blood Supply**

- Greater curvature: Right and left gastroepiploic arteries.
- Lesser curvature: Right and left gastric arteries.
- Pylorus: Gastroduodenal artery.
- Fundus: Short gastric arteries.

**Innervation**

See Figure 8-2.

- Anterior gastric wall: Left vagus nerve (gives branch to liver).
- Posterior gastric wall: Right vagus nerve (gives celiac branch and the “criminal nerve of Grassi”).
- Gastroduodenal pain: Sensation via sympathetic afferents from level T5 (below nipple line) to T10 (umbilicus).

**Histology and Physiology**

- Proximal cardiac glands: Secrete mucus.
- Fundus and body:
  - Parietal (oxyntic) cells: Secrete hydrochloric acid (HCl), accounting for the acidic pH of stomach; secrete intrinsic factor for absorption of vitamin B₁₂ in the terminal ileum.
  - Chief (peptic) cells: Secrete pepsinogen, a proenzyme activated by gastric HCl to form pepsin, which digests protein.

**WARD TIP**

Causes of Vitamin B₁₂ deficiency:
- Gastrectomy: Loss of intrinsic factor-secreting tissue.
- Disease or resection of terminal ileum: Causing malabsorption of B₁₂.
- Pernicious anemia: Autoimmune destruction of parietal cells.
- Insufficient dietary intake (B₁₂ is found in most foods of animal origin).

**OR TIP**

*Innervation of Stomach:*
- Left anterior
- Right posterior
- Antrum: G cells—secrete gastrin, which stimulates gastric acid secretion, pepsin secretion, and mucosal growth of the gastrointestinal (GI) tract (trophic action).
- Acid secretion by parietal cells is stimulated by the vagus nerve (acetylcholine via muscarinic M3 receptors), histamine (via H2 receptors), and gastrin (via gastrin receptors); the final common pathway is through the proton pump (H+/K+ ATPase).
- Gastrin release is stimulated by gastrin-releasing peptide (GRP) and the presence of digested protein products (amino acids) in the stomach; it is inhibited by somatostatin and low antral pH (< 2.5).
- Gastric mucosal barrier (protective gel layer) is enhanced by prostaglandin E (PGE) and damaged by nonsteroidal anti-inflammatory drugs (NSAIDs).
- Gastric bicarbonate secretion into the mucous gel is inhibited by NSAIDs, acetazolamide, alpha blockers, and alcohol.

### Peptic Ulcer Disease (PUD)

PUD is classified by location; most commonly duodenal ulcers (DUs) vs. gastric ulcers (GUs).

#### Epidemiology

- Environmental factors: Helicobacter pylori infection, NSAID use, smoking.
- Other risk factors: Family history of ulcers, Zollinger–Ellison (gastrinoma), corticosteroids (high dose and/or prolonged course).
- Ulcer incidence increases with age for both GU and DU; DU emerges two decades earlier than GU, particularly in males.

#### Complications

- Bleeding is the most common complication (20% incidence): Dizziness, syncope, hematemesis, melena.
- Perforation (7% incidence): Sudden, severe epigastric pain radiating to right shoulder, peritoneal signs, free peritoneal air.
- Obstruction: Due to scarring and edema; early satiety, anorexia, vomiting, weight loss.

### Duodenal Ulcer (DU)

#### Pathophysiology

Increased acid production (in contrast to GUs); also, H. pylori infection may weaken mucosal defenses.

#### Etiology

- H. pylori: A bacterium that produces urease, which breaks down the protective mucous lining of the stomach; 70–90% of patients with PUD have H. pylori infection.
- NSAIDs/Steroids: Inhibit production of PGE, which stimulates mucosal barrier production.

### WARD TIP

**Typical scenario:** A patient with known PUD presents with sudden onset of severe epigastric pain. Physical exam reveals guarding and rebound tenderness. Think: Perforation.

### WARD TIP

**Alarm symptoms** that indicate need for esophagogastroduodenoscopy (EGD):
- Weight loss
- Recurrent vomiting
- Dysphagia
- Bleeding
- Anemia

### EXAM TIP

**Typical scenario:** A 52-year-old woman presents due to 3 months of early satiety, weight loss, and nonbilious vomiting. Think: Gastric outlet obstruction.

### WARD TIP

Zollinger–Ellison syndrome accounts for 0.1–1% of patients with ulcer, but over 90% of patients with ZE have PUD (can see jejunal ulcers).
Zollinger–Ellison (ZE) syndrome: Gastrinoma (gastrin-secreting tumor in or near the pancreas; two thirds are malignant); 20% of ZE patients have associated multiple endocrine neoplasia type 1 (MEN-1: parathyroid hyperplasia, pancreatic islet tumors, pituitary tumors); diarrhea is common.

In the instance of a patient with recurrent duodenal ulcers not responding to H. pylori treatment, think Zollinger–Ellison syndrome, and check for concomitant pituitary and parathyroid problems (MEN 1).

**Clinical Features**

- Burning, gnawing epigastric pain that occurs with an empty stomach and is relieved by food or antacids (in contrast to GUs).
- Night time awakening (when stomach empties).
- Nausea, vomiting.
- Associated with blood type O.

**Diagnosis**

- DU: Endoscopy; however, most symptomatic cases of DU are easily diagnosed clinically.
- H. pylori:
  - Endoscopy with biopsy—allows culture and sensitivity for H. pylori (organism is notoriously hard to culture—multiple specimens required during biopsy).
  - Serology: Anti–H. pylori immunoglobulin G (IgG) indicates current or prior infection.
  - Urease breath test: C\textsuperscript{13/14} labeled urea is ingested. If gastric urease is present, the carbon isotope can be detected as CO\textsubscript{2} isotopes in the breath.
- ZE: A fasting serum gastrin level > 1,000 pg/mL is pathognomonic for gastrinoma. Secretin stimulation test: Secretin (a gastrin inhibitor) is delivered parenterally and its effect on gastrin secretion is measured. In ZE syndrome, there is a paradoxical astronomic rise in serum gastrin.

**Treatment**

**Medical**

- Risk modification:
  - Discontinue NSAIDs, steroids, smoking.
  - Prostaglandin analogues (e.g., misoprostol).
- Acid reduction:
  - Proton pump inhibitor (PPI): Omeprazole, lansoprazole, pantoprazole; 90% cure rate after 4 weeks.
  - H\textsubscript{2} blockers (cimetidine, ranitidine, famotidine, nizatidine): 85–95% cure rate after 8 weeks.
  - Antacids: Over the counter, good for occasional use for all causes of dyspepsia.
- Eradication of H. pylori:
  - Triple therapy (2-week regimen with bid dosing): PPI + amoxicillin + clarithromycin.
  - If patient is penicillin allergic, can substitute metronidazole for amoxicillin.
  - If patient fails one course of therapy, can try an alternate regimen using a different combination of drugs or quadruple therapy (2-week regimen of PPI + bismuth + tetracycline + metronidazole).

**WARD TIP**

H. pylori may colonize 50% of the global population—infestation does not necessitate disease.

**EXAM TIP**

Typical scenario: A 33-year-old female smoker presents with burning epigastric pain that is improved after eating a meal. Think: Duodenal ulcer.

**WARD TIP**

Triple therapy has 70–85% eradication rate.

Quadruple therapy has 75–90% eradication rate.
Since the advent of highly effective medical therapy, elective surgery for PUD is quite rare.

Surgery is indicated when ulcer is refractory to 12 weeks of medical treatment or if hemorrhage, obstruction, or perforation is present.

Truncal vagotomy and selective vagotomy are not commonly performed anymore due to associated morbidity (high rate of dumping syndrome) despite good protection against recurrence.

Procedure of choice is highly selective vagotomy (parietal cell vagotomy, proximal gastric vagotomy) (see Figure 8-2).

Individual branches of the anterior and posterior nerves of Latarjet in the gastrohepatic ligament going to the lesser curvature of the stomach are divided from a point 6 cm proximal from the pylorus to a point 6 cm proximal to the esophagogastric junction. The terminal branches to the pylorus and antrum are spared, preserving pyloroantral function and thus obviating the need for gastric drainage.

Preferred due to its lowest rate of dumping; however, it does have the highest rate of recurrence.

Recurrence depends on site of ulcer preop: Prepyloric ulcers have the highest recurrence rate at 30%. Least recurrence rate is with vagotomy + antrectomy.

Laparoscopic option: A posterior truncal vagotomy coupled with an anterior seromyotomy is being done laparoscopically.

For ZE: The tumor is resected. Occasionally, when focus of tumor cannot be found, a total gastrectomy may be considered in severe cases refractory to medical management.

**Gastric Ulcer (GU)**

- Decreased protection against acid; acid production may not be elevated (in contrast to DUs).
Can be caused by reflux of duodenal contents (pyloric sphincter dysfunction) and decreased mucus and bicarbonate production.

**Etiology**

- NSAIDs and steroids inhibit production of prostaglandins (PGE stimulates production of protective gastric mucosal barrier).
- *H. pylori* produces urease, which breaks down the gastric mucosal barrier.

**Classification**

- Location determines classification and is important in determining treatment (Table 8-1).
- Aid to memory: One is Less, Two has Two, Three is Pre, Four is by the Door.

**Signs and Symptoms**

- Burning, gnawing epigastric pain that occurs with anything in the stomach; pain is worst after eating (in contrast to DU).
- Anorexia/weight loss.
- Vomiting.
- Associated with blood type A.

**Diagnosis**

- Double contrast barium swallow.
- Endoscopy.
- All GUs are biopsied—3% are associated with gastric cancer.

**Treatment**

- Medical options: Same as for DUs.
- Surgical options: See Tables 8-1 and 8-2.

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**Table 8-1. Classification, Pathogenesis, and Surgical Treatment Options for Gastric Ulcer**

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<thead>
<tr>
<th>Type</th>
<th>Location of Ulcer</th>
<th>Pathogenesis</th>
<th>Surgical Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Most common; near angularis incisura on lesser curvature</td>
<td>Normal or decreased acid secretion; decreased mucosal defense</td>
<td>Distal gastrectomy with ulcer excision</td>
</tr>
<tr>
<td>II</td>
<td>Associated with DU (active or quiescent)</td>
<td>Normal or increased acid secretion</td>
<td>Antrectomy with truncal vagotomy and ulcer excision</td>
</tr>
<tr>
<td>III</td>
<td>Prepyloric</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Near gastroesophageal junction</td>
<td>Normal or subnormal acid secretion; decreased mucosal defense</td>
<td>Distal gastrectomy with ulcer excision and esophagagogastrojejunostomy</td>
</tr>
</tbody>
</table>

---

**WARD TIP**

- Smoking is a risk factor for GU.

**WARD TIP**

- Signs of duodenal perforation: Bleeding from the Back (posterior duodenal erosion/perforation, involving gastroduodenal artery). Free air from anterior duodenal perforation. Note: Anterior perforation is more common than posterior.

**Exam Tip**

- Typical scenario: A 45-year-old Japanese male smoker presents with weight loss and epigastric pain exacerbated by eating. Think Gastric ulcer.
**Table 8-2. Surgical Options in the Treatment of Duodenal and Gastric Ulcer Disease**

<table>
<thead>
<tr>
<th>Indication</th>
<th>Duodenal</th>
<th>Gastric</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bleeding</td>
<td>1. Oversew&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1. Oversew and biopsy&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2. Oversew, V+D</td>
<td>2. Oversew, biopsy, V+D</td>
</tr>
<tr>
<td></td>
<td>3. V+A</td>
<td>3. Distal gastrectomy&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Perforation</td>
<td>1. Patch&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1. Biopsy and patch&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>2. Patch, HSV&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2. Wedge excision, V+D</td>
</tr>
<tr>
<td></td>
<td>3. Patch, V+D</td>
<td>3. Distal gastrectomy&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Obstruction</td>
<td>1. HSV + GJ</td>
<td>1. Biopsy; HSV + GJ</td>
</tr>
<tr>
<td></td>
<td>2. V+A</td>
<td>2. Distal gastrectomy&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Intractability/nonhealing</td>
<td>1. HSV&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1. HSV and wedge excision</td>
</tr>
<tr>
<td></td>
<td>2. V+D</td>
<td>2. Distal gastrectomy&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>3. V+A</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>Unless the patient is in shock or moribund, a definitive procedure should be considered.

<sup>b</sup>Operation of choice in low-risk patient.

GI = gastrojejunostomy; HSV = highly selective vagotomy; V+ A = vagotomy and antrectomy; V+ D = vagotomy and drainage.


---

### Special Gastric Ulcers

- Curling’s ulcers: Gastric stress ulcers in patients with severe burns.
- Cushing’s ulcers: Gastric stress ulcer related to severe central nervous system (CNS) damage.

### Gastritis

**Definition**

Acute or chronic inflammation of the stomach lining.

**Etiology, Signs, and Symptoms**

Similar to PUD; endoscopy needed to differentiate.

**Diagnosis**

Diagnosis is made by endoscopy.

**Treatment**

Same as medical treatment of GUs.

---

**WARD TIP**

- Burnt paper CURLS.
- Cushing’s ulcer (think: head = CNS trauma/tumor).

**WARD TIP**

- Always biopsy a gastric ulcer to rule out cancer. Duodenal ulcers are almost always associated with H. pylori and don’t need to be biopsied.

---

**WARD TIP**

Biologies of gastritis:

- Gastric reflux (bile or pancreatic secretions)
- Nicotine
- Alcohol
- Stress
- Helicobacter pylori and other infections
- Ischemia
- NSAIDs
- Glucocorticoids (long-term use)
Compl ICat IonS

Chronic gastritis leads to:
- Gastric atrophy.
- Gastric metaplasia.
- Pernicious anemia (decreased production of intrinsic factor from gastric parietal cells due to idiopathic atrophy of the gastric mucosa and subsequent malabsorption of vitamin $B_{12}$).

Postgastrectomy Complications

poSt vagot omy dIar r Hea

dumpIng SyndromeS

- Complication of gastric surgery thought to result from unregulated movement of gastric contents from stomach to small intestine.
- Symptoms typically occur 5–15 minutes postprandially (early dumping syndrome) due to high osmolar load reaching the small intestine or 2–4 hours postprandially (late dumping syndrome) due to hypoglycemia.
- Nausea, vomiting, belching, diarrhea, tachycardia, palpitations, flushing, diaphoresis, dizziness, syncope.
- Treated by dietary modification: Small, multiple low-carbohydrate/meals; avoid excessive liquid intake.
- Severe cases (1%) that do not respond to dietary modifications can be treated with octreotide (synthetic somatostatin—helps delay gastric emptying time and transit through small intestine).

alkalIne r eF lux  gaS tr ItIS

- Diagnosis of exclusion after recurrent ulcer has been ruled out; nonspecific EGD and biopsy findings (edematous, inflamed gastric mucosa).
- Presents with postprandial pain and bilious vomiting.
- Medical treatment is difficult.
- Surgical management: Roux-en-Y gastrojejunostomy with a long (~50-cm) Roux limb. Bilious vomiting may improve, but symptoms (early satiety, bloating) may persist.

aFFer ent loop Syndr ome

- Obstruction of afferent limb following gastrojejunostomy (Bilroth II); two thirds present in postop week 1.
- Symptoms: Postprandial right upper quadrant (RUQ) pain, bilious vomiting, steatorrhea (with concomitant malabsorption of fats, $B_{12}$), anemia.
- Diagnosis: Afferent loop will be devoid of contrast of UGI series.
- Treatment: Endoscopic balloon dilatation or surgical revision of loop if that fails.

nu tr It lonal deFCIenCieS

- Vitamin $B_{12}$ deficiency anemia.
- Iron deficiency anemia.
- Osteoporosis (due to reduced calcium absorption).
Gastric Outlet Obstruction

**Common Causes**
- Malignant tumors of stomach and head of pancreas.
- Obstructing gastric or DUs.
- Usually with DU.
- Chronic ulcer causes secondary edema or scarring, which occludes lumen.

**Symptoms**

**Early**
- Early satiety.
- Gastric reflux.
- Abdominal distention.

**Late**
- Vomiting.
- Dehydration.
- Hypochloremic, hypokalemic metabolic alkalosis with paradoxical aciduria.
- Weight loss.

**Diagnosis**
Endoscopy or barium swallow x-ray.

**Treatment**
- Truncal vagotomy and pyloroplasty or gastrojejunostomy after 7 days of nasogastric (NG) decompression and antisecretory treatment.
- NG decompression is necessary to normalize the size of the dilated stomach.

Gastrointestinal Hemorrhage

**etiology**
- Ulcer (peptic)
- Varices
- Gastritis
- Arteriovenous malformation
- Mallory–Weiss tear

**Signs and Symptoms**
- Hematemesis (bright red or coffee grounds).
- Hypotension.
- Tachycardia.

WARD TIP
**CONTRAST:**

**Mallory–Weiss syndrome:**
Postemetic tears in gastric mucosa (near gastroesophageal junction).

**Boerhaave syndrome:**
Postemetic esophageal rupture.

WARD TIP

Coffee grounds is the term used to describe old, brown digested blood found on gastric lavage. It usually indicates a source of bleeding proximal to the ligament of Treitz.

WARD TIP

A bleeding scan detects active bleeding by infusing technetium-labeled autologous red blood cells ($^{99m}$Tc-labeled RBCs) and watching for their collection in the GI tract. It can be completed in ~1 hour and can detect bleeds as slow as 0.1 mL/min, but location specificity is only 60–70%. CT angiography is faster and detects bleeds up to 0.5 mL/min.
Bleeding that produces 60 cc of blood or more will produce black, tarry stool (melena).

Very brisk upper GI bleeds can be associated with bright red blood per rectum (hematochezia) and hypotension.

**Diagnosis**
- Gastric lavage with normal saline or free water to assess severity of bleeding (old vs. new blood).
- Rectal exam.
- Complete blood count (CBC).
- Endoscopy.
- Bleeding scan.
- Arteriography.

**Treatment**
- Depends on etiology and severity.
- Bleeding varices are ligated, or sclerosed via endoscopy (see Hepatobiliary System chapter).
- Most Mallory–Weiss tears resolve spontaneously.
- For severe bleeds:
  - Intravenous fluids and blood products as needed.
  - Somatostatin (inhibits gastric, intestinal, and biliary motility, decreases visceral blood flow).
  - Consider balloon tamponade for esophageal varices.
- Surgery:
  - About 5% of the time, upper GI bleeding cannot be controlled via endoscopic or other methods and emergent laparotomy will be necessary.
  - For DUs, a longitudinal incision is made across the pylorus and proximal duodenum. Bleeding is controlled by undersewing the vessel on either side of the hemorrhage.

**Bariatric Surgery**

**Indication**

BMI (body mass index) = weight in kg/(height in M)²:
- BMI > 35 with comorbidity (e.g., hypertension, diabetes mellitus).
- BMI ≥ 40 with or without comorbidity.

Prerequisite: Participation in supervised dietary program without success.

**Types**
- Restrictive: Reduction of the quantity of food intake.
  - Vertical banded gastroplasty (VBG): Partitioning of the stomach into a small proximal pouch (<20 mL) and a more distal one (see Figure 8-3).
  - Sleeve gastric resection.
  - Laparoscopic adjustable band placement (Figure 8-4).
- Malabsorptive: Limit nutrient absorption by bypassing duodenum and small intestine.
  - Biliopancreatic diversion with or without duodenal switch (Figure 8-5).
  - Roux-en-Y gastric bypass (also has a restrictive component; see Figure 8-6).
Malignant Tumors

Most common stomach malignancies are Adenocarcinoma (95%), Lymphoma (4%), and GIST (1%).

**Adenocarcinoma**

- **Epidemiology**
  - In general, gastric cancer is a disease of the elderly (age > 60), men > women, blacks > whites.
  - Adenocarcinoma comprises 95% of malignant gastric cancer.
  - Leading cause of cancer-related death in Japan.

- **Risk Factors**
  - Familial adenomatous polyposis.
  - Chronic atrophic gastritis.
  - H. pylori infection (6× increased risk).
  - Post-partial gastrectomy (15+ years).
  - Pernicious anemia.
- Diet (foods high in nitrites—preserved, smoked, cured).
- Cigarette smoking.
  - Epstein–Barr virus infection.

**Pathology**
- Polypoid: 25–50%, no substantial necrosis or ulceration.
- Ulcerative: 25–50%, sharp margins.
- Superficial spreading: 3–10%, involves mucosa and submucosa only, best prognosis.
- Linitis plastica: 7–10%, “leather bottle” type, involves all layers, extremely poor prognosis.

**Signs and Symptoms**
- Early: Mostly asymptomatic.
- Late: Anorexia/weight loss, nausea, vomiting, dysphagia, melena, hematemesis; pain is constant, nonradiating, exacerbated by food.
- Anemia—from blood loss, pernicious.
- Krukenberg’s tumor—metastasis to ovaries.
- Blumer’s shelf—metastasis to pelvic cul-de-sac, felt on digital rectal exam.
- Virchow’s node—metastasis to lymph node palpable in left supraclavicular fossa.
- Sister Mary Joseph’s nodule—periumbilical metastatic nodules.

**Diagnosis and Preoperative Staging**
- Upper GI endoscopy: Best method for diagnosis, allows for biopsy, definitive > 95% sensitivity and specificity.
- Upper GI series: With double contrast; 80–96% sensitivity, 90% specificity (operator dependent); excellent method in skilled hands.
- Abdominal and pelvic CT: With IV and oral contrast for preop staging to detect metastatic disease.
- Endoscopic ultrasound: For preop local assessment of tumor including depth of invasion and perigastric nodes.

**Staging**
See Table 8-3.

**Treatment**
- Radical subtotal gastrectomy can be curative in early disease confined to the superficial layers of the stomach (less than one third of all patients due to typical late presentation). Patients with proximal cancer may need total gastrectomy with Roux-en-Y reconstruction.
- Chemotherapy: Sometimes used palliatively for nonsurgical candidates; marginal benefit in adjuvant setting.

**Prognosis**
- Treatment is a major prognostic factor for gastric cancer—patients who are not resected have a poor prognosis.
- Location: Proximal gastric cancer has less favorable prognosis than distal lesions.
- Tumor markers: High preop serum levels of carcinoembryonic antigen (CEA) and CA 19-9 have been associated with less favorable outcomes.
- Other factors: Histologic grade, regional lymphatic spread.
<table>
<thead>
<tr>
<th>Stage</th>
<th>Tumor Classification</th>
<th>Lymph Nodes</th>
<th>Metastasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Tis</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>IA</td>
<td>T1</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>IB</td>
<td>T2</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T1</td>
<td>N1</td>
</tr>
<tr>
<td>IIA</td>
<td>T3</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T2</td>
<td>N1</td>
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</tr>
<tr>
<td>IIIIB</td>
<td>T4b</td>
<td>N0</td>
<td>M0</td>
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<td>N2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>T3</td>
<td>N3</td>
</tr>
</tbody>
</table>

(continues)
gaStr IcLymphoma

- Second most common malignant gastric cancer.
- Stomach is most common site for primary GI lymphoma (majority are B-cell non-Hodgkin’s type) but lymphoma comprise only 4% of all gastric tumors.
- Increased risk with H. pylori infection.

Signs and Symptoms
Non-specific; include abdominal discomfort, nausea, vomiting, anorexia, weight loss, and hemorrhage; occult bleeding and anemia (50% of patients).

Diagnosis
- Made by endoscopic biopsy, not readily distinguishable from adenocarcinoma by simple inspection.
- Bone marrow aspiration and gallium bone scans can diagnose metastases.

Treatment
- MALT (low grade)—treat H. pylori.
- MALT (high grade) or non-MALT—radiation/chemo ± surgical resection.
- Resection reserved for patients with bleeding or perforation.

Prognosis
Poor prognostic factors include:
- Involvement of the lesser curvature of the stomach.
- Large tumor size.
- Advanced stage.

gastrIC IntEstinal Stromal tumor (gISt)

- Mesenchymal (smooth muscle/interstitial cells of Cajal) tumors arising from the gastric stroma; submucosal and slow growing.
- Stomach is the most common site.
- Variable histology—from spindle cell tumors to epithelioid to pleomorphic.
- Approximately 95% of GISTs have c-kit (CD117) expression, leading to a constitutively active receptor tyrosine kinase.
- All GISTs are regarded as malignant.
- Treated by surgical resection and Gleevec (imatinib) (inhibits the tyrosine kinase receptor).
- Prognosis depends on completeness of resection, presence of metastases, and the mitotic index.
Benign Lesions

Benign tumors/adenomatous polyps

- Account for 10–20% of all gastric polyps.
- Are the only ones with any real malignant potential; others are mostly asymptomatic and uncommon.
- Biopsy lesions > 5 mm to check for neoplasia.

Ménétrier’s disease

Definition

- Hypertrophic gastropathy (enlarged, tortuous gastric rugae).
- Protein-losing enteropathy.
- Mucosal thickening secondary to hyperplasia of glandular cells replacing chief and parietal cells.
- Low-grade inflammatory infiltrate—not a form of gastritis.

Signs and Symptoms

- Most common: Middle-aged man who presents with epigastric pain, weight loss, diarrhea, hypoproteinemia.
- Less common: Nausea, vomiting, anorexia, occult GI bleed.
- Gastric acid secretion can be high, normal, or low.

Diagnosis

- Endoscopy with deep mucosal biopsy is definitive.
- Barium swallow will reveal large gastric folds and thickened rugae.

Treatment

- Anticholinergics, H₂ blockers to reduce protein loss.
- High-protein diet.
- Treatment of ulcers/cancer if present and eradication of H. pylori.
- Severe disease may require gastrectomy.

Dieulafoy’s lesion

Definition

Mucosal end artery that causes pressure necrosis and erodes into stomach and ruptures.

Symptoms

Massive, recurrent painless hematemesis.

Diagnosis

Upper GI endoscopy.

Treatment

- Endoscopic sclerosing therapy or electrocoagulation.
- Wedge resection.
**Gastric Volvulus**

**Definition**
Torsion/twisting of stomach typically along long axis. Often associated with paraesophageal hernia. May be acute, but most often chronic.

**Symptoms**
Brochardt’s triad:
- Intermittent severe epigastric pain and distention.
- Inability to vomit.
- Difficult passage of NG tube.

**Diagnosis**
Upper GI contrast study.

**Treatment**
- Surgical repair of accompanying hernia.
- Gastropexy—fixes stomach to anterior abdominal wall.
- Gastric resection if there is necrosis.
Introduction

The small bowel is the principal site for the absorption and digestion of nutrients, as well as for the maintenance of fluid homeostasis in the gastrointestinal system. It also serves as a major component of both the endocrine and immune systems.

Gastrointestinal (GI) Embryology

Fourth Week

The primitive gut tube, formed from the endoderm, begins to develop into the foregut, midgut, and hindgut.
- Endoderm becomes intestinal epithelium and glands.
- Mesoderm becomes connective tissue, muscle, and wall of intestine.

Fifth Week

- Intestine elongates and midgut loop herniates through umbilical ring.
- Midgut loop continues to lengthen extracoelomically until approximately week 10.

tenth Week

Midgut loop rotates 270° counterclockwise and returns back into the abdominal cavity.

Gross Anatomy

General

- Total length: 5–10 m (average 6 m).
- Consists of three parts: Duodenum, jejunum, ileum
- Aside from the first 2 cm, the duodenum is a retroperitoneal structure, while the jejunum and ileum are intraperitoneal structures.

Duodenum

- Extends from the pylorus to the duodenojejunal junction.
- Consists of four parts:
  - Superior (first) part—duodenal bulb: 5 cm long; site of most ulcers.
  - Descending (second) part—10 cm long; curves around the head of pancreas.
  - Transverse (third) part—10 cm long; crosses anterior to aorta and inferior vena cava (IVC) and posterior to the SMA and superior mesenteric vein (SMV).
  - Ascending (fourth) part—5 cm long; ascends past left side of aorta, then curves anteriorly to meet with jejunum, forming the duodenojejunal junction, which is suspended by the ligament of Treitz.
- Duodenum ends and jejunum begins at the ligament of Treitz.
- Plicae circulares (transverse mucosal folds in the lumen of the small bowel) are more prominent in the proximal small bowel (duodenum and jejunum) than in the distal small intestine (ileum).

**Duodenal Blood Supply**

- Arterial supply:
  - Proximal (up to ampulla of Vater): Gastroduodenal artery (first branch of proper hepatic artery) bifurcates into the anterior and posterior superior pancreaticoduodenal arteries.
  - Distal (beyond ampulla of Vater): Inferior pancreaticoduodenal artery (first branch of SMA) bifurcates into the anterior and posterior inferior pancreaticoduodenal arteries.
- Venous drainage:
  - Anterior and posterior pancreaticoduodenal veins drain into the SMV, which joins the splenic vein behind the neck of the pancreas to form the portal vein.
  - Prepyloric vein of Mayo is landmark for pylorus.

**Jejunum and Ileum**

- No anatomical boundary between the two.
- Jejunum is the proximal 40% of small intestine distal to ligament of Treitz.
- Ileum is the distal 60% of small intestine.
- Combined length is 5–10 m (average 6 m).
- Mesentery tethers the jejunum and ileum to posterior abdominal wall.
- Arterial supply:
  - Both jejunum and ileum supplied by branches of SMA, which runs in the mesentery.
  - The arteries loop to form arcades that give rise to straight arteries—vasa recta.
- Venous drainage: The SMV drains both the jejunum and ileum.

**Lymphatics**

- Drainage: Bowel wall → mesenteric nodes → lymphatic vessels parallel the corresponding arteries → cisterna chyli (a retroperitoneal structure between the aorta and IVC) → thoracic duct (also between the aorta and IVC) → left subclavian vein.
- Participate in absorption of fat.

**Innervation**

**Parasympathetic System**

- Source: Fibers originate from vagus and celiac ganglia.
- Function: Enhances bowel secretion, motility, and other digestive processes.

**Sympathetic System**

- Source: Fibers originate from ganglion cells that reside in a plexus at the base of the SMA.
- Function: Opposes effects of parasympathetic system on bowel.

**Enteric Nervous System**

Consists of Meissner plexus at base of submucosa and Auerbach plexus between the inner circumferential and outer longitudinal layers of the muscle wall.
**Intestinal contractions at a rate of 1–2 cm/sec.**
- Main function is to move chyme through the intestine.

**GI Hormones**

See Table 9-1.

**AbSorption over vieW**

See Table 9-2.

**Intestinal immune Function**
- Gut is largest immune organ in human body.
- Immunoglobulin A (IgA) is most prevalent type of immunoglobulin in lumen of GI tract, part of initial immune defense.
- Lymphoid nodules, mucosal lymphocytes, and isolated lymphoid follicles in appendix and mesenteric lymph nodes together constitute the mucosa-associated lymphoid tissue (MALT).

**Disorders of the Small Bowel**

**Small Bowel Obstruction (SBo)**

**Definition**
- Cessation, impairment, or reversal of the physiologic transit of intestinal contents secondary to a mechanical or functional cause.
- Most common cause is adhesions from prior abdominal surgery (75%).

**etiology**

**Mechanical**
- Extrinsic (adhesion, hernia, cancer, abscess, congenital).
- Intraluminal (gallstone ileus, foreign body, intussusception).
- Intramural (Crohn’s disease, lymphoma, radiation enteritis).

**Functional (Paralytic ileus)**
- Postoperative.
- Electrolyte abnormalities (e.g., hypokalemia).
- Peritonitis.
- Medications (opiates, anticholinergics).
- Hemoperitoneum/retroperitoneal hematoma.

**Pathophysiology**
- Gas and fluid begin to accumulate within lumen, proximal to site of obstruction.
- The bowel distends, intramural and intraluminal pressure rises.
- If intramural pressure exceeds the pressure in the microvasculature, then perfusion of the intestine is decreased, resulting in small bowel ischemia, and ultimately necrosis.
- Impairment of perfusion is termed strangulation.
### Table 9-1. Hormones of the GI Tract

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Site of Release</th>
<th>Action</th>
<th>Stimulated By</th>
<th>Inhibited By</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrin</td>
<td>Antrum</td>
<td>- Gastric acid secretion</td>
<td>- Vagus</td>
<td>- Food in antrum</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Cell growth</td>
<td>- Food in antrum</td>
<td>- Gastric distention</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Calcium</td>
<td>- Calcium</td>
</tr>
<tr>
<td>Cholecystokinin (CCK)</td>
<td>Duodenum</td>
<td>- Gallbladder contraction stimulates pancreatic acinar cell growth</td>
<td>- Polypeptides</td>
<td>- Chymotrypsin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Inhibits gastric emptying</td>
<td>- Amino acids</td>
<td>- Trypsin</td>
</tr>
<tr>
<td>Secretin</td>
<td>Duodenum</td>
<td>- Stimulates pancreatic secretion of H₂O and HCO₃⁻</td>
<td>- Low pH (acid)</td>
<td>- Intraluminal duodenal pH</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Bile secretion of HCO₃⁻</td>
<td>- Intraluminal duodenal fat</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Pepsin secretion</td>
<td>- Fat</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Inhibits gastric acid secretion</td>
<td>- Hydrochloride (HCl)</td>
<td></td>
</tr>
<tr>
<td>Somatostatin</td>
<td>Pancreas</td>
<td>- Increases small bowel reabsorption of H₂O and electrolytes</td>
<td>- Intraluminal fat</td>
<td>- Acetylcholine release</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Inhibits cell growth; GI motility; gallbladder contraction; pancreatic, biliary, and enteric secretion of gastric acid; and secretion/action of all GI hormones</td>
<td>- Gastric and duodenal mucosa</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Catecholamines</td>
<td></td>
</tr>
<tr>
<td>Pancreatic polypeptide</td>
<td>Pancreas</td>
<td>- Clinical usefulness of pancreatic polypeptide is limited to being a marker for other endocrine tumors of the pancreas</td>
<td>- Cephalic—vagus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Gastric—reflexes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Intestinal—food in small bowel</td>
<td></td>
</tr>
<tr>
<td>Neurotensin</td>
<td>Small bowel/colon</td>
<td>- Pancreatic secretion</td>
<td>- Fat</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Vasodilation</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Inhibits gastric acid secretion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peptide YY</td>
<td>Small bowel/colon</td>
<td>- Inhibits gastric acid secretion, pancreatic exocrine secretion, and migrating myoelectric complexes (MMCs)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucagon</td>
<td>Small bowel/colon</td>
<td>- Increases glycogenolysis, lipolysis, gluconeogenesis</td>
<td>- Low serum glucose</td>
<td>- Somatostatin</td>
</tr>
<tr>
<td>Motilin</td>
<td></td>
<td>- Inhibits MMCs</td>
<td>- Vagus</td>
<td>- Pancreatic polypeptide</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Increases gastric emptying</td>
<td>- Fat</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Increases pepsin secretion alkaline environment</td>
<td>- Intraduodenal</td>
<td></td>
</tr>
</tbody>
</table>
**EXAM TIP**

Short-chain fatty acids, unlike carbohydrates, are absorbed in the large intestine and can be used as an alternative energy source for persons who suffer from carbohydrate malabsorption.

**EXAM TIP**

The common causes of small bowel obstruction are: Henry a te Volumes In Chicago, Gaining Success Nowhere Hernia adhesions Volvulus Intussusception/Ileus Crohn’s disease Gallstone ileus SMA syndrome Neoplasm

---

**Table 9-2. Absorption in the Small Intestine**

<table>
<thead>
<tr>
<th></th>
<th>Duodenum</th>
<th>Jejunum</th>
<th>Ileum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Sodium</td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Potassium</td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Chloride</td>
<td>+</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Fats</td>
<td>++</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Proteins</td>
<td>+ +</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Carbohydrates</td>
<td>+ +</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Bile salts</td>
<td>0</td>
<td>0</td>
<td>+++</td>
</tr>
<tr>
<td>Fat-soluble vitamins</td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Water-soluble vitamins</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin B₁₂</td>
<td>0</td>
<td>0</td>
<td>+++</td>
</tr>
<tr>
<td>Folic acid</td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Ascorbic acid</td>
<td>?</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Minerals</td>
<td>+++</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>Iron</td>
<td>+++</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>Calcium</td>
<td>++</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Magnesium</td>
<td>+</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Zinc</td>
<td>0</td>
<td>+</td>
<td>++</td>
</tr>
</tbody>
</table>

* Vitamin K (endogenously produced fraction) absorbed in colon.
  
* Based on animal studies.

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**Types**

- Partial SBO—only part of the lumen is occluded, allowing some passage of intraluminal contents.
- Complete SBO—no passage of intraluminal contents distally.
- Closed loop obstruction:
  - Blockage of both proximal and distal segments of small intestine.
  - Seen with incarcerated hernia, torsion, adhesions, volvulus.
  - Requires emergent surgery because progression to strangulation is quite rapid.
**Risk Factors**
- Previous abdominal surgery (most common risk factor).
- Hernia.
- Inflammatory bowel disease (Crohn’s disease secondary to stricture formation).
- Diverticular disease.
- Cholelithiasis.
- Ingested foreign body.

**Signs and Symptoms**
- Colicky abdominal pain.
- Abdominal distention.
- Nausea.
- Vomiting.
- Obstipation.
- Hyperactive bowel sounds.
- Signs of decreased intravascular volume (hemoconcentration, electrolyte abnormalities) secondary to decreased PO intake, vomiting, and accumulation of fluid in bowel lumen and wall (third spacing).

**Diagnosis**
- Should differentiate between:
  - Mechanical vs. functional
  - Partial vs. complete
  - Simple vs. strangulation
- History should explore possible risk factors that may point to specific etiology.
- Physical exam should include meticulous abdominal exam, search for possible hernias, and examination of stool for gross or occult blood, which may indicate presence of strangulation.
- Confirm by abdominal series:
  - Supine abdominal x-ray: Dilated loops of small intestine with paucity of air in colon.
  - Upright abdominal x-ray: Multiple air-fluid levels in a “stepladder” (Figure 9-1).
  - Upright chest radiograph: Can detect presence of free air under the diaphragm and thus possible bowel perforation.
- Abdominal computed tomography (CT) scan more sensitive and specific than x-rays (Figure 9-2).
- Findings: Transition zone, with dilation of bowel proximally and decompression of the bowel distally, no contrast present distal to transition point, and paucity of gas and fluid in colon.
- Useful in acute setting to rule out other diagnoses as well.

**Treatment**
If the patient is stable or has partial SBO, give a trial of nonoperative management:
- NPO.
- IV hydration to counter effects of third spacing.
- Nasogastric tube (NGT) for gastric decompression; decreases nausea, vomiting, distention.
- Foley catheter to monitor urine output.
- Monitor electrolytes for signs of hypokalemia, base deficit/metabolic acidosis (signs of ischemia).
- Patients with suspected strangulation need to be resuscitated with fluids prior to surgery.

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**WARD TIP**
As opposed to large bowel obstruction, SBO is rarely caused by neoplasm. If neoplasm is the cause, it is most likely secondary to extrinsic compression as opposed to intraluminal obstruction.

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**WARD TIP**
Features associated with strangulated SBO include:
- Tenderness
- Tachycardia
- Fever
- Markedly elevated WBC count
- Acidosis with elevated lactate level

**Beware:** These indicators are NOT present in 5–15% of patients with intestinal infarction, especially the elderly and the immunocompromised.

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**WARD TIP**
There are no clinical or laboratory parameters that can reliably differentiate between simple obstruction and strangulated obstruction before the onset of ischemia.

---

**WARD TIP**
“Never let the sun rise or set on a small bowel obstruction.” While this adage was strictly adhered to in the past, currently, stable patients, particularly those with partial obstruction, are more likely to be treated nonoperatively. As with any patient, if symptoms worsen, or signs of strangulation develop, the patient should be brought to the OR.
If the patient fails conservative management (24 hours without improvement, abdominal tenderness worsens, fever, rising WBC count, other signs of clinical deterioration), then laparotomy should be performed.

The surgical procedure depends on the cause of the obstruction:
- Adhesions call for lysis of adhesions (LOA).
- Hernias should be reduced and repaired or, if contents of sac are strangulated, needs intestinal resection.
- Cancer requires en bloc resection with lymph node sampling.
- Crohn’s disease requires resection or stricturoplasty of affected area only.
Whatever the cause, the entire small bowel should be examined, and non-viable intestine should be resected.

Primary anastomosis should be performed in hemodynamically stable patients who have had small segments of bowel resected.

**Crohn’s Disease**

**Definition**
Inflammatory bowel disease characterized by transmural granulomatous inflammation involving any part of the GI tract, from mouth to anus, of unknown etiology. The inflammation is discontinuous, resulting in skip lesions, and often leads to fibrosis and ultimately obstruction, as well as to the formation of fistulae.

**Epidemiology**
- Eighty percent of patients have involvement of small bowel, usually the distal ileum (one third of these patients just have ileitis).
- Fifty percent of patients have involvement of both the ileum and colon.
- Twenty percent of patients have involvement of the colon only. Differentiate from ulcerative colitis because Crohn’s disease patients tend to have rectal sparing.
- One third of patients have perianal disease as well.
- Diagnosis most common between ages 15 and 40, although there is a second peak between 50 and 80 years of age (bimodal distribution).

**Risk Factors**
- Jewish descent.
- Positive family history.
- Urban dwelling.
- Smoking.

**Signs and Symptoms**
Typical symptoms include:
- Crampy abdominal pain (typically right lower quadrant [RLQ]), diarrhea, weight loss most common symptoms.
- Fever, fatigue.
- Bleeding (hemoccult + stools common, but gross lower GI bleeding less common than in ulcerative colitis).
- Perianal disease including skin tags, anal fissures, perirectal abscesses, and anorectal fistulae.
- Signs and symptoms of intestinal perforation and/or fistula formation (e.g., combination of localized peritonitis, fever, abdominal pain, tenderness, and palpable mass on physical exam).
- Extraintestinal manifestations include oral involvement (e.g., aphtous ulcers), joint involvement (e.g., arthritis), ocular involvement, dermatologic involvement (e.g., erythema nodosum, pyoderma gangrenosum), hepatobiliary involvement (e.g., sclerosing cholangitis).

**Diagnosis**
- Typical history of prolonged diarrhea with abdominal pain, weight loss, and fever with or without gross bleeding.
- Physical exam can be nonspecific or suggestive of Crohn’s disease, such as perianal skin tags, sinus tracts, or a palpable abdominal mass.

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**Typical Scenario**
A 5-year-old child presents with increasing irritability, colicky abdominal pain, and rectal bleeding with stools that have a currant jelly appearance. A tubular mass is palpated in the right lower quadrant. Upright abdominal x-ray shows air-fluid levels with a stepladder pattern. Think: Intussusception. Barium enema is both diagnostic and therapeutic.

**Differential Diagnosis of RLQ Pain**
- Appendicitis.
- Crohn’s disease.
- Infectious colitis—Yersinia, Campylobacter.
- Gynecologic pathology (ovarian torsion, abscess, ectopic pregnancy).
- Ischemic colitis.
Colonoscopy (with biopsy), visualization of the terminal ileum may reveal focal ulcerations adjacent to areas of normal mucosa along with a cobblestone appearance to the intestinal mucosa.

- Esophagogastroduodenoscopy (EGD) for proximal disease.
- Imaging (small bowel contrast studies) is helpful in characterizing length of involvement and areas of stricture, especially in parts of small bowel that are inaccessible via colonoscopy.
- Radiographic appearance: Mucosal nodularity, narrowed lumen, ulceration, string sign, presence of abscesses and fistulae.

**Treatment**

- Medical: Corticosteroids, aminosalicylates (sulfasalazine, 5-ASA), immune modulators (azathioprine, 6-mercaptopurine, cyclosporine), infliximab (anti-TNF-α), metronidazole.
- Many patients require surgery to relieve symptoms that do not respond to drugs, or to treat complications such as obstruction, abscesses, fistulae, perforation, perianal disease, or cancer.
- Surgery should be avoided if possible, since Crohn’s disease is not curable by surgery as opposed to ulcerative colitis.
- Surgical resection of an affected area does not preclude future disease development in adjacent or distant parts of the bowel.
- Surgical procedure depends on indication:
  - One third of patients require surgery to relieve intestinal obstruction by strictures, either via segmental small bowel resection or stricturoplasty (Figures 9-3 to 9-5).
  - For fistulae: Seton placement and drainage, fistulotomy, fistulectomy.
  - For extensive colonic disease: Total colectomy with end ileostomy.
  - For cancer: En bloc resection with lymph node dissection.

**WARD TIP**

Unlike ulcerative colitis, which can be cured with surgical resection, Crohn’s disease is incurable surgically.
**Prognosis**

- Typical course is one of intermittent exacerbations followed by periods of remission.
- Ten to 20% of patients experience prolonged remission after initial presentation.
- Approximately 80% of patients ultimately require surgical intervention.
- Like ulcerative colitis, there is an increased risk of colon cancer in patients with long-standing Crohn’s colitis.
- Resection with anastomosis has 10–15% clinical recurrence rate per year.
- Total colectomy with ileostomy has 10% recurrence rate over 10 years in remaining small bowel.
Benign neoplasms of small intestine

**Incidence**

- Adenomas > leiomyomas > lipomas (but leiomyomas most likely to cause symptoms).
- Most small intestinal benign neoplasms are found in the duodenum.
- Most common cause of adult intussusception.
- Most patients in 5th or 6th decade of life.

**Risk Factors**

- Hereditary syndromes:
  - Peutz–Jeghers syndrome (hamartomatous polyps).
  - Gardner syndrome (adenoma).
  - Familial adenomatous polyposis (adenoma).
- Consumption of red meat and salt-cured foods.

**Signs and Symptoms**

- Most are asymptomatic until they become large.
- Intermittent obstruction: Crampy abdominal pain, distention, nausea, and vomiting.
- Occult or overt bleeding.
- Palpable abdominal mass.
- Obstructive jaundice (periampullary lesion).

**Diagnosis**

- Endoscopic:
  - EGD: Can visualize proximal duodenum; most duodenal neoplasms are found incidentally on EGD.
  - Endoscopic ultrasound (EUS): Can offer more information such as the depth of intestinal wall involved.
- Radiographic:
  - Small bowel series—low sensitivity.
  - CT scan.
  - Enteroclysis: Test of choice; high sensitivity; used to detect tumors in distal small intestine.
- Majority of patients: Small bowel series with follow-through followed by enteroclysis.
- High-risk patients: Enteroclysis.

**Types**

See Table 9-3.

**Treatment**

- All symptomatic lesions should be resected, either surgically or endoscopically.
- Tumors located in proximal duodenum, even asymptomatic lesions, should be removed either endoscopically (< 1 cm) or surgically (> 2 cm).
- Tumors in second portion of duodenum, near ampulla, may require pancreaticoduodenectomy (Whipple procedure).

Malignant neoplasms of small intestine

- Rare.
- Adenocarcinoma > carcinoid > gastrointestinal stromal tumor (GIST) > lymphoma.
### Table 9-3. **Benign Neoplasms of the Small Intestine**

<table>
<thead>
<tr>
<th><strong>Type</strong></th>
<th><strong>Risk Factors</strong></th>
<th><strong>Signs and Symptoms</strong></th>
<th><strong>Location</strong></th>
<th><strong>Treatment</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenoma (35% of benign small bowel tumors)</td>
<td>Gardner's syndrome (GS)</td>
<td>Obstruction</td>
<td>Duodenum (20%)</td>
<td>Endoscopic or surgical excision for GS and familial adenomatous polyposis (FAP): - Screening EGD 2nd–3rd decade - Adenomas resected endoscopically - Adenoma recurrence requires pancreaticoduodenectomy (risk of ampullary carcinoma)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Bleeding</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Jejunum (30%)</td>
<td>Excision</td>
</tr>
<tr>
<td>FAP</td>
<td></td>
<td></td>
<td>Ileum (50%)</td>
<td>Excision</td>
</tr>
<tr>
<td>Leiomyoma</td>
<td></td>
<td>Obstruction, bleeding</td>
<td>Jejunum</td>
<td>Excision</td>
</tr>
<tr>
<td>Lipoma</td>
<td></td>
<td>Obstruction, incidental finding</td>
<td>Duodenum, ileum</td>
<td>Excision is required only if symptomatic</td>
</tr>
<tr>
<td>Hamartoma</td>
<td>Peutz–Jeghers syndrome</td>
<td>Recurrent colicky abdominal pain (from intermittent intussusception), obstruction, bleeding</td>
<td>Duodenum, ileum</td>
<td>Resection of segment responsible for symptoms</td>
</tr>
<tr>
<td>Hemangioma</td>
<td>(3–4% of benign small bowel tumors)</td>
<td>Bleeding</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fibroma</td>
<td></td>
<td>Obstruction, asymptomatic mass</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- Risk factors, signs and symptoms, and diagnosis essentially the same as benign neoplasms of small intestine (see Table 9-4):
  - Lymphoma: Celiac disease, immunodeficiency states, autoimmune disorders.

**Treatment**

- Wide en bloc resection of involved intestine.
- For adenocarcinomas, wide local excision of the intestine with its accompanying mesentery is performed along with regional lymph nodes.
- GISTs usually treated with segmental resection of affected intestine and Gleevec (imatinib), a tyrosine kinase inhibitor.
- Patients with duodenal lesions may require a Whipple procedure.
- Bypass may be required for palliation.

**Exam Tip**

**Typical scenario:** A patient presents with pigmented spots on his lips and a history of recurrent colicky abdominal pain. What is the cause of his abdominal pain? Think: Peutz–Jeghers syndrome. The hamartomatous polyps are likely causing intermittent intussusception.
### Table 9-4. Malignant Neoplasms of the Small Intestine

<table>
<thead>
<tr>
<th>Type</th>
<th>Risk Factors</th>
<th>Signs and Symptoms</th>
<th>Location (in Small Gut)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenocarcinoma (25–50% of primary small bowel malignancies)</td>
<td>Crohn's disease, celiac disease, FAP, Peutz-Jeghers syndrome</td>
<td>Obstruction</td>
<td>Duodenum (most found in duodenum)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bleeding</td>
<td>Ileum (in Crohn's disease most adenocarcinomas found in ileum)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mass</td>
<td></td>
</tr>
<tr>
<td>Carcinoid (up to 40% of primary small bowel malignancies)</td>
<td>Often asymptomatic</td>
<td>Obstruction</td>
<td>Ileum</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Carcinoid syndrome</td>
<td></td>
</tr>
<tr>
<td>GIST (CD117 [c-kit])</td>
<td></td>
<td>Hemorrhage</td>
<td>No regional preference</td>
</tr>
<tr>
<td>Lymphoma</td>
<td>Celiac disease</td>
<td>Obstruction</td>
<td>Ileum</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Perforation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Immunosuppression</td>
<td>Weight loss</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Autoimmune disease</td>
<td>Pain</td>
<td>Jejunum</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fatigue</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mass</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bleeding</td>
<td>Ileum</td>
</tr>
<tr>
<td>Sarcoma (most common leiomyosarcoma)</td>
<td></td>
<td>Obstruction (late symptom)</td>
<td>Meckel's diverticulum</td>
</tr>
<tr>
<td>Neuroendocrine</td>
<td></td>
<td>Mass</td>
<td>Proximal small intestine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hormone-specific symptoms</td>
<td></td>
</tr>
<tr>
<td>Metastatic</td>
<td>History of melanoma, breast, lung, ovarian, colon, or cervical cancer</td>
<td>Obstruction; bleeding</td>
<td></td>
</tr>
</tbody>
</table>

- Localized lymphoma is treated with segmental resection of the intestine, and neighboring mesentery.
- Diffuse lymphoma is the only situation where chemotherapy, rather than surgical resection, should be the primary therapy.

### Prognosis
- Overall 5-year survival: ≤ 20%.
- Five-year survival by tumor type:
  - Complete resection of duodenal adenocarcinoma: 50–60%.
  - Complete resection of jejunal/ileal adenocarcinoma: 5–30%.

**WARD TIP**

Small bowel is frequently affected by metastasis or invasion from cancers originating in other organs, particularly melanoma.
carcinoid

Definition
- Malignant tumor of enterochromaffin cell origin, part of amine precursor uptake and decarboxylation (APUD) system.
- Most common in appendix.

Incidence
- Carcinoid tumors represent between 29 and 40% of primary small bowel malignancies.
- Peak incidence between 50 and 70 years of age.
- More than 90% diagnosed in GI system:
  - The appendix is the most common site of GI carcinoid followed by small bowel followed by the rectum.

Signs and Symptoms
- Slow growing; therefore, frequently asymptomatic, and are usually found incidentally.
- In symptomatic patients, vague abdominal pain is the most common symptom.
- Intermittent obstruction—25% of patients.
- Rectal bleeding (from rectal carcinoid), pain, weight loss.
- Carcinoid syndrome: Approximately 10% of cases:
  - Due to production of serotonin, bradykinin, or tryptophan by tumor and exposure of these products to systemic circulation prior to breakdown by the liver.
  - Characterized by cutaneous flushing, sweating, watery diarrhea, wheezing, dyspnea, valvular lesions (right > left).

Diagnosis
- Most found incidentally during radiographic studies, appendectomy, or surgery for intestinal obstruction.
- If patient has carcinoid syndrome:
  - 5-HIAA (hydroxyindolacetic acid) in 24-hour urine collection.
  - Plasma chromogranin A—independent predictor of prognosis.
  - Pentagastrin provocation test: Pentagastrin administration induces flushing (used in patients with only marginally elevated 5-HIAA, but who describe flushing symptoms).
  - Otherwise diagnosed as any other small bowel neoplasm.

Treatment
- Medical: Serotonin antagonists (e.g., cyproheptadine) or somatostatin analogues (e.g., octreotide) for symptoms of carcinoid syndrome.
- Surgical:
  - Appendiceal carcinoid < 2 cm: Appendectomy.
  - Appendiceal carcinoid > 2 cm: Right hemicolectomy.
  - Small intestinal carcinoid: Resect tumor with mesenteric lymph nodes, as well as inspection of entire small bowel for synchronous lesions.
  - Otherwise, resect tumor and any solitary liver metastasis considered resectable.

EXAM TIP
The carcinoid syndrome develops when the tumor produces amines and peptides outside of the portovenous circulation. Classically, appendiceal and small intestinal carcinoids cause the carcinoid syndrome only after they have metastasized to the liver.

WARD TIP
Patients who present with carcinoid syndrome are typically not surgical candidates since they already have extensive metastatic disease.

EXAM TIP
Typical scenario: A 60-year-old male presents with a history of cutaneous flushing, diarrhea, wheezing, and an unintentional 15-lb weight loss. Think: Carcinoid syndrome; the wheezing is a clue that the lesion may be endobronchial. Order a 24-hour urine 5-HIAA level to confirm the diagnosis.
Prognosis
- Overall survival roughly 54%.
- Five-year survival after palliative resection is 25%; after curative resection is 70%.

FiSt u l a

Definition
A communication between two epithelialized surfaces. It can form between two parts of the GI or genitourinary (GU) tract, an internal fistula (e.g., choledochoduodenal or colovesical), or between an internal organ and an outer epithelialized surface (e.g., enterocutaneous), an external fistula.

Risk Factors
- Previous abdominal surgery (most common—80%).
- Diverticular disease.
- Crohn’s disease.
- Colorectal cancer.

Signs and Symptoms
- Iatrogenic fistulae usually appear 5–10 days postoperatively.
- If associated with an abscess, can be accompanied by fever and leukocytosis.
- Drainage of succus entericus (bowel contents) from skin (enterocutaneous fistula).
- Diarrhea, usually secondary to malabsorption (enteroenteric; especially if between proximal and distal small bowel or colon since a large portion of the small bowel absorptive surface is bypassed).
- Pneumaturia and symptoms of urinary tract infection (colovesicular fistula).

Diagnosis
- CT with enteral contrast shows leakage of contrast from intestinal lumen.
- Small bowel series.
- Enteroclysis.
- Fistulogram: Contrast is injected directly into fistula tract.

Treatment
- Stabilization: Manage electrolyte abnormalities, fluid losses, and nutritional status.
- Drainage of abscesses (if present).
- Allow time for spontaneous closure:
  - Bowel rest.
  - Provide nutrition (usually total parental nutrition [TPN]).
  - Consider octreotide (somatostatin analogue) for high-output and pancreatic fistulas.
- If 6–8 weeks pass without improvement, then surgery should be performed to resect the fistula tract, together with the segment of small bowel from which it originates.

Prognosis
- Enterocutaneous fistulas have 15–20% mortality related to complications of sepsis and underlying disease.
- Surgery is associated with considerable morbidity and a high recurrence rate.
meSenteric iSchemia

**Definition**
- Reduction in blood flow to the small bowel secondary to a variety of mechanisms.
- Can result in two distinct conditions: acute mesenteric ischemia and chronic mesenteric ischemia.

**Acute meSenteric iSchemia (AMI)**
- Rapid onset of intestinal hypoperfusion.
- Can be caused by arterial occlusion (usually single artery):
  - SMA embolism—50% of all AMI.
  - SMA thrombosis (acute)—15–25% of all AMI.
  - Vasospasm (nonocclusive mesenteric ischemia)—20–30% of all AMI.
  - Venous obstruction (either thrombosis or strangulation usually of SMV)—5% of all AMI.
- Regardless of cause, AMI can lead to mucosal sloughing within 3 hours of onset, and to intestinal infarction 6 hours after onset.

**Risk Factors**
- Cardiac: Low cardiac output states, cardiac arrhythmias, severe cardiac valvular disease, recent myocardial infarction.
- Age.
- Atherosclerosis.
- Hypercoagulable states.

**Signs and Symptoms**
- Abdominal pain out of proportion to tenderness on physical exam (hallmark).
- Pain is colicky and diffuse, typically in mid-abdomen.
- Can be associated with nausea, vomiting, and diarrhea.
- Following infarction, peritonitis (rebound and rigidity), abdominal distention, and passage of bloody stools occur.

**Diagnosis**
- High clinical suspicion especially if history of cardiac disease or hypercoagulability.
- No lab test is sensitive for diagnosis of AMI. Findings may include leukocytosis, acidosis, hemoconcentration, and occult blood in stool.
- If there are peritoneal signs, then patient should undergo emergent laparotomy.
- Otherwise, pursue diagnostic tests:
  - Mesenteric angiography: Most sensitive and specific method for diagnosing AMI, albeit invasive.
  - CT scan with IV contrast:
    - Good initial test for most patients with possible AMI.
    - Can rule out other causes of abdominal pain.
    - Can reveal evidence of ischemia of intestine and mesentery.
    - Can reveal occlusion/stenosis of vasculature.

**Therapy**
- Stabilization of patient:
  - Hemodynamic monitoring and fluid resuscitation.
  - Correction of electrolyte abnormalities, acidosis.
- Broad-spectrum antibiotics.
- Placement of a nasogastric tube for gastric decompression.
- Patient with peritoneal features: Midline laparotomy with assessment of intestinal viability.
- For embolic causes: Intraoperative embolectomy.
- For CA/SMA thrombosis: Bypass of site of obstruction using a saphenous vein graft (from supraceliac aorta to distal SMA).
- Therapy via angiography includes intra-arterial vasodilators (papaverine) or thrombolytic agents (streptokinase, urokinase, tPA), angioplasty, placement of a stent, and embolectomy.
- Venous thrombosis requires immediate anticoagulation (heparin).

**Prognosis**
- Acute arterial mesenteric ischemia mortality rate: 59–93%.
- Acute mesenteric venous thrombosis mortality rate: 20–50% (30% recurrence rate if not anticoagulated).

**Chronic Mesenteric Ischemia (CMI)**
- Insidious, episodic, or constant state of intestinal hypoperfusion.
- Rarely leads to infarction of small bowel due to development of collateral circulation over time.
- Can be caused by:
  - Arterial ischemia (most common): Associated with atherosclerosis of more than one mesenteric and splanchnic vessels.
  - Venous thrombosis: Thrombosis of portal or splenic veins, leading to portal hypertension with subsequent development of esophageal varices and splenomegaly.
  - Vasculitis.

**Risk Factors**
- Atherosclerotic vascular disease (half of patients have history of peripheral vascular disease or coronary artery disease).
- Smoking.

**Signs and Symptoms**
- “Intestinal angina”: Dull, crampy, postprandial abdominal pain leading to food aversion and weight loss (hallmark).
- Patients with chronic venous thrombosis may be asymptomatic due to collateral formation, or may present with esophageal variceal bleeding.

**Diagnosis**
- High clinical suspicion.
- Must rule out other causes of chronic abdominal pain, weight loss, and food aversion, particularly malignancy.
- Diagnosis is supported by demonstration of high-grade stenoses in multiple mesenteric vessels.
- Diagnostic tests:
  - Angiography: Gold standard.
  - CT and magnetic resonance (MR) angiography: Good initial tests since they can identify whether a stenosis is present and serve as a guide for angiography.
  - Mesenteric duplex ultrasonography: Can be used as a screening test to detect stenoses of the celiac and superior mesenteric arteries.

**WARD TIP**
Thrombolytic therapy for intra-arterial emboli is most successful when initiated within 8–12 hours of symptom onset.

**WARD TIP**
Patients with acute mesenteric venous thrombosis should be evaluated for hereditary and acquired thrombophilias.

**OR TIP**
CMI can occur due to compression of celiac artery by diaphragm—celiac artery compression syndrome or median arcuate ligament syndrome. Treatment is release of the arcuate ligament and bypass of persistent stricture.
Therapy
- Arterial CMI:
  - Surgical revascularization:
    - Aortomesenteric bypass graft
    - Mesenteric endarterectomy
  - Percutaneous transluminal angioplasty (PTA) with or without placement of a stent:
    - Less relief of symptoms and less durability than surgery.
    - Serves as alternative to surgery for those patients who are not optimal surgical candidates due to considerable comorbidities.
  - Chronic venous mesenteric thrombosis: Anticoagulation (heparin).

Prognosis
- Perioperative mortality rates range from 0 to 16%.
- Initial relief of symptoms with surgery: 90%.
- Recurrence rate:
  - Following surgery: < 10%.
  - Following PTA: 10–67%.

Short Bowel Syndrome

Definition
- Presence of < 200 cm of small bowel in adult patients.
- Results in decreased small bowel absorption, leading to diarrhea, malnutrition, and dehydration.

Etiologies
- Adults: Acute mesenteric ischemia, Crohn’s disease, malignancy.
- Pediatrics: Volvulus, intestinal atresia, necrotizing enterocolitis.

Pathophysiology
- Malabsorption occurs following resection of 50–80% of small bowel.
- Presence of intact colon, as well as an ileocecal valve, also decreases severity of malabsorption.
- Resection of ileum is associated with increased malabsorption secondary to inability to absorb bile salts and vitamin $B_{12}$.
- Small bowel adaptation period lasts approximately 1–2 years following surgery.

Therapy
- Medical:
  - Repletion of fluid and electrolytes: Initially, most patients require TPN.
  - Proton pump inhibitors/H₂ blockers to decrease gastric acid secretion.
  - Antimotility agents to decrease transit through small bowel.
  - Octreotide may be used to decrease intestinal secretions.
- Surgical:
  - Restoration of intestinal continuity in patients with stomas, in order to increase absorptive capacity.
  - Surgeries aimed at slowing transit through the small intestine include:
    - Segmental reversal of the small bowel.
    - Placement of a segment of colon between two segments of small bowel.
    - Creation of artificial small bowel valves.

Typical scenario: A 70-year-old male with a history of peripheral vascular disease and hyperlipidemia presents to the emergency department with severe, diffuse abdominal pain. His blood pressure is 170/100 and his pulse is 90 bpm. Supine abdominal radiograph shows free air in the abdomen and within the wall of the small intestine. What is the most likely diagnosis? Think: Small bowel infarction.

WARD TIP
TPN is associated with considerable morbidity including catheter sepsis, liver and kidney failure, and venous thrombosis.
Surgeries aimed at lengthening the small bowel include:
- Longitudinal intestinal lengthening and tailoring (LILT).
- Serial transverse enteroplasty procedure (STEP).
- Small bowel transplantation:
  - One hundred performed in the United States each year.
  - Indicated for patients with life-threatening intestinal failure, or complications from TPN.
  - Eighty percent of survivors have full intestinal function without need for TPN.
  - Significant risks including acute or chronic rejection, as well as CMV infection.

Prognosis
- Fifty to 70% of patients who initially require TPN can eventually be completely weaned off of TPN.
- Pediatric patients are more likely than adults to gain independence from TPN.
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LARGE BOWEL OBSTRUCTION
EMBRYOLOGY AND ANATOMY

**Embryology**

- Origin: Embryonic midgut (up to mid-transverse colon) and hindgut (rest of colon, and proximal anus). Distal anus derived from ectoderm.
- The dentate line marks the transition between hindgut and ectoderm.

**Gross Anatomy**

See Figure 10-1.

**Colon**

- Extends from the ileocecal valve to the rectum; consists of: ascending colon, transverse colon, descending colon, sigmoid colon.
- 3–5 feet in length.
- Cecum is widest; the colon progressively narrows distally.
- Unlike the small intestine, the colon has taenia coli, haustra, and appendices epiploicae (fat appendages that hang off antimesenteric side of colon).
- Taenia coli—three distinct bands of longitudinal muscle—converge at the appendix and spread out to form a longitudinal muscle layer at the proximal rectum.
- Retroperitoneal attachments of ascending and descending colon fix it to the posterior abdominal wall.
- Retroperitoneal: Ascending colon, descending colon, posterior hepatic and splenic flexures.
- Intraperitoneal: Cecum, transverse colon, sigmoid colon.
- One end of omentum attaches to anterior-superior aspect of transverse colon; other end attaches to stomach.

**Rectum**

- 12–15 cm in length.
- Rectum has distinct peritoneal covering.

**Example Tip**

The blood supply is based on embryology:
- Midgut: SMA
- Hindgut: Inferior mesenteric artery (IMA)
- Distal anus: Internal pudendal artery branches

**WARD Tip**

In development, the midgut loop rotates 270° counterclockwise around the axis of the superior mesenteric artery (SMA). Developmental anomalies include malrotation or failure of right colon to elongate.

**Figure 10-1.** Bowel anatomy.
**Fascia:**
- Waldeyer’s fascia: Rectosacral fascia that extends from S4 vertebral body to rectum.
- Denonvilliers’ fascia: Anterior to lower third of rectum.

**Pelvic floor:** Levator ani (composed of pubococygeus, iliococcygeus, and puborectalis muscles); innervated by S4 nerve.

**Anus**
- Anal canal runs from pelvic diaphragm to anal verge (junction of anoderm and perianal skin).
- Dentate line: A mucocutaneous line that separates proximal, pleated mucosa from distal, smooth anoderm (1–1.5 cm above anal verge).
- Anal mucosa proximal to dentate line lined by columnar epithelium; mucosa distal to dentate line (anoderm) lined by squamous epithelium and lacks glands and hair.
- Columns of Morgagni: 12–14 columns of pleated mucosa superior to the dentate line separated by crypts. Perianal glands discharge their secretions at the base of the columns.
- Anal sphincter:
  - Internal: Consists of specialized rectal smooth muscle (from inner circular layer); involuntary, contracted at rest, responsible for 80% of resting pressure.
  - External: Consists of three loops of voluntary striated muscle; a continuation of puborectalis muscle; responsible for 20% of resting pressure and 100% of voluntary pressure.

**Blood supply**

**Arterial**
- **Superior mesenteric artery (SMA):** Supplies the cecum, ascending colon, and proximal two thirds of the transverse colon via the ileocolic, right colic, and middle colic arteries, respectively.
- **Inferior mesenteric artery (IMA):** Supplies the distal two thirds of the transverse colon, sigmoid colon, and superior rectum via the left colic, sigmoidal, and superior rectal (hemorrhoidal) arteries, respectively.
- **Internal iliac artery:** Supplies the middle and distal rectum via the middle rectal and inferior rectal arteries, respectively (the inferior rectal artery is a branch of the internal pudendal artery).
- **Internal pudendal artery:** Supplies the anus; is a branch of the internal iliac artery.

**Venous**
- **Superior mesenteric vein (SMV):** Drains the cecum and ascending and transverse colon before joining the splenic vein.
- **Inferior mesenteric vein (IMV):** Drains the descending colon, sigmoid colon, and proximal rectum before joining the splenic vein.
- **Internal iliac vein:** Drains the middle and distal rectum.
- **Middle rectal vein:** A branch of the internal iliac vein; drains upper anus.
- **Inferior rectal vein:** A branch of the internal pudendal vein; drains lower anus.
- **Hemorrhoidal complexes:** Three complexes within the anus that drain into the superior rectal veins.

**WARD TIP**
- The ileocecal valve functions to prevent reflux of bowel contents from the cecum back to the ileum.

**WARD TIP**
- The anatomy and physiology of the colon affect how colon cancers typically present. Left-sided colon cancers tend to present with a change in bowel habits (e.g., small-caliber stools, obstruction, and hematochezia). Right-sided colon cancers tend to present in a more indolent fashion with microcytic anemia, fatigue, and melena (dark, tarry stools) because the proximal colon has a larger circumference and the stool is less solid.

**WARD TIP**
- The splenic flexure represents a “watershed” area between the areas supplied by the superior and inferior mesenteric arteries. This watershed area is particularly susceptible to ischemic injury as seen in ischemic colitis. The other two watershed zones are the ileocecal area and the junction of descending and sigmoid colon.

**WARD TIP**
- The rectum has two major angles that play a significant role in continence. The first angle is formed at the origin of the rectum at the sacral promontory as it bends posteriorly and inferiorly, following the curve of the sacrum. The second angle, the anorectal angle, is formed by the puborectalis muscle as it joins the anus, pulling the rectum forward. A Valsalva maneuver enhances these angles, closing off the rectum.
Lymphatics of the colon, rectum, and anus generally follow the arterial supply, with several levels of nodes as one moves centrally toward the aorta (e.g., ileocolic nodes, superior mesenteric nodes, etc.).

**innervation**

- Derives primarily from autonomic nervous system.
- Sympathetic nerves: Inhibit peristalsis.
- Parasympathetic nerves: Stimulate peristalsis.

**histology**

From inner lumen to outer wall:
- Colon: Mucosa, submucosa, inner circular muscle layer, outer longitudinal muscle (taenia coli).
- Rectum: Mucosa, submucosa, inner circular muscle, outer longitudinal muscle (confuent).
- Anus: Anoderm (epithelium that is richly innervated, but without secondary skin appendages).

**microbiology**

- Colon sterile at birth; normal flora established shortly thereafter.
- Normal flora: 99% anaerobic (predominantly Bacteroides fragilis); 1% aerobic (predominantly Escherichia coli).

**Physiology**

**General**

The colon and rectum have three primary physiologic functions:
1. Absorption of water and electrolytes from stool
2. Storage of feces
3. Motility

**Motility**

Characterized by three types of contractions:
1. Retrograde movements: From transverse colon to cecum, these movements slow the transit of luminal contents, thereby prolonging their exposure to absorptive epithelium.
2. Segmental contractions: The most common variety, these are localized simultaneous contractions of the longitudinal and circular muscles of the colon.
3. Mass movements:
   - Contractions of long segments of colon that are 30 seconds in duration and result in antegrade propulsion of luminal contents at a rate of 0.5–1 cm/sec.
   - Occur 3–4 times each day, especially after waking up or after eating, and may result in bowel movements.
Neuronal control of colon:
1. Extrinsic: Parasympathetic and sympathetic (as described above).
2. Intrinsic (from mucosa to bowel wall): Mucosa, submucosal (Meissner’s) plexus, circular muscle layer, myenteric (Auerbach’s) plexus, longitudinal muscle layer, subserosal plexus, serosa.

**Defecation**
1. Mass movement causes feces to move into rectal vault.
2. Sampling reflex: Rectal distention leads to involuntary relaxation of internal sphincter, allowing descent of rectal contents and sensation of feces at transitional zone.
3. Voluntary relaxation of external sphincter pushes contents down anal canal.
4. Voluntary increase in intra-abdominal pressure assists in propelling rectal contents out of anus.

**Disorders of Motility**

**Irritable Bowel Syndrome (IBS)**

**Definition**
- Abnormal state of intestinal motility modified by psychosocial factors for which no anatomic cause can be found.
- IBS is often regarded as a wastebasket diagnosis for a change in bowel habits along with complaints of abdominal pain after other causes have been excluded.

**Constipation**

**Definition**
< 3 stools/week.

**Diagnosis**
By history; differentiate between acute constipation (persistent change in bowel habits for < 3 months), and chronic constipation (persistent change in bowel habits > 3 months). Constipation with absence of flatus is a hallmark of obstruction and is termed obstipation.

**Common causes**
Diet related (fluid, fiber), lack of physical activity, medications (especially opiates and anticholinergics), medical illness (IBS, diabetes, hypothyroidism), depression, neurologic disease (Parkinson’s disease, multiple sclerosis), fecal impaction.

**Treatment**
Depends on cause:
- Short-term: Stool softeners; enema if suppository fails. Fecal disimpaction (if present).
- Long-term: Encourage dietary changes (increasing fiber and fluid consumption).
- If dietary changes fail, assess colonic transit time. Defecography or anometry may prove helpful.
**Diarrhea**

- **Definition**: Passage of > 3 loose stools/day.
- A workup may be indicated to rule out infectious or ischemic etiologies.
- Diarrhea may occur due to extensive small bowel resection (short bowel syndrome), due to disruption of innervation, or even as an expected outcome (gastric bypass).

**Diagnosis**

- Stool sample for enteric pathogens and *Clostridium difficile* toxin.
- Check stool for white blood cells (WBCs) (IBD or infectious colitis), red blood cells (RBCs) without WBCs (ischemia, invasive infectious diarrhea, cancer). Fat content (steatorrhea, seen in chronic pancreatitis).
- Colonoscopy with biopsies in chronic diarrhea to rule out microscopic colitis.

**Treatment**

Individualized based on the treatable cause, and is addressed with the specific problems that may cause diarrhea (colitis, ischemia).

---

**PsEudomEmbr Anous Colitis**

**Definition**

- An acute colitis characterized by formation of an adherent inflammatory exudate (pseudomembrane) overlying the site of mucosal injury. Most commonly due to overgrowth of *C. difficile*, a gram-positive, anaerobic, spore-forming bacillus.
- Typically occurs after broad-spectrum antibiotics (especially clindamycin, ampicillin, or cephalosporins) eradicate the normal intestinal flora.

**Signs and symptoms**

Vary from a self-limited diarrheal illness to invasive colitis with megacolon.

**Diagnosis**

Detection of *C. difficile* toxin in stool; proctoscopy or colonoscopy if diagnosis uncertain.

**Treatment**

Stop offending antibiotic; give flagyl or vancomycin PO (if patient unable to take PO, give flagyl IV).

**Prognosis**

High rate of recurrence (20%) despite high response rate to treatment.

---

**Radiation-induced Colitis**

See Table 10-1.

- Associated with external radiation therapy (XRT) to pelvis usually for endometrial, cervical, prostate, bladder, and rectal cancer.
- **Risk factors**: Atherosclerosis, diabetes, hypertension, old age, adhesions from previous abdominal operation.
### Radiation-Induced Colitis

#### Early (During Course of XRT)
- **Signs and symptoms**: Nausea, vomiting, cramps, diarrhea, tenesmus, rectal bleeding
- **Diagnosis**: Plain abdominal films, barium enema
- **Etiology**: Mucosal edema, hyperemia, acute ulceration
- **Treatment**: Treat symptoms
  - If no improvement, decrease dose of XRT or discontinue treatment

#### Late (Weeks to Years Later)
- **Signs and symptoms**: Tenesmus, bleeding, abscess, fistula involving rectum (rectal pain, stool per vagina)
- **Diagnosis**: Barium enema, CT scan
- **Etiology**: Submucosal arteriolar vasculitis, microvascular thrombosis, wall thickening, mucosal ulceration, strictures, perforation
- **Treatment**: Treat with stool softener, topical 5-ASA, corticosteroid enema
  - Strictures: Gentle dilatation or diverting colostomy after excluding cancer
  - Rectovaginal fistula: Proximal colostomy and low colorectal anastomosis or coloanal temporary colostomy

#### Chance of developing disease is dose dependent:
- < 4,000 cGy: No patients
- 5,000–6,000 cGy: Some patients
- > 6,000 cGy: Most patients

#### Ischemic Colitis

**Definition**
Acute or chronic intestinal ischemia secondary to decreased intestinal perfusion or thromboembolism:
- Embolus or thrombus of the IMA.
- Poor perfusion of mucosal vessels from arteriole shunting or spasm.

**Incidence**
Most common in the elderly.

**Risk Factors**
- Old age
- Status post abdominal aortic aneurysm (AAA) repair
- Hypertension
- Coronary artery disease, atrial fibrillation
- Cocaine abuse
- Prothrombotic conditions
- Sickle cell anemia
- Unaccustomed exertion

---

**WARD TIP**

Ischemic colitis often affects the splenic flexure.
**Typical Scenario:** A 70-year-old white male with a history of hypertension develops cramping lower abdominal pain 1 day s/p AAA repair. A few hours later he develops bloody diarrhea. What’s the diagnosis? Think: Ischemic colitis should be suspected in any elderly patient who develops acute abdominal pain followed by rectal bleeding. Furthermore, the most common setting for ischemic colitis is the early postoperative period after AAA repair when impaired blood flow through the IMA may put the colon at risk.

**Signs and Symptoms**
- Mild lower abdominal pain and rectal bleeding, classically after AAA repair.
- Pain more insidious in onset than small bowel ischemia.

**Diagnosis**
- Clinical history.
- Plain abdominal x-ray—may reveal pneumatosis (air in bowel wall) or “thumbprinting” (submucosal edema).
- Computed tomographic (CT) scan of the abdomen may reveal segmental thickening of bowel wall.
- Colonoscopy may show pale mucosa with petechial bleeding.

**Treatment**
If symptoms are mild, administer IV fluids and observe; if moderate (with fever and increased WBC), give IV antibiotics; if severe (with peritoneal signs), exploratory laparotomy with colostomy.

---

**Inflammatory Bowel Disease: Ulcerative Colitis**

**Definition**
Inflammation confined to mucosal layer of colon that extends from the rectum proximally in a continuous fashion. This is an autoimmune process.

**Incidence**
Age between 15 and 40 or 50 and 80 (bimodal age distribution).
- Whites 4× > nonwhites
- Industrialized nations >> developing nations

**Risk Factors**
- Jewish descent
- White race
- Urban dwelling
- Positive family history
- Nicotine decreases risk (unlike Crohn’s disease)

**Signs and Symptoms**
- Mild disease (confined to rectum or rectosigmoid): Intermittent rectal bleeding, passage of mucus from rectum, mild diarrhea.
- Moderate disease: Frequent loose bloody stools, mild abdominal pain, low-grade fever.
- Severe disease: Frequent loose stools, severe abdominal pain, bleeding necessitating blood transfusion. Patients may have rapid weight loss.

**Diagnosis**
- Flexible sigmoidoscopy with histopathologic evaluation of biopsies.
- Barium enema: “Lead pipe” appearance of colon due to loss of haustral folds, but no longer test of choice.

**Treatment**
- Medical: Similar to Crohn’s (see Table 10-2):
  - Mild/moderate disease: 5-ASA, corticosteroids PO or per rectum.
  - Severe disease: IV steroids.
  - Proctitis: Topical steroids.
  - Refractory disease: Immunosuppression.
### Table 10-2: Ulcerative Colitis vs. Crohn's Disease

<table>
<thead>
<tr>
<th></th>
<th>Ulcerative Colitis</th>
<th>Crohn’s Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pathology</strong></td>
<td>Inflammation of the mucosa only (exudate of pus, blood, and mucus from the “crypt abscess”)</td>
<td>Inflammation involves all bowel wall layers, which is what may lead to fistulas and abscesses</td>
</tr>
<tr>
<td></td>
<td>Always starts in rectum (up to one third don’t progress)</td>
<td>Rectal sparing in 50%</td>
</tr>
<tr>
<td></td>
<td>Limited to colon and rectum</td>
<td>May affect mouth to anus</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>Continuous lesions</td>
<td>Skip lesions: Interspersed normal and diseased bowel</td>
</tr>
<tr>
<td></td>
<td>Rare</td>
<td>Aphthous ulcers</td>
</tr>
<tr>
<td></td>
<td>Lead pipe colon appearance due to chronic scarring and subsequent retraction and loss of haustra</td>
<td>Cobblestone appearance from submucosal thickening interspersed with mucosal ulceration</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td>Perforation</td>
<td>Abscess</td>
</tr>
<tr>
<td></td>
<td>Stricture</td>
<td>Fistulas</td>
</tr>
<tr>
<td></td>
<td>Megacolon</td>
<td>Obstruction</td>
</tr>
<tr>
<td></td>
<td>Cancer</td>
<td>Cancer</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Perianal disease</td>
</tr>
</tbody>
</table>

- **Surgical:**
  - **Indications:** Failure of medical therapy, increasing risk of cancer in long-standing disease, bleeding, perforation.
  - **Procedure:** Proctocolectomy with end ileostomy or restorative J-pouch creation (curative).
  - If patient is acutely ill and unstable due to perforation, a diverting loop colostomy is indicated. Once stabilized, the patient may undergo a more definitive operation.
  - In Crohn’s disease the treatment is stricturoplasty or segmental resections because recurrence is the rule and the goal is to preserve as much healthy intestine as possible.

**Prognosis**

Approximately 1–2% risk of cancer at 10 years, and 1%/year thereafter.

### Diverticular Disease

**Definition**

- Hemiation of the mucosa through the muscular layers of the bowel wall at sites where arterioles penetrate, forming small outpouchings or diverticula.
- Are generally numerous, collectively referred to as **diverticulosis**.
- **Diverticulitis** refers to the inflammation of diverticula.

**Incidence**

- > 50% of Americans over 70 years of age.
- Men and women equally affected.

**EXAM TIP**

Unless all the colonic and rectal mucosa is removed in ulcerative colitis, the patient is still at risk for cancer.

**EXAM TIP**

Patients with UC need colonoscopy 10 years after diagnosis to screen for CRC.
Sigmoid colon most commonly involved with progressively decreasing frequency of involvement as one proceeds proximally.

**Risk factors**
- Old age
- Low-fiber diet

**Signs and symptoms**

**Diverticulosis**
- Eighty percent of patients are asymptomatic.
- **Massive, painless lower GI bleeding is classic** (notably absent in diverticulitis).

**Diverticulitis**
- Persistent abdominal pain often localized to the left lower quadrant (LLQ) with development of peritoneal signs as the disease progresses.
- LLQ and/or pelvic tenderness.
- Anorexia, nausea, vomiting, and change in bowel habits.
- Fever.
- Elevated WBC.

**Diagnosis**

**Diverticulosis**
- Characteristic history and physical exam confirmed by diverticula identified on CT/barium enema and/or colonoscopy.
- Treatment: High-fiber diet.
- See section on lower GI bleed (LGIB) for management of patients who present with acute LGIB.

**Diverticulitis**
- Characteristic history and physical exam.
- Elevated WBCs.
- CT scan (test of choice): Pericolonic inflammation with or without abscess formation.
- Barium enema and colonoscopy may induce perforation and are contraindicated in the acute setting but should be obtained in follow-up (see Figure 10-2).
- Abdominal x-ray: Ileus, distention, and/or free intraperitoneal air.

**EXAM TIP**

The diverticula of common diverticulosis are false diverticula; all the layers of the bowel wall would need to herniated to be a “true” diverticula.

**EXAM TIP**

Any generalized peritonitis in diverticulitis means it should be classified as at least Hinchey III or IV.

**EXAM TIP**

Pathophysiology of diverticulitis: A peridiverticular inflammation caused by microperforation of the diverticulum. Feces extravasate onto the serosal surface but infection is usually well contained in a patient with normal immune function.

---

**Figure 10-2.** CT scan demonstrating multiple small sigmoid diverticuli (arrows).

(Reproduced, with permission, from Gupta H, Dupuy DE. Surg Clin North Am 6(77); December 1997.)
• **Uncomplicated diverticulitis:**
  - Outpatient management: Clear liquid diet, PO antibiotics, and non-opioid analgesics with close follow-up.
  - Follow-up includes colonoscopy and dietary recommendations once acute infection has subsided.
  - If outpatient therapy fails, admit for IV antibiotics and IV hydration with bowel rest. Nasogastric tube (NGT) is placed when there is evidence of ileus or small bowel obstruction (SBO), with nausea and vomiting.
  - The Hinchey staging system is often used to describe the severity of complicated diverticulitis:
    - Stage I includes colonic inflammation with an associated pericolic abscess.
    - Stage II includes colonic inflammation with a retroperitoneal or pelvic abscess.
    - Stage III is associated with purulent peritonitis.
    - Stage IV is associated with fecal peritonitis.
      - Stage I and II are treated by IV antibiotics and CT-guided aspiration.
      - If the abscess is inaccessible to drainage and not responding to antibiotics, then it is treated surgically (drainage with Hartmann pouch or sigmoid colectomy).
      - Stage III and IV need operative management.
      - In the emergent setting, a Hartmann’s procedure is performed.
      - Elective resection of affected bowel may be considered in the patient who has recurrent episodes of diverticulitis requiring treatment or complications of diverticulitis.
    - All patients with diverticulitis should undergo a full colonoscopy 4–6 weeks after the attack to rule out malignancy, as sometimes colon cancer mimics diverticulitis.

**Prognosis**

One third of patients remain asymptomatic, one third have episodic pain, and one third progress to have a recurrence. Chronic complications include stricture, colovesical/colovaginal fistula.

**Lower GI Bleed (LGIB)**

**Definition**

- GI bleeding distal to the ligament of Treitz.
- LGIB is considered massive when the patient requires 3 or more units of blood within 24 hours.
- Most common causes are diverticulosis and angiodysplasia. Other causes include cancer, IBD, ischemic colitis, hemorrhoids.
- Anticoagulation treatment increases the risk for LGIB.

**Management**

See Table 10-3.
### Table 10-3. Management of Lower GI Bleeding (LGB)

<table>
<thead>
<tr>
<th>Diverticulosis</th>
<th>Angio Dysplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incidence</strong></td>
<td>50% of patients are &gt; 60</td>
</tr>
<tr>
<td><strong>Character</strong></td>
<td>Painless, &gt; 60% site of bleeding proximal to splenic flexure</td>
</tr>
<tr>
<td><strong>Quantity and rate</strong></td>
<td>Massive and rapid</td>
</tr>
<tr>
<td><strong>Signs and symptoms</strong></td>
<td>Melena and/or hematochezia often with symptoms of orthostasis</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>First rule out upper GI bleeding with nasogastric lavage</td>
</tr>
<tr>
<td></td>
<td>To identify site of bleed:</td>
</tr>
<tr>
<td></td>
<td>1. Colonoscopy</td>
</tr>
<tr>
<td></td>
<td>2. $\geq 0.5 \text{ mL/min}$: Bleeding scan with $^{99m}$Tc-sulfur colloid identifies bleeding; label lasts for up to 24 hours so a patient can be easily rescanned when rebleeding occurs after a negative initial scan</td>
</tr>
<tr>
<td></td>
<td>3. $\geq 1 \text{ mL/min}$: Angiography (selective mesenteric angiography best method to diagnose angiodysplasia)</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>1. Resuscitation</td>
</tr>
<tr>
<td></td>
<td>2. Therapeutic options if site identified: Octreotide, embolization, vasoconstriction with epinephrine, vasodestruction with alcohol or sodium compounds, or coagulation/cautery with heat</td>
</tr>
<tr>
<td></td>
<td>3. If site identified but bleeding massive or refractory, segmental colectomy</td>
</tr>
<tr>
<td></td>
<td>4. Without identification of bleeding site and persistent bleeding in an unstable patient, an exploratory laparotomy with possible total abdominal colectomy with ileostomy</td>
</tr>
<tr>
<td><strong>Cause</strong></td>
<td>Disruption of arteriole at either dome or antimesenteric neck of diverticulum (almost always on mucosal side, so bleed occurs into lumen rather than into peritoneal cavity)</td>
</tr>
<tr>
<td></td>
<td>Chronic intermittent obstruction of submucosal veins secondary to repeated muscular contractions results in dilated venules with incompetent precapillary sphincters and thus arteriovenous communication</td>
</tr>
<tr>
<td><strong>Prognosis</strong></td>
<td>10% overall mortality</td>
</tr>
</tbody>
</table>

### Large Bowel Obstruction

**in CE**

Most commonly occurs in elderly patients; much less common than SBO.

**Signs and Symptoms**

Abdominal distention, cramping abdominal pain, nausea, vomiting, obstipation.

**Diagnosis**

- Supine and upright abdominal films: Distended proximal colon, air-fluid levels, and no distal rectal air.
- Establish 8- to 12-hour history of obstipation; passage of some gas or stool indicates partial bowel obstruction.
- Barium enema: May be necessary to distinguish between ileus and obstruction.
1. Correction of fluid and electrolyte abnormalities.
2. Nasogastric tube for intestinal decompression (as gastric emptying is reflexly inhibited).
3. Broad-spectrum IV antibiotics (e.g., cefoxitin).
4. Relieve obstruction surgically (colonic obstruction is a surgical emergency since a nasogastric tube will not decompress the colon).

**Volvulus**

**Definition**

Rotation of a segment of intestine about its mesenteric axis; characteristically occurs in the sigmoid colon (75% of cases) or cecum (25%).

**Incidence**

More than 50% of cases occur in patients over 65.

**Risk Factors**

- Elderly (especially institutionalized patients).
- Chronic constipation.
- Psychotropic drugs.
- Hypermobile cecum secondary to incomplete fixation during intrauterine development (cecal volvulus).

**Signs and Symptoms**

See Large Bowel Obstruction.

**Diagnosis**

- Clinical presentation.
- Abdominal films: Markedly dilated sigmoid colon or cecum with a “kidney bean” appearance.
- Barium enema: Characteristic “bird’s beak” at areas of colonic narrowing.

**Treatment**

- Cecal volvulus: Right hemicolecotomy.
- Sigmoid volvulus (see Figure 10-3):
  - Sigmoidoscopy with rectal tube insertion to decompress the volvulus.
  - Emergent laparotomy if sigmoidoscopy fails or if strangulation or perforation is suspected.
  - Elective resection in same hospital admission to prevent recurrence (nearly 50% of cases recur after nonoperative reduction).
Pseudo-obstruction (Ogilvie Syndrome)

**Definition**

Massive colonic dilation without evidence of mechanical obstruction.

**Incidence**

More common in older, institutionalized patients.

**Risk Factors**

- Severe infection, recent surgery or trauma.
- Polypharmacy, particularly antipsychotics.

**Signs and Symptoms**

Marked abdominal distention with mild abdominal pain and decreased or absent bowel sounds.

**Diagnosis**

- Abdominal radiograph with massive colonic distention.
- Exclude mechanical cause for obstruction with water-soluble contrast enema and/or colonoscopy.

**Treatment**

- NGT and rectal tube for proximal and distal decompression, respectively.
- Correction of electrolyte abnormalities.

**WARD TIP**

Ogilvie syndrome is associated with any severe acute illness, neuroleptics, opiates, malignancy, and certain metabolic disturbances.

**WARD TIP**

In Ogilvie syndrome, pharmacologic decompression of the bowel with neostigmine is particularly useful but should be performed in a monitored setting. Do not give neostigmine if bradycardic!
- Discontinue narcotics, anticholinergics, or other offending medications.
- Consider pharmacologic decompression with neostigmine. Neostigmine is a cholinesterase inhibitor and is contraindicated in patients with cardiac disease.
  - Colonoscopic decompression is an alternative to neostigmine with similar success.
- If peritoneal signs develop, the patient should undergo prompt exploratory laparotomy to treat possible perforation.
- Refractory cases may need total colectomy.

**Benign Tumors of the Large Bowel**

**Color ECAl polyps**

**Morphology**

Can be classified into sessile (fat) and pedunculated (on a stalk).

**Histologic types**

- Inflammatory (pseudopolyp): Seen in UC.
- Lymphoid: Mucosal bumps containing intramucosal lymphoid tissue; no malignant potential.
- Hyperplastic: Overgrowth of normal tissue; no malignant potential.
- Adenomatous: Premalignant; are classified as tubular (75%), tubulovillous (15%), and villous (10%).
- Hamartomatous: Normal tissue arranged in abnormal configuration; juvenile polyps, Peutz–Jeghers polyps.

**Incidence**

Thirty to 40% of individuals over 60 in the United States.

**Signs and Symptoms**

- Asymptomatic (most common)
- Melena
- Hematochezia
- Mucus
- Change in bowel habits

**Diagnosis**

Flexible endoscopy (sigmoidoscopy or colonoscopy).

**Treatment**

- Colonoscopic resection.
- If colonoscopic resection not possible a segmental colon resection is indicated.

**Polyposis Syndromes**

See Table 10-4.
### Table 10-4: Polyposis Syndromes of the Bowel

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Inheritance Pattern</th>
<th>Risks/Associated Findings</th>
</tr>
</thead>
</table>
| Familial polyposis coli (FAP)                      | Autosomal dominant  | • Hundreds to thousands of polyps develop between the second and fourth decades; colon cancer inevitable without prophylactic colectomy  
• Caused by abnormal gene on chromosome 5, APC gene  
• Indication for operation: Polyps  
• Operations:  
  1. Proctocolectomy with ileostomy  
  2. Colectomy with ileorectal anastomosis  
  3. Proctocolectomy with ileal pouch—anal anastomosis |
| Gardner’s syndrome                                 | Autosomal dominant  | Innumerable polyps with associated osteomas, epidermal cysts, and fibromatosis; colon cancer inevitable without surgery |
| Turcot’s syndrome                                  | Autosomal dominant  | Multiple adenomatous colonic polyps with central nervous system (CNS) tumors (especially gliomas) |
| Peutz–Jeghers syndrome                            | Autosomal dominant  | Hamartomatous polyps of the entire GI tract with melanotic pigmentation of face, lips, oral mucosa, and palms; increased risk for cancer of the pancreas, cervix, lung, ovary, and breast |
| Hereditary nonpolyposis colon cancer syndrome (HNPCC or Lynch syndrome) | Autosomal dominant  | Lynch syndrome I: Patients without multiple polyps who develop predominantly right-sided colon cancer at a young age. Lynch syndrome II: Same as Lynch I but additional risk for extracolonic adenocarcinomas of the uterus, ovary, cervix, and breast |

### Colorectal Carcinoma (CRC)

**In CE**
- Second most common cause of cancer deaths overall (behind lung cancer).
- 130,000 new cases and 55,000 deaths each year.
- Incidence increases with increasing age starting at age 40 and peaks at 60–79 years of age.
- See Table 10-5 for screening recommendations from the U.S. Preventative Services Task Force.

### Risk Factors
- Age > 50.
- Personal history of resected colon cancer or adenomas.
- Family history of colon cancer or adenomas.
- Low-fiber, high-fat diet.
- Inherited colorectal cancer syndrome (familial adenomatous polyposis [FAP], hereditary nonpolyposis colon cancer [HNPCC]).
- Long-standing UC or Crohn’s disease.

**Exam Tip**

At diagnosis of CRC: 10% in situ disease; one third local disease; one third regional disease; 20% metastatic disease.
### Screening Guidelines for Colorectal Cancer

<table>
<thead>
<tr>
<th>Population</th>
<th>Initial Age</th>
<th>Recommended Screening Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average risk</td>
<td>50 years</td>
<td>Annual FOBT or Flexible sigmoidoscopy every 5 years or Annual FOBT and flexible sigmoidoscopy every 5 years or Air-contrast barium enema every 5 years or Colonoscopy every 10 years</td>
</tr>
<tr>
<td>Adenomatous polyps</td>
<td>50 years</td>
<td>Colonoscopy at first detection; then colonoscopy every 3 years If no further polyps, colonoscopy every 5 years If polyps, colonoscopy every 3 years Annual colonoscopy for &gt; 5 adenomas</td>
</tr>
<tr>
<td>Colorectal cancer</td>
<td>At diagnosis</td>
<td>Pretreatment colonoscopy; then at 12 months after curative resection; then colonoscopy after 3 years; then colonoscopy every 5 years, if no new lesions</td>
</tr>
<tr>
<td>Ulcerative colitis, Crohn’s colitis</td>
<td>At diagnosis; then after 8 years for pancolitis, after 15 years for left-sided colitis</td>
<td>Colonoscopy with multiple biopsies every 1–2 years</td>
</tr>
<tr>
<td>FAP</td>
<td>10–12 years</td>
<td>Annual flexible sigmoidoscopy Upper endoscopy every 1–3 years after polyps appear</td>
</tr>
<tr>
<td>Attenuated FAP</td>
<td>20 years</td>
<td>Annual flexible sigmoidoscopy Upper endoscopy every 1–3 years after polyps appear</td>
</tr>
<tr>
<td>HNPCC</td>
<td>20–25 years</td>
<td>Colonoscopy every 1–2 years Endometrial aspiration biopsy every 1–2 years</td>
</tr>
<tr>
<td>Familial colorectal cancer first-degree relative</td>
<td>40 years or 10 years before the age of the youngest affected relative</td>
<td>Colonoscopy every 5 years Increase frequency if multiple family members are affected, especially before 50 years</td>
</tr>
</tbody>
</table>

FAP = familial adenomatous polyposis; FOBT = fecal occult blood testing; HNPCC = hereditary nonpolyposis colon cancer.


---

### Ad En o mA-CAr Gi n o mAs EQu En CE

Normal $\rightarrow$ hyperproliferative $\rightarrow$ early adenoma $\rightarrow$ intermediate adenoma $\rightarrow$ late adenoma $\rightarrow$ carcinoma ($\rightarrow$ metastatic disease).

1. APC gene loss or mutation
2. Loss of DNA methylation
3. Ras (gene) mutation
4. Loss of DCC gene
5. Loss of p53 gene

### Signs and Symptoms

- Typically asymptomatic for a long period of time; symptoms, if present, depend on location and size.

---

**WARD TIP**

Rule out metastases from colorectal cancer with chest CT, CT of abdomen and pelvis. Measure carcinoembryonic antigen (CEA) to establish a baseline level.
Right-sided cancers: Occult bleeding with melena, anemia, and weakness.
Left-sided cancers: Rectal bleeding, obstructive symptoms, change in bowel habits and/or stool caliber.
Both: Weight loss, anorexia.

**Diagnosis**
- Colon cancer: Flexible sigmoidoscopy or colonoscopy (need to evaluate entire colon and rectum to look for synchronous lesions).
- Rectal cancer: Digital rectal exam, proctoscopy/colonoscopy, barium enema, also consider transrectal ultrasound (TRUS), CT, or magnetic resonance imaging (MRI) to assess depth of local tumor invasion and local lymph node status.

**Staging and Prognosis**

**TNM System (more current system)**

- **Tx:** Primary tumor cannot be assessed
- **T0:** No evidence of primary tumor
- **Tis:** Carcinoma in situ: intraepithelial or invasion of lamina propria
- **T1:** Invasion of submucosa
- **T2:** Invasion of muscularis propria
- **T3:** Invasion of subserosa, or nonperitonealized pericolic or perirectal tissues
- **T4:** Invasion of visceral peritoneum/direct invasion of other organs
- **Nx:** Regional lymph nodes cannot be assessed
  - **N0:** No nodal disease
  - **N1:** 1–3 pericolic or perirectal lymph nodes
  - **N2:** 4 or more lymph nodes
  - **N3:** 4 or more lymph nodes
- **M0:** No evidence of distant metastasis
- **M1:** Distant metastasis

**Treatment**
- Surgical resection (see Table 10-6 and Figure 10-5):
  - Goal is to remove primary tumor along with lymphatics draining involved bowel.
  - In rectal cancer, the circumferential radial margin (CRM) is crucial to local recurrence. Total mesorectal excision (TME) reduces the rates of local recurrence.
- Adjuvant treatment:
  - Stage III: 5-fluorouracil (5-FU)-based chemotherapy.
  - Rectal cancer: Preop radiation using 5-FU as a radiosensitizer (this sequence is called “neoadjuvant” therapy because it occurs prior to the definitive surgical treatment).

**WARD TIP**
Microcytic anemia in an elderly male or postmenopausal woman is colon cancer until proven otherwise.

**EXAM TIP**
CRC 5-year survival by stage:
- **Stage I:** T1N0M0/T2N0M1 (90%)
- **Stage II:** T3N0M0/T4N0M0 (75%)
- **Stage III:** any Tn1M0/any Tn2–3M0 (50%)
- **Stage IV:** any Tany Nm1 (5%)
### Hemorrhoids

<table>
<thead>
<tr>
<th>Tumor Location</th>
<th>Operation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cecum</td>
<td>Right hemicolectomy:</td>
</tr>
<tr>
<td></td>
<td>- Resection of terminal ileum, cecum, ascending and proximal transverse colon</td>
</tr>
<tr>
<td>Right colon</td>
<td>Right hemicolectomy</td>
</tr>
<tr>
<td>Proximal/mid-transverse colon</td>
<td>Extended right hemicolectomy:</td>
</tr>
<tr>
<td></td>
<td>- Resection as above plus remainder of transverse colon and splenic flexure</td>
</tr>
<tr>
<td>Splenic flexure and left colon</td>
<td>Left hemicolectomy:</td>
</tr>
<tr>
<td></td>
<td>- Resection of distal transverse, descending and sigmoid colon</td>
</tr>
<tr>
<td>Sigmoid or rectosigmoid colon</td>
<td>Sigmoid colectomy</td>
</tr>
<tr>
<td>Proximal rectum</td>
<td>Low anterior resection (LAR):</td>
</tr>
<tr>
<td></td>
<td>- Tumors &gt; 4 cm from anal verge</td>
</tr>
<tr>
<td></td>
<td>- Must be able to get 2-cm margin</td>
</tr>
<tr>
<td></td>
<td>- Includes total mesorectum excision</td>
</tr>
<tr>
<td></td>
<td>- Involves complete mobilization of rectum, with division of lateral ligaments, posterior mobilization through Waldeyer's fascia to tip of coccyx, dissection between rectum and vagina or prostate</td>
</tr>
<tr>
<td></td>
<td>- Complications: Incontinence, urinary dysfunction, sexual dysfunction, anastomotic leak (5–10%), stricture (5–20%)</td>
</tr>
<tr>
<td>Distal rectum</td>
<td>Abdominal-perineal resection (APR):</td>
</tr>
<tr>
<td></td>
<td>- Tumors too low for LAR (less than 2 cm from anorectal ring)</td>
</tr>
<tr>
<td></td>
<td>- Involves creation of end ostomy, with resection of rectum, total mesorectal excision (TME), and closure of anus</td>
</tr>
<tr>
<td></td>
<td>- Complications: Stenosis, retraction or prolapose of ostomy, perineal wound infection</td>
</tr>
<tr>
<td>Other situations</td>
<td>Obstructing cancer: Attempt to decompress</td>
</tr>
<tr>
<td></td>
<td>Perforated cancer: Remove disease and perforated segments</td>
</tr>
<tr>
<td></td>
<td>Synchronous or metachronous lesions, or proximal perforation with distal cancer: Subtotal colectomy with ileosigmoid or ileorectal anastomosis</td>
</tr>
<tr>
<td></td>
<td>Very distal rectal tumor and/or patient not stable for big operation: Transanal excision of tumor, endoscopic microsurgery, or endocavitary radiation</td>
</tr>
<tr>
<td></td>
<td>En-bloc resection for malignant fistulas</td>
</tr>
</tbody>
</table>

### Perianal and Anal Problems

**Hemorrhoids**

See Table 10-7.

- Prolapse of the submucosal veins located in the left lateral, right anterior, and right posterior quadrants of the anal canal.
- Classified by type of epithelium: Internal if covered by columnar mucosa (above dentate line), external if covered by anoderm (below dentate line), and mixed if both types of epithelia are involved.
- **Incidence:** Male = female.
**FIGURE 10-5.** Terminology of types of colorectal resections. A to C, ileocecectomy; + A + B to D, ascending colectomy; + A + B to F, right hemicolectomy; + A + B to G, extended right hemicolectomy; + E + F to G + H, transverse colectomy; G to I, left hemicolectomy; F to I, extended left hemicolectomy; J + K, sigmoid colectomy; + A + B to J, subtotal colectomy; + A + B to K, total colectomy; + A + B to L, total proctocolectomy.

**TABLE 10-7.** Grade Description, Symptoms, and Treatments of Internal Hemorrhoids

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Protrudes into lumen, no prolapse</td>
<td>Bleeding</td>
<td>Nonresectional measures&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>II</td>
<td>Prolapse with straining, spontaneous return</td>
<td>Bleeding, perception of prolapse</td>
<td>Nonresectional measures</td>
</tr>
<tr>
<td>III</td>
<td>Prolapse, requires manual reduction</td>
<td>Bleeding, prolapse, mucous soilage, pruritus</td>
<td>Consider trial of nonresectional measures; many require excision</td>
</tr>
<tr>
<td>IV</td>
<td>Prolapse cannot be reduced</td>
<td>Bleeding, prolapse, mucous soilage, pruritus, pain&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Excision</td>
</tr>
</tbody>
</table>

<sup>a</sup>Nonresectional methods (rubber-band ligation, infrared coagulation, or injection sclerotherapy) can be used in insensate tissue only (above the dentate line).

<sup>b</sup>Pain if thrombosed or ischemic.

- **Risk factors:** Constipation, pregnancy, increased pelvic pressure (ascites, tumors), portal hypertension.
- **Diagnosis:** Clinical history, physical exam, visualize with anoscope.

### Anal Fissure

**Definition**

Painful linear tears in the anal mucosa below the dentate line; induced by constipation or excessive diarrhea.

**Signs and Symptoms**

- Pain with defecation.
- Bright red blood on toilet tissue.
- Markedly increased sphincter tone and extreme pain on digital examination.
- Visible tear upon gentle lateral retraction of anal tissue.

**Diagnosis**

History and physical exam.

**Treatment**

- Sitz baths.
- Fiber supplements, bulking agents.
  - Medical management with stool softeners, nitropaste, Botox, or diltiazem.
  - Increased fluid intake.
  - Topical nifedipine or nitroglycerine ointment.
  - Chemodenervation of the internal sphincter with Botulinum toxin injection.
  - Lateral internal sphincterotomy.

### Anal or Rectal Abscess

**Definition**

Obstruction of anal crypts with resultant bacterial overgrowth and abscess formation within the intersphincteric space.

**Risk Factors**

- Constipation/diarrhea/IBD.
- Immunocompromise.
- History of recent surgery or trauma.
- History of CRC.
- History of previous anorectal abscess.

**Signs and Symptoms**

Rectal pain, often of sudden onset, with associated fever, chills, malaise, leukocytosis, and a tender perianal swelling with erythema and warmth of overlying skin.

**Treatment**

Surgical drainage.

**Typical Scenario:** A 24-year-old male with chronic constipation complains of intense anal pain. He has a tender, swollen, bluish lump at the anal orifice. What is the treatment? Think: This young man's history of constipation and physical exam findings are classic for a thrombosed external hemorrhoid. The treatment is surgical excision if the pain has been present for < 48 hours or is persistent. Pain typically subsides after 48 hours, and treatment is symptomatic.

**Exam Tip**

- Anal fissures are always posterior and midline.
Anorectal fistulas

**Definition**

Tissue tracts (abnormal connections between two areas) originating in the glands of the anal canal at the dentate line that are usually the chronic sequelae of anorectal infections, particularly abscesses.

**Classification of anorectal Fistulas**

1. **Intersphincteric (most common):** Fistula tract stays within intersphincteric plane.
2. **Transsphincteric:** Fistula connects the intersphincteric plane with the ischiorectal fossa by perforating the external sphincter.
3. **Suprasphincteric:** Similar to transsphincteric, but the fistula loops above the external sphincter to penetrate the levator ani muscles.
4. **Extraspincteric:** Fistula passes from rectum to perineal skin without penetrating sphincteric complex.

**Signs and Symptoms**

Recurrent or persistent perianal drainage that becomes painful when one of the tracts becomes occluded.

**Diagnosis**

- Digital rectal exam.
- Anoscopy.
- If the internal opening cannot be identified by direct probing, it should be identified by probing the external opening or by injecting methylene blue or hydrogen peroxide into the tract.

**Treatment**

Intraoperative unroofing of the entire fistula tract with or without placement of setons (vessel loop placed in the fistula tract to keep it patent for drainage).

---

**Goodsall’s rule** (Figure 10-6) can be used to clinically predict the course of an anorectal fistula tract. Imagine a line that bisects the anus in the coronal plane. Any fistula that originates anterior to the line will course anteriorly in a direct route. Fistulae that originate posterior to the line will have a curved path.

**Figure 10-6.** Goodsall’s rule to identify the internal opening of fistulas-in-ano.

**Pilonidal Disease**

**Definition**
A cystic inflammatory process generally occurring at or near the cranial edge of the gluteal cleft.

**Incidence**
Most commonly seen in hirsute men in their late teens to the third decade.

**Signs and Symptoms**
Can present acutely as an abscess (fuctuant mass) or chronically as a draining sinus with pain at the top of the gluteal cleft.

**Treatment**
- Acute presentation: incision and drainage under local anesthesia with removal of involved hairs.
- Chronic disease: Hair removal and observation or excision.

---

**Anal Cancer**

**Definition**
Neoplasms of the anorectal region that are classified into tumors of the perianal skin (anal margin carcinomas) and tumors of the anal canal.

**Incidence**
Rare (1–2% of all colon cancers).

**Risk Factors**
- Human papillomavirus (HPV)
- Human immunodeficiency virus (HIV)
- Cigarette smoking
- Multiple sexual partners
- Anal intercourse
- Immunosuppressed state

**Signs and Symptoms**
Often asymptomatic; can present with anal bleeding, a lump, or itching; an irregular nodule that is palpable or visible externally (anal margin tumor) or a hard, ulcerating mass that occupies a portion of the anal canal (anal canal tumor).

**Diagnosis**
- Surgical biopsy with histopathologic evaluation.
- **Histology**: Anal margin tumors include squamous and basal cell carcinomas, Paget’s disease, and Bowen’s disease. Anal canal tumors are usually...
epidermoid (squamous cell carcinoma or transitional cell/cloacogenic carcinoma) or malignant melanoma.

- **Clinical staging:** Involves history, physical exam, colonoscopy, abdominal or pelvic CT or MRI, CXR, and liver function tests.

  - Epidermoid carcinoma of anal canal: Chemoradiation is mainstay—5-FU, mitomycin C, and external beam radiation (Nigro protocol) surgery is reserved for recurrence.
  - Other anal margin tumors: Wide local excision alone or in combination with radiation and/or chemotherapy is successful in 80% of cases without abdominal-perineal resection (APR) if tumor is small and not deeply invasive.
  - Anal canal tumors: Local excision not an option; combined chemotherapy (5-FU and mitomycin C) with radiation often successful; APR if chemoradiation fails.

- **Prognosis**
  - Anal margin tumors: 80% overall 5-year survival.
  - Anal canal tumors:
    - Epidermoid carcinoma: 50% overall 5-year survival.
    - Malignant melanoma: 10–15% 5-year survival.
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Embryology and Development

- The appendix begins to bud off from the cecum at around the sixth week of embryological development.
- The base of the appendix remains in a fixed position with respect to the cecum, whereas the tip can end up in various positions (Figure 11-1).

Anatomy/Structure

- **Mesoappendix** is the mesentery that suspends the appendix from the terminal ileum. It contains the **appendicular artery**, the blood supply to the appendix.
  - The appendicular artery is a branch of the ileocolic artery.
  - The ileocolic artery is a branch of the superior mesenteric artery.
- The appendix is composed of the same layers as the colon wall.
  - Mucosa, submucosa, inner circular muscle, outer longitudinal muscle, serosa.
  - The three distinct bands of outer longitudinal muscle, the **taeniae coli**, converge on the appendix.
- Although many have claimed that the appendix is merely a vestigial organ, it is actually an immunological organ and secretes IgA. However, it is not an essential organ and can be removed without immunological compromise.
- The length can range from 2 to 20 cm but averages 6–9 cm.
- Luminal capacity is < 1 mL.

---

**OR TIP**

After locating the cecum, one can easily find the appendix by following the three taeniae coli until they converge at the base of the appendix.

**EXAM TIP**

The lifetime incidence of acute appendicitis in the United States is about 7%.
Acute Appendicitis

**Incidence**

One of the most common acute surgical diseases.
- Highest in early adulthood, at the peak of lymphoid tissue growth.
- Second peak in the incidence of appendicitis occurs in the elderly.
- There is a higher incidence of appendicitis in males than females (1.3:1).

**Pathophysiology**

The probable sequence of events in acute appendicitis is:

1. **Luminal obstruction.**
   - In young patients, more commonly by lymphoid tissue hyperplasia.
   - In older patients, fecalith is an increasingly common cause of obstruction.

2. **Distention** and increased intraluminal pressure.
   - The appendiceal mucosa continues to secrete normally despite being obstructed.
   - The resident bacteria multiply rapidly, further increasing intraluminal pressure.

3. Venous congestion.
   - The intraluminal pressure eventually exceeds capillary and venule pressures.
   - Arteriolar blood continues to flow in, causing vascular congestion and engorgement.

4. Impaired blood supply renders the mucosa ischemic and susceptible to bacterial invasion.

5. **Inflammation** and ischemia progress to involve the serosal surface of the appendix.

**Symptoms**

- Right lower quadrant (RLQ) pain.
- Migration of pain from the periumbilical region to localize in the RLQ.
- Nausea/vomiting.
- Fever.

**Signs**

- Direct rebound tenderness, which is maximal at (or around) McBurney’s point.
- Leukocytosis.
- Rovsing’s sign: Palpation pressure exerted over left lower quadrant (LLQ) causes pain in RLQ.
- Iliopsoas sign: Pain on extension at the right hip.
  - The patient will get relief by flexing the right thigh at the hip, which relaxes the psoas muscle.
  - This sign signifies retrocecal appendicitis.
- Obturator sign (signifies pelvic appendicitis): Pelvic pain on internal rotation of the right thigh.

**WARD TIP**

The initial dull, diffuse (visceral) pain that occurs at the onset of acute appendicitis is a result of the stimulation of visceral afferent stretch fibers. These nerve endings fire as a result of the sudden-onset distention, and the pain is commonly felt around the umbilicus (T10 distribution).

**EXAM TIP**

If clinical presentation is most likely appendicitis (umbilical pain migrating to RLQ, fever, nausea and vomiting, elevated WBC) go straight to appendectomy. Imaging is not necessary.

**WARD TIP**

The shift from dull, diffuse (visceral) pain to sharp, localized (somatic) RLQ pain occurs when the inflamed serosa contacts the parietal peritoneum, causing peritoneal irritation. This pain is felt in the area directly overlying the appendix.

**EXAM TIP**

McBurney’s point: One third the distance along a line drawn from the right anterior superior iliac spine to the umbilicus.
**Different Diagnoses**

### Gastrointestinal Conditions
- Gastroenteritis
- Mesenteric adenitis
- Meckel’s diverticulum
- Intussusception
- Typhoid fever
- Primary peritonitis

### Genitourinary Conditions
- Ectopic pregnancy
- Pelvic inflammatory disease
- Ovarian torsion/cyst/tumor
- Urinary tract infection/pyelonephritis
- Ureteral stone

### Labs
- **Complete blood count:**
  - Leukocytosis (> 10,000 in 90% of cases), usually with concomitant left shift (polymorphonuclear neutrophil [PMN] predominance).
  - Consider perforation or abscess if WBC > 18,000.
- **Urinalysis:**
  - Helpful in ruling out genitourinary causes of symptoms.
  - RBCs and WBCs may be present secondary to extension of appendiceal inflammation to the ureter.
  - Significant hematuria or pyuria, and bacteriuria from a catheterized specimen should suggest underlying urinary tract pathology.
- **Abdominal x-ray:**
  - Diagnostic Imaging
  - Not particularly useful in most cases.
  - May reveal appendicolith/fecalith (< 15% of cases).
- **Abdominal CT with contrast:**
  - Very sensitive (95–98%) and somewhat specific (83–90%).
  - Useful in identifying several other inflammatory processes that may present similarly to appendicitis.
  - Positive findings include:
    - Dilatation of appendix to > 6 mm in diameter.
    - Thickening of appendiceal wall (representing edema).
    - Periappendiceal streaking (densities within perimesenteric fat).
    - Presence of appendicolith (see Figure 11-2).
- **Graded compression ultrasonography:**
  - Sensitivity of 85% and specificity of 92% for diagnosing appendicitis.
  - Positive finding: Enlarged (> 6 mm), noncompressible appendix.
  - Especially useful in ruling out gynecologic pathology.
  - Useful in pregnancy.
  - MRI is used in pregnancy instead of CT scan.

### Treatment
- See Figure 11-3.
- Preoperative: IV fluids plus antibiotics.
- Prompt appendectomy (open or laparoscopic) is recommended.
- Laparoscopic approach is preferred. It is associated with reduction in surgical site infection, shorter hospital stay, and reduction in long-term risk of bowel obstruction.
For free perforation: Peritoneal washout and parenteral antibiotics.
- For localized perforation (with abscess) CT scan guided drainage and interval appendectomy. (An “interval appendectomy” is an elective appendectomy performed after successful medical management of appendicitis to prevent recurrence. Its necessity remains controversial.)
- If greater than 5 days of onset of symptoms, or if abscess is present, one can manage medically with antibiotics. No appendectomy is done unless there is failure to respond to antibiotics or appendicitis recurs.

Figure 11-2. Abdominal CT scan demonstrating appendicolith and acute appendicitis.

Figure 11-3 A-K. Technique of open appendectomy. A: Incision. B: After delivery of the tip of the cecum, the mesoappendix is divided. C: The base is clamped and ligated with a simple throw of the knot. The next step—inversion of the stump—is optional. D: A clamp is placed to hold the knot during inversion with a purse-string suture of fine silk. E: The loosely tied inner knot on the stump assures that there is no closed space for the development of a stump abscess. (A-E: Reproduced, with permission, from Doherty GM. Current Diagnosis & Treatment: Surgery. 14th ed. New York, NY: McGraw-Hill Education; 2015. Figure 28-1.) (Continued)
**Figure 11.3 A-K** F-K Laparoscopic appendectomy technique. (F-K Reproduced, with permission, from Zinner MJ, Ashley SW. Maingot’s Abdominal Operations. 12th ed. New York, NY: McGraw-Hill Education; 2013. Figure 31-8.)
Appendicitis in Special Populations

**Pregnant Patients**
- Appendicitis is the most common surgical emergency in pregnant patients.
- Fetal mortality increases 3–8% with appendicitis and 30% with perforation.
- Surgery is the standard treatment, though 10–15% of women will experience premature labor.

**Elderly Patients**
- Tend to present atypically, leading to delayed diagnosis.
  - Present later in the course and with less pain, **may present as a small bowel obstruction**.
  - Delayed leukocytosis.
  - Higher risk of perforation and higher mortality than in younger patients.

**Immunocompromised Patients (e.g., AIDS Patients, Recipients of High-dose Chemotherapy)**
- Although they may not have absolute leukocytosis, compared to baseline WBC count, they will demonstrate relative leukocytosis.
- The differential diagnosis is expanded to include opportunistic infections such as cytomegalovirus (CMV)-related bowel perforation and neutropenic colitis.

Appendiceal Neoplasms

**Carcinoid**
- A relatively low-grade neuroendocrine tumor (it secretes enzymes aberrantly; the enzymes typically cause nausea, diarrhea, and flushing).
  - The appendix is the most common site of carcinoid tumors in the GI tract.
  - Carcinoid is the second most common type of appendiceal tumor (commonest being mucinous adenocarcinoma).

**Diagnosis**
- Increased urinary 5-hydroxyindoleacetic acid (5-HIAA) and increased serum serotonin.

**Treatment**
- Size is the major determinant of treatment and malignant potential:
  - Tumors < 2 cm are treated with appendectomy.
  - Tumors > 2 cm or at base of the appendix, are treated with right hemicolectomy.
  - Serotonin antagonists (e.g., cyproheptadine) or somatostatin analogues (e.g., octreotide) can be used for symptoms of carcinoid syndrome.

**WARD TIP**
The enlarged uterus may push the appendix up, explaining why pregnant patients may present with RUQ pain.

**EXAM TIP**
Patients will not typically have carcinoid syndrome unless the tumor has metastasized to the liver.

**EXAM TIP**
Patients with carcinoid syndrome should also be monitored for pellagra/vitamin B/niacin deficiency (3 Ds—dementia, dermatitis, and diarrhea), because tryptophan is used up to make serotonin, instead of niacin.
**Intraperitoneal Mucinous Tumors**

- Can rupture, causing **pseudomyxoma peritonei** with mucin implants on peritoneal surfaces and the omentum.
  - More common in women (ratio of 3:2).
  - Complications include bowel obstruction and perforation.
  - Have been associated with migratory thrombophlebitis.

**Adenocarcinoma**

- Colon cancer that arises from the appendix; very rare and almost never diagnosed preoperatively.
- Rapid spread to regional lymph nodes, ovaries, and peritoneal surfaces.
- If confined to appendix and local lymph nodes, right hemicolecction is the treatment of choice.

**Appendiceal Abscess**

- **Signs and symptoms:** Similar to acute appendicitis.
  - Increasing RLQ pain.
  - Tender, fluctuant RLQ mass that is palpable on rectal examination.
  - Anorexia.
  - Fever.
  - Localizing peritonitis.
  - Leukocytosis.
- **Diagnosis:** Confirmed by CT scan.
- **Treatment:** Percutaneous or operative drainage.

**Recent Advances**

Natural orifice transluminal endoscopic surgery (NOTES): In this procedure the appendix is removed via upper gastrointestinal endoscopy with the surgeon operating through the gastric wall and ultimately removing the appendix through the mouth without any external scar. The gastrotomy is closed from within the stomach.

- It can also be done via the vagina in females.
- NOTES is only being performed in limited centers worldwide under clinical study protocols.
Overview

- Hernias are a common surgical problem.
- It is estimated that 10% of the population develops some types of hernia during life and that they are present in 3–4% of the male population.
- Fifty percent are indirect inguinal hernias, 25% are direct inguinal, and 15% are femoral.
- The male-to-female ratio is 7:1.
- Abdominal wall hernias are the most common condition requiring major surgery.

Definitions

- **Hernia:** A protrusion of a viscus through an abnormal opening in the wall of a cavity in which it is contained.

Classification of Hernias

- **External hernia:** The sac protrudes completely through the abdominal wall. Examples: Inguinal (indirect and direct), femoral, umbilical, and epigastric.
- **Intraparietal hernia:** The sac is contained within the abdominal wall. Example: Spigelian hernia.
- **Internal hernia:** The sac is within the visceral cavity. Examples: Diaphragmatic hernias (congenital or acquired) and the small intestine herniating in the paraduodenal pouch.
- **Reducible hernia:** The protruding viscus can be returned to the abdomen.
- **Irreducible (incarcerated) hernia:** The protruding viscus cannot be returned to the abdomen.
- **Strangulated hernia:** The vascularity of the viscus is compromised—**surgical emergency.**

General Groin Anatomy

See Figure 12-1.

Layers of the abdominal wall

Skin, subcutaneous fat, Scarpa’s fascia, external oblique muscle, internal oblique muscle, transversus abdominis muscle, transversalis fascia, peritoneal fat, and peritoneum.

Internal structures

- Inguinal canal: Length, 4 cm; boundaries:
  - Anterior wall: External oblique aponeurosis.
  - Posterior wall: Transverse abdominal muscle aponeurosis and transversalis fascia.
- Spermatic cord: Begins at the deep ring and contains the vas deferens and its artery (descend to the seminiferous tubules), one testicular artery and two to three veins, lymphatics (incline superiorly to the kidney region), autonomic nerves, and fat.
**Genital:**
- Location: Travels along with the cremaster vessels to form a neurovascular bundle.
- Originates: From L1 and L2.
- Motor and sensory: Innervates the cremaster muscle, skin of the side of the scrotum, and labia.
- May substitute for the ilioinguinal nerve if it’s deficient.
- Iliohypogastric, ilioinguinal nerves, and the genital branch of the genitofemoral nerve:
  - Iliohypogastric and ilioinguinal intertwine.
  - Originates: From T12 and L1.
  - Sensory: To skin of groin, base of penis, and medial upper thigh.
- Genital branch of genitofemoral nerve: Located on top of the spermatic cord in 60% of people but can be found behind or within the cremaster muscle. Often cannot be found or is too small to be seen.

**femoral canal structures**

From lateral to medial: Nerve, Artery, Vein, Empty space, Lymph nodes.

**anatomical triangles**

- **Hesselbach’s triangle:** The triangular area in the lower abdominal wall. It is the site of direct inguinal hernia. The boundaries of Hesselbach’s triangle are:
  - Inferior border: Inguinal ligament.
  - Medial border: Rectus abdominis.
  - Lateral border: Inferior epigastric vessels (lateral umbilical fold).
- **Triangle of Grynfeltt** (superior lumbar) bounded by the 12th rib superiority and the internal oblique muscle anteriorly, with the floor composed of fibers of the quadratus lumbarum muscle.

**WARD TIP**

The inguinal area is examined with the patient standing and facing the physician. Presence of a discrete bulge in the inguinal area reveals the hernia. Valsalva's maneuver and cough may accentuate the bulge, making it clearly visible or palpable.

**WARD TIP**

Memory aid for the femoral canal structures: n a V el.

**EXAM TIP**

med’s lie: Medial to inferior epigastric vessels in Direct hernia, Lateral to inferior epigastric vessels in Indirect hernias.
- **Triangle of Petit** (inferior lumbar triangle) bounded by:
  - Posteriorly: Latissimus dorsi muscle.
  - Anteriorly: External oblique muscle.
  - Inferiorly: Iliac crest.
  - The floor is composed of fibers from the internal oblique and transversus abdominis muscle.

### Inguinal Hernia

- Hernias arising above the abdominocrural crease.
- Most common site for abdominal hernias.
- Males: Indirect > direct (2:1).
- Female: Direct is rare.
- Incidence, strangulation, and hospitalization all increase with age.
- Cause 15–20% of intestinal obstructions.

#### Risk Factors

- Abdominal wall hernias occur in areas where aponeurosis and fascia are devoid of protecting support of striated muscle.
- They can be congenital or acquired by surgery or muscular atrophy.
- Female predisposition to femoral hernias: Increased diameter of the true pelvis as compared to men, proportionally widens the femoral canal.
- Muscle deficiency of the internal oblique muscles in the groin exposes the deep ring and floor of the inguinal canal, which are further weakened by intra-abdominal pressure.
- Connective tissue destruction (transverse aponeurosis and fascia): Caused by physical stress secondary to intra-abdominal pressure; smoking; aging; connective tissue disease; systemic illnesses; fracture of elastic fibers; alterations in structure, quantity, and metabolism of collagen.
- Other factors: Abdominal distention, ascites with chronic increase in intra-abdominal pressure, and peritoneal dialysis.

#### Symptoms

- Asymptomatic: Some patients have no symptoms.
- Symptomatic: Wide variety of nonspecific discomforts related to the contents of the sac and the pressure by the sac on adjacent tissue.
- Pain: Worse at the end of the day and relieved at night when patient lies down (because the hernia reduces).
- Groin hernias do not usually cause testicular pain. Likewise, testicular pain doesn’t usually indicate the onset of a hernia.

#### Diagnosis

Physical exam: In the standing position, have patient strain or cough. The hernia sac with its contents will enlarge and transmit a palpable impulse.

#### Differential Diagnosis

See Table 12-1.

#### Radiology

Hernia is a clinical diagnosis, and radiological tests (ultrasound/CT scan) are done only in special circumstances (e.g., morbid obesity, where clinical examination is not reliable).
**Table 12-1. Differential diagnosis of Croin Hernia**

<table>
<thead>
<tr>
<th>Malignancy</th>
<th>Lymphoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retropitoneal sarcoma</td>
<td>Metastasis</td>
</tr>
<tr>
<td>Testicular tumor</td>
<td>Primary testicular</td>
</tr>
<tr>
<td>Varicoce</td>
<td>Epididymitis</td>
</tr>
<tr>
<td>Testicular torsion</td>
<td>Hydrocele</td>
</tr>
<tr>
<td>Hydrole</td>
<td>Ectopic testicle</td>
</tr>
<tr>
<td>Undescended testicle</td>
<td>Femoral artery aneurysm or pseudoaneurysm</td>
</tr>
<tr>
<td>Lymph node</td>
<td>Sebaceous cyst</td>
</tr>
<tr>
<td>Hidradenitis</td>
<td>Cyst of the canal of Nuck (female)</td>
</tr>
<tr>
<td>Saphenous varix</td>
<td>Psoas abscess</td>
</tr>
<tr>
<td>Hematoma</td>
<td>Ascites</td>
</tr>
</tbody>
</table>


---

**Management**

**Principles of Treatment**

- Tension-free repair of the hernia defect.
- Repair using fascia, aponeurosis, or mesh.
- Suture material used should hold until fibrous tissue is formed over it.
- Resuscitation in case of strangulated hernia with gangrene, with shock, or with intestinal obstruction.

**Nonsurgical**

- No role of medical management in a patient who can tolerate surgery.
- Can be considered in moribund patients.
- Hernia truss is a device to keep a reducible hernia contained by external pressure. Its use is very limited in modern practice.

**Surgical**

- Treatment modality of choice.
- Herniotomy is the operation where hernia sac is identified, freed, its neck ligated, and the sac reduced. This may be sufficient in young, muscular individuals and in children.
- Herniorrhaphy and hernioplasty are herniotomy along with repair of the posterior wall of the inguinal canal and the internal ring.
Types of Groin Hernia Surgery

**Open**

- **Tissue repair:** Uses the patient’s tissue for reinforcement.
  - Bassini’s repair: Suturing of conjoint tendon to the incurved part of inguinal ligament. Bassini’s repair is still widely practiced as it is simple to perform with good results.
  - Shouldice repair: Double breastling of transversalis fascia. Technically challenging with excellent results.
  - McVay’s repair/Cooper’s repair (used for femoral hernias): Conjoint tendon sutured to Cooper’s ligament.
- **Mesh repair:** Uses prosthetic mesh and/or plug for tension-free repair (preferred).
  - Anterior approach: For example, Lichtenstein’s repair (most common).
  - Posterior approach: For example, Kugel’s method, Stoppa’s repair.

**Laparoscopic**

- Requires an experienced laparoscopic surgeon, has decreased postop pain, requires general or regional anesthesia, and more expensive. Wound infection has been shown to decrease with laparoscopic repair.
  - Transabdominal pre-peritoneal procedure (TAPP).
  - Totally extraperitoneal procedure (TEP)—preferred.
- The choice of hernia surgery depends on the surgeon’s expertise. TEP is being performed in larger numbers as surgical proficiency in laparoscopy increases, and the results are promising (see Figure 12-2).

---

**Figure 12-2.** Trocar placement for (a) transabdominal preperitoneal repair and (b) totally extraperitoneal repair. (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz’s Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015. Figure 37-24.)
indications for surgery

Generally, all hernias should be repaired unless the risks of surgery outweigh the benefits of the repair.

complications of groin hernia surgery

- Chronic groin pain.
  - Cord and testicular (hematoma, injury of vas deferens, ischemic orchitis, testicular atrophy).
  - Mesh-related complications (infection, erosion, displacement, contraction, fracture).
  - Laparoscopic and general complications.

prognosis

Recurrence:

- Recurrence rates of about 1% are seen with the Shouldice repair and as low as 0.2% with the tension-free mesh repair.
- Caused by excessive tension on repair, deficient tissue, inadequate hernioplasty, or overlooked hernias.
- More common with direct hernias.

Classification of Inguinal Hernias

- Direct
- Indirect

direct inguinal hernia

A direct inguinal hernia enters the inguinal canal through its weakened posterior wall. The hernia does not pass through the internal ring.

- Lies posterior to the spermatic cord.
- Practically never enters the scrotum.
- Wide neck (strangulation uncommon).
- Occurs almost exclusively in males.
- Common in older age groups.
- Common in smokers due to weakened connective tissue.
- Predisposing factors: Hard labor, cough, straining, and so on.
- Can lead to damage to the ilioinguinal nerve.

symptoms

- Bulge in groin.
- Dull dragging pain in the inguinal region referred to testis.
- Pain increases with hard work and straining.

indirect inguinal hernia

Herniation through the internal inguinal ring traveling to the external ring. If complete, can enter the scrotum while exiting the external ring.

- If congenital, associated with a patent processus vaginalis.
- Bilateral in one third of cases.
- Most common hernia in both males and females.

WARD TIP

A reducible hernia in a patient with ascites should not be corrected until the ascites is controlled.
- Occurs at all ages.
- More common in males than in females.
- In the first decade of life, the right-sided hernia is more common than left (because of late descent of right testis).

### Femoral Hernia

A form of indirect hernia arising out of the femoral canal beneath the inguinal ligament (medial to the femoral vessels).
- Female-to-male ratio of 2:1.
- Males affected are in a younger age group.
- Rare in children.
- Uncommon—around 2.5% of all groin hernias.
- Left side 1:2 right side: Secondary to the sigmoid colon tamponading the left femoral canal.
- Common in elderly patients.
- High incidence of incarceration due to narrow neck.
- Twenty-two percent strangulate after 3 months, and 45% after 21 months.

#### Anatomy

- The femoral canal is 1.25 cm long and arises from the femoral ring to the saphenous opening.
- Femoral sac originates from the femoral canal through a defect on the medial side (common) or the anterior (uncommon) side of the femoral sheath.

#### Symptoms

- Dull dragging pain in the groin, with swelling.
- If obstructed, can cause vomiting and constipation.
- If strangulated, can lead to severe pain and shock.
- Swelling arises from below the inguinal ligament.

#### Differential Diagnosis

- Inguinal hernia.
- Saphenous varix.
- Enlarged femoral lymph node.
- Lipoma.
- Femoral artery aneurysm.
- Psoas abscess.

### Acquired Umbilical Hernia

- Abdominal contents herniate through a defect in the umbilicus.
- Common site of herniation, especially in females.

#### Associated Factors

Ascites, obesity, and repeated pregnancies.
Complications

- Strangulation of the colon and omentum is common.
- Rupture occurs in chronic ascitic cirrhosis. Emergency portal decompression is needed.

Treatment

- Surgical repair:
  - < 2 cm fascial defect: Closed by loosely placed polypropylene suture.
  - > 2 cm fascial defect: Managed with a prosthesis repair.
  - Mayo hemioplasty is the classical repair (not used often).

Pediatric Umbilical Hernia

- Secondary to a fascial defect in the linea alba with protruding abdominal contents, covered by umbilical skin and subcutaneous tissue.
- Caused by a failure of timely closure of the umbilical ring, and leaves a central defect in the linea alba.
- Common in infants.
- Incarceration is rare and reduction is contraindicated.

Management

Usually close spontaneously within 3 years if the defect is < 1.0 cm. Surgical repair indicated if:

- The defect > 2 cm.
- Child is > 4–5 years of age.
- Protrusion is disfiguring and disturbing to the child and parents.

Pediatric Wall Defects

See Pediatric Surgery chapter.

Esophageal Hiatal Hernia

- A hernia in which an anatomical part (such as the stomach) protrudes through the esophageal hiatus of the diaphragm.
- Three types of esophageal hiatal hernia are identified:
  1. The sliding hernia, type I, characterized by an upward dislocation of the cardia in the posterior mediastinum.
  2. The rolling or paraesophageal hernia, type II, characterized by an upward dislocation of the gastric fundus alongside a normally positioned cardia.
  3. The combined sliding-rolling or mixed hernia, type III, characterized by an upward dislocation of both the cardia and the gastric fundus.

The end stage of type I and type II hernias occurs when the whole stomach migrates up into the chest by rotating 180° around its longitudinal axis, with the cardia and pylorus as fixed points. In this situation, the abnormality is usually referred to as an intrathoracic stomach.
Sliding esophageal hernia (type i)
- The gastroesophageal junction and the stomach herniate into the thoracic cavity.
- These account for more than 90% of all hiatal hernias.
- Can lead to reflux and esophagitis that can predispose to Barrett’s esophagus.
- Management can be done medically with antacids and head elevation.
- Only 15% require surgery, consisting of wrapping of the stomach fundus around the lower esophageal sphincter (Nissen fundoplication).
- See diagram in GERD section of Esophagus chapter.

Paraesophageal hiatal hernia (type ii)
- Herniation of the stomach into the thorax by way of the esophageal hiatus, without disruption of the gastroesophageal junction.
- Rare (<5%).
- High frequency of complications (i.e., obstruction, strangulation, and hemorrhage).
- **Warrants prompt surgical correction** via transthoracic or transabdominal approach.
- Both laparoscopic and open approaches can be used.
- Reduction, repair with or without fundoplication is done.

Other Hernias
- **Richter’s hernia:** Only part of the intestine wall circumference is in the hernia. May strangulate without obstruction. Seen commonly in femoral and obturator hernias.
- **Littre’s hernia:** The hernial sac contains Meckel’s diverticulum. It may become inflamed.
- **Garengoff’s hernia:** The hernial sac has the appendix. Importance is that it may form an infamed hernia.
- **Pantaloon hernia:** A combination of a direct and an indirect inguinal hernia.
- **Maydl’s hernia:** W type of intestinal loop herniates; may strangulate with the gangrenous part being inside the abdomen, or may be reduced into the abdomen without noticing the gangrenous part.
- **Spigelian hernia:** The sac passes through the spigelian or semilunaris fascia.
- **Sliding inguinal hernia:** Any hernia in which part of the sac is the wall of a viscus. On the right, the cecum, ascending colon, or appendix is commonly involved; on the left, the sigmoid colon is involved.
- **Cooper’s hernia:** Hernia that involves the femoral canal and tracts to the labia majora in females and to the scrotum in males.
- **Lumbar hernia:** Divided into congenital, spontaneous, traumatic, and incisional. Can pass through the triangle of Grynfeltt, through the inferior lumbar triangle of Petit, or previous incision.
- **Perineal hernia:** Located through pelvic diaphragm, anterior (passes through labia majora—females only) or posterior (male: enters the ischiorectal fossa; female: close to the vagina) to the superficial transverse perineal muscle.
- **Incisional hernia:** Resulting as a surgical complication. These could enlarge beyond repair. Associated with obesity, diabetes, and infection.
- **Eventration:** Loss of integrity of the abdominal wall, reducing the intra-abdominal pressure, and resulting in external herniation of bowel.
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Hepatic Anatomy

See Figure 13-1.

- Arterial supply:
  - Celiac trunk comes off the aorta, giving off the left gastric, splenic, and common hepatic arteries.

![Hepatic Anatomy Diagram](image-url)
- Common hepatic artery divides into the hepatic artery proper and the gastroduodenal artery.
- Hepatic artery divides into the right and left hepatic arteries.
- Venous drainage: The left, middle, and right hepatic veins drain into the inferior vena cava (IVC).
- Ligaments of the liver:
  - Falciform ligament: Connects the anterior abdominal wall to the liver; contains the ligamentum teres (obliterated umbilical vein).
  - Coronary ligament: Peritoneal reflection on the cranial aspect of the liver that attaches it to the diaphragm.
  - Triangular ligaments: The right and left lateral extensions of the coronary ligament.
  - Bare area: The posterior section of the liver against the diaphragm; has no peritoneal covering.
- Glisson's capsule: The peritoneal membrane that covers the liver.
- Cantlie’s line (portal fssure): A line that passes from the left side of the gallbladder to the left side of the IVC, dividing the liver into right and left lobes. It is based on blood supply and is the basis of the four classic types of hepatic resection.
- Liver enzymes: Aspartate transaminase (AST) and alanine transaminase (ALT) are made by hepatocytes; alkaline phosphatase (alk phos) is made by ductal epithelium.

**Diagnostic Imaging**

**Ultrasound**
- Ultrasound remains the cornerstone for diagnosing gallstones and is a great tool as it is noninvasive, fast, inexpensive, and very reliable for diagnosing gallstones.
- Its limitation is that it is user dependent.
- It should be the first test to be performed in all patients with a suspicion of gallstones.
- Can also detect masses and cysts.
- Test of choice in pregnancy.

**Cholangiogram**
- Provides image of the biliary tree.
- Oral and IV cholangiography—obsolete.
- T-tube cholangiogram: Performed after open exploration of the bile duct to rule out retained stones or strictures just before removal of the tube.
- Intraoperative cholangiogram: Performed on the operating table via cannulation of the cystic duct to confirm the anatomy and diagnose CBD stones.

**Hepatobiliary iminodiacetic acid (hida) scan**
- A radionucleotide scan in which patient’s own RBCs tagged with technetium-99m labeled iminodiacetic acid is injected intravenously which are taken up by the hepatocytes.
- Components in interpreting a HIDA scan
  - Normal uptake in the liver—if abnormal suggests hepatic dysfunction.
  - Uptake in the gallbladder—if absent—suggests cystic duct obstruction—cholecystitis.

**Liver Blood Supply**
- The right and left hepatic arteries supply 50% of the liver’s oxygen. The portal vein supplies the remaining 50%.
- However, the liver receives 75% of its blood supply from the portal vein and only 25% from the hepatic arteries. The different degrees of oxygen saturation within the arterial and venous systems account for this fact.

**Hepatoduodenal ligament**
- The hepatoduodenal ligament contains the common bile duct (CBD), portal vein, and proper hepatic artery. It forms the anterior boundary of the epiploic foramen of Winslow and connects the greater and the lesser peritoneal cavities.
• Uptake in the extrahepatic biliary tree—if absent—suggests CBD obstruction
• Uptake in the duodenum and small intestine—if absent—suggests CBD obstruction at the level of the papilla
• A normal gallbladder would be visualized within 1 hour. Most sensitive for acute cholecystitis.
• Drawback is that it is time consuming and stones are not visualized.

**Computed tomography (CT) scan**

• Useful for hepatic lesions and for visualizing lymph nodes.
• Not very useful for the biliary tree.
• Expensive.
• Contraindicated in pregnancy (radiation exposure).

**Endoscopic retrograde cholangiopancreatography (ERCP)**

• Involves passage of a side-viewing endoscope into the duodenum, introduction of a catheter into the ampulla of Vater, and injection of contrast medium into the CBD and/or pancreatic duct; conscious sedation is necessary.
• Can be diagnostic (biopsy and show pancreatic cancer or cholangiocarcinoma) and therapeutic (remove stones, or stent an obstruction).
• One percent risk of pancreatitis from procedure.

**ERCP with sphincterotomy (papillotomy)**

• A cut through the sphincter of Oddi to allow the passage of stones from the CBD into the duodenum. Care to be taken to avoid injury to CBD and/or pancreatic duct.
• Often performed during ERCP but can also be performed as part of open surgery (sphincteroplasty).

**Percutaneous transhepatic cholangiography (PTC)**

• Involves passing a needle through the skin and subcutaneous tissues into the hepatic parenchyma and advancement into a peripheral bile duct. When bile is aspirated, a catheter is introduced through the needle and radiopaque contrast medium is injected.
• It is very useful in cases with distal obstruction where ERCP is not possible or has failed.
• It requires either USG or CT guidance.

**Bile**

• Constituents: Cholesterol, lecithin, bile acids, and bilirubin.
• Function: Emulsifies fats.
• Enterohepatic circulation: Bile acids are released from the liver into the duodenum, reabsorbed at the terminal ileum, and transported back to the liver via the portal vein.
• Cholecystokinin:
  • Released by duodenal mucosal cells.
  • Stimulates gallbladder contraction and release of bile (along with vagal stimulation).
  • Causes opening of the ampulla of Vater and slows gastric emptying.
- Is stimulated by fat, protein, amino acids, and hydrochloride (HCl).
- Trypsin and chymotrypsin inhibit its release.

Jaundice

definition

Yellowing of the skin and sclera due to an elevation in total bilirubin > 2.5. Categorized into prehepatic, hepatic, or posthepatic causes (see Table 13-1).

signs and symptoms

- Yellow skin and sclera.
- Pruritus.
- Can have hepatomegaly, tenderness of the right upper quadrant (RUQ), or signs of cirrhosis.
- Dark urine, clay-colored stools, anorexia, and nausea indicate obstructive jaundice.

diagnosis

See Table 13-1.

treatment

Treat the underlying disorder.

Liver Injury

See Table 13-2.

initial treatment

- Airway, breathing, circulation (ABCs).
- Ascertain details about mechanism of injury.

<table>
<thead>
<tr>
<th>Classification</th>
<th>Causes</th>
<th>Direct Bilirubin</th>
<th>Indirect Bilirubin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prehepatic</td>
<td>Hemolysis, Gilbert’s disease, Crigler-Najjar syndrome</td>
<td>Normal</td>
<td>High</td>
</tr>
<tr>
<td>Hepatic</td>
<td>Alcoholic cirrhosis, acute hepatitis, primary biliary cirrhosis</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Posthepatic</td>
<td>Gallstones, tumor</td>
<td>High</td>
<td>Normal</td>
</tr>
</tbody>
</table>
Table 13-2. American association for Surgery of Trauma (aaST) Liver injury Scale (1994 revision)

<table>
<thead>
<tr>
<th>Grade*</th>
<th>Type of Injury</th>
<th>Description of Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Hematoma</td>
<td>Subcapsular, &lt; 10% surface area</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear, &lt; 1 cm parenchymal depth</td>
</tr>
<tr>
<td>II</td>
<td>Hematoma</td>
<td>Subcapsular, 10–50% surface area; intraparenchymal &lt; 10 cm in diameter</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Capsular tear, 1–3 parenchymal depth, &lt; 10 cm in length</td>
</tr>
<tr>
<td>III</td>
<td>Hematoma</td>
<td>Subcapsular, &gt; 50% surface area of ruptured subcapsular or parenchymal hematoma;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>intraparenchymal hematoma; intraparenchymal hematoma &gt; 10 cm or expanding 3-cm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>parenchymal depth</td>
</tr>
<tr>
<td></td>
<td>Laceration</td>
<td>Parenchymal disruption involving 25–75% hepatic lobe or 1–3 Couinaud's segments</td>
</tr>
<tr>
<td>IV</td>
<td>Laceration</td>
<td>Parenchymal disruption involving &gt; 75% of hepatic lobe or &gt; 3 Couinaud’s segments</td>
</tr>
<tr>
<td></td>
<td></td>
<td>within a single lobe</td>
</tr>
<tr>
<td>V</td>
<td>Vascular</td>
<td>Juxtahepatic venous injuries (i.e., retrohepatic vena cava/central major hepatic veins)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hepatic avulsion</td>
</tr>
</tbody>
</table>

*Advance one grade for bilateral injuries up to grade III

- CT: Will detect blood and solid organ damage; is useful for grading injury. CT is contraindicated in the unstable or marginal patient.
- Ultrasound, if accessible, should be performed initially as focused abdominal sonography for trauma (FAST), and may then be used for serial examinations following delineation of injury on CT.

**nonoperative management**

- Approximately one half of patients are eligible.
- For penetrating trauma: Operative management remains standard of care.
- For blunt trauma: May attempt trial of observation if:
  - Patient is stable or stabilizes after fluid resuscitation.
  - There are no peritoneal signs.
  - The injury can be precisely delineated and graded by CT scan.
  - There are no associated injuries requiring laparotomy.
  - There is no need for excessive hepatic-related blood transfusions.
  - Repeat CT scan in 2–3 days to look for expansion or resolution of injury.
  - Patients may resume normal activities after 2 months.

**operative management**

- As a rule, any hemodynamically unstable patient due to a liver injury should be explored.
  - Generally needed for 20% of patients with grade III or higher injuries who present with hemodynamic instability due to hemorrhage.
  - Laparotomy is undertaken through a long midline incision.

**EXAM TIP**

Though the liver has been found to be protective against other organ injuries, for the same reasons (size, position), it is very vulnerable to injury itself.

**EXAM TIP**

Use hemodynamic stability as the deciding factor in choosing operative or nonoperative management.

**EXAM TIP**

Imaging of the liver: If contrast pool or blush is noted on CT and patient remains stable, consider an angiogram by interventional radiology for possible embolization.
The primary goal is the control of bleeding with direct pressure and packing. Patient should then be resuscitated as needed, with attention to temperature control, volume status, and acid–base balance.

Specifics of trauma liver surgery include:
- Pringle maneuver: Occlusion of the portal triad manually or with an atraumatic vascular clamp.
- Finger fracture of liver to expose damaged vessels and bile ducts.
- Debridement of nonviable tissue.
- Placement of an omental pedicle (with its blood supply) at the site (using omentum to plug up bleeding site).
- Closed suction drainage.
- Major hepatic resection is indicated when the parenchyma was totally destroyed by the trauma, the extent of injury is too great for packing, the injury itself caused a near-resection, or resection is the only way to control life-threatening hemorrhage.
- Damage control surgery: Packing the perihepatic space with a planned reoperation in 24–36 hours is indicated when the patient is severely coagulopathic, there is bilobar bleeding that cannot be controlled, there is a large expanding hematoma, other methods to control bleeding have failed, or the patient requires transfer to a level I trauma center.

Complications:
- Hemorrhage (5%).
- Hemobilia (1%): Signs and symptoms—upper gastrointestinal (GI) bleed, RUQ pain, positive fecal occult blood, and jaundice.
- Abscess.
- Biliary fistula (7–10%): Definition—> 50 mL/d drainage for > 14 days.

**Nontraumatic Liver Conditions**

**Hepatic Abscesses**

**Definition**
A collection of pus in the liver of bacterial, fungal, or parasitic origin that most commonly involves the right lobe. The two main subtypes are pyogenic (bacterial) and amebic.

**Incidence**
- Pyogenic: 8–15/100,000
- Amebic: 1.3/100,000

**Risk Factors**
- Pyogenic: Usually secondary to bacterial sepsis or biliary or portal vein infection; can also occur from a perforated infected gallbladder, cholangitis, diverticulitis, liver cancer, or liver metastases.
- Amebic: Patients from Central America, homosexual men, institutionalized patients, and alcoholics.

**Signs and Symptoms**
Fever, chills, RUQ pain, RUQ abdominal tenderness, jaundice, sepsis, and weight loss; amebic abscesses tend to have a more protracted course.

**Diagnosis**
- Leukocytosis.
- Elevated liver function tests (LFTs).
Ultrasound, CT, or MRI of the liver.

Serology for amebic abscesses.

**Treatment**

- Pyogenic: Ultrasound or CT-guided percutaneous drainage with IV antibiotics; operative drainage indicated if percutaneous attempts fail or cysts are multiple or loculated.
- Amebic: Operative drainage not indicated unless abscesses do not resolve with IV metronidazole or are superinfected with bacteria.

**Prognosis**

Mortality is low for uncomplicated abscesses, but complicated abscesses carry a 40% mortality risk.

**Hydatid Cysts**

**Definition**

A hepatic cyst caused by Echinococcus multilocularis or Echinococcus granulosus that is usually solitary and involves the right lobe of the liver.

**Risk Factors**

Exposure to dogs, sheep, cattle, foxes, wolves, domestic cats, or foreign travel.

**Signs and Symptoms**

Most commonly asymptomatic; can cause hepatomegaly.

**Diagnosis**

Often picked up incidentally on ultrasound, CT, or abdominal films, which may show calcifications outlining the cyst; eosinophilia, serology. Also will have positive Casoni skin test.

**Treatment**

Never aspirate these cysts or they may spill their contents leading to anaphylactic shock. Treat with albendazole or mebendazole followed by resection.

**Nonparasitic Cysts**

**Definition**

Benign cysts within the liver parenchyma that most commonly involve the right lobe; are thought to be of congenital origin.

**Incidence**

Rare; 4:1 female-to-male ratio.

**Signs and Symptoms**

Most cysts are small and asymptomatic; large cysts (rare) can present with increasing abdominal pain and girth and can bleed or become infected.

**Diagnosis**

Usually incidental; ultrasound or CT.
Small asymptomatic cysts require no treatment; large, symptomatic cysts should be surgically treated.

**Benign Liver Tumors**

**Cavernous Hemangioma**

**Definition**
A benign vascular tumor resulting from abnormal differentiation of angioblastic tissue during fetal life; usually located in the right posterior segment of the liver.

**Incidence**
Most common tumor of the liver; occurs at all ages.

**Signs and Symptoms**
Usually asymptomatic; rarely presents with pain, a mass, biliary obstruction or hepatomegaly.

**Diagnosis**
Usually discovered incidentally; can be detected by ultrasound, CT, magnetic resonance imaging (MRI), radionuclide scan, or arteriography; do not biopsy, as hemorrhage can occur.

**Treatment**
Surgical resection if symptomatic or in danger of rupture; otherwise, observe.

**Hemangioma**

**Definition**
- A benign focal lesion of the liver that consists of normal tissue that has differentiated in an abnormal fashion.
- Multiple subtypes, depending on the types of cells involved (e.g., bile duct hamartoma, mesenchymal hamartoma).

**Incidence**
Rare.

**Signs and Symptoms**
Typically asymptomatic; can present with RUQ pain or fullness.

**Diagnosis**
Usually discovered incidentally during radiologic imaging; may require histopathologic evaluation.

**Treatment**
Surgical excision is usually needed for diagnosis.

**Hepatocellular Adenomas**

**Definition**
A mass lesion of the liver characterized by a benign proliferation of hepatocytes.

**Kasabach–Merritt Syndrome**
Consumptive thrombocytopenia in patients with a hepatic hemangioma. Can lead to bleeding.

**Hepatocellular Adenomas**
Present with abdominal pain secondary to tumor rupture or bleeding in approximately one third of patients.

**Typical Scenario:** A 27-year-old female presents to her obstetrician with a history of a hepatocellular adenoma that resolved after discontinuing oral contraceptives (OCPs). She now wants to get pregnant. Does she have any specific health risks? Think: Hepatocellular adenomas treated by cessation of OCPs rather than by resection are at risk for rupture and hemorrhage during future pregnancies.
Incidence
Ninety-five percent occur in women of childbearing years.

Risk factors
OCP use, long-term anabolic steroid therapy, glycogen storage disease.

Signs and Symptoms
- Abdominal pain.
- Abdominal mass.
- Bleeding (from spontaneous rupture of large tumors).
- Can also be asymptomatic.

Diagnosis
- Ultrasound demonstrates solitary, well-demarcated, heterogeneous mass.
- CT scan with IV contrast—well marginated and isoattenuating to liver. They demonstrate transient relatively homogenous enhancement returning to near isodensity on portal venous and delayed phase.

Treatment
- Cessation of OCPs
- Surgical excision is recommended as it can be a precursor for hepatocellular carcinoma.

Focal Nodular hyperplasia (FNH)
Definition
A benign hepatic tumor, thought to arise from hepatocytes and bile ducts, that has a characteristic “central scar” on pathologic evaluation.

Incidence
Most common in premenopausal females.

Signs and Symptoms
Usually asymptomatic; 10% of patients present with abdominal pain and/or an RUQ mass.

Diagnosis
- Usually incidental on ultrasound or CT. Classically a central scar is seen.
- Can be differentiated from hepatocellular adenoma by a Tc-99 study.

Treatment
Resect if patient is symptomatic (abdominal pain or compressive symptoms) or there is diagnostic uncertainty.

Malignant liver tumors

Hepatocellular Carcinoma (hepatoma)
Definition
A malignant tumor derived from hepatocytes frequently found in association with chronic liver disease, particularly cirrhosis.

Incidence
- Accounts for 80% of liver cancers, but < 2% of all cancers.
- Much more common in males (3:1).
- Usually diagnosed in the fifth or sixth decade.
RISK FACTORS
- Hepatitis B
- Hepatitis C
- Cirrhosis
- Smoking and alcohol
- Aflatoxins (found in peanuts)
- Liver flukes
- Hemochromatosis
- α₁-antitrypsin deficiency
- Anabolic steroid use
- Carbon tetrachlorides (found in cleaning agents)

SYMPTOMS
- Weight loss
- Weakness
- Dull pain in the RUQ or epigastrium
- Nausea, vomiting
- Jaundice
- Nontender hepatomegaly
- Splenomegaly (33%)
- Ascites (50%)

DIAGNOSIS
- Increased alk phos, AST, ALT, γ-glutamyl transferase (GGT), α-fetoprotein (AFP), and des-γ-carboxy prothrombin (DCP).
- Contrast CT and ultrasound can visualize the tumor. Enhances in the arterial phase with quick washout in the late phase and portal venous phase. Usually solitary but can be multifocal or diffuse.
- CT or ultrasound-guided needle biopsy will give the definitive diagnosis.

TREATMENT
- Surgical resection is the only cure, consisting of either lobectomy or segmental resection. A 1-cm margin is required.
- Transplant is also a possibility, but there is a high recurrence rate due to the continued presence of the underlying risk factor (e.g., hepatitis B, hepatitis C, etc.). Patients are selected for liver transplantation based on the Milan criteria.
- Local chemotherapy infusion into the hepatic artery, hepatic artery embolization, and liposomal chemotherapy are the newer treatment options being explored.

PROGNOSIS
Most patients die within the first 4 months if the tumor is not resected. After resection or transplant, the 5-year survival rate is 25–40%.

METASTATIC NEOPLASMS

SYMPTOMS
- Patients are usually asymptomatic until the disease has become advanced and the liver begins to fail.
- Symptoms may include fatigue, weight loss, epigastric fullness, dull RUQ pain, ascites, jaundice, or fever.

DIAGNOSIS
- Increased alk phos, GGT, lactic dehydrogenase (LDH), AST, and ALT (nonspecific).
- Metastases will enhance on contrast CT (see Figure 13-2).
- Intraoperative ultrasound with liver palpation is the most sensitive diagnostic tool.

WARD TIP
- Abruit can commonly be heard over a hepatocellular carcinoma due to its abundant vascularity.

WARD TIP
- Milan criteria:
  - Single tumor with diameter ≤ 5 cm, or up to 3 tumors each with diameter ≤ 3 cm.

WARD TIP
- Why do ascites and anasarca occur in liver disease? Because the liver makes albumin, which is necessary to generate oncotic pressure and maintain fluids in the vasculature.

WARD TIP
- The most common hepatic malignancy is metastases. The primary is usually from colon, breast, stomach, or lung, with bronchogenic carcinoma being the most common primary cancer.
Treatment

- Resection, if possible, is the treatment of choice.
- Radiofrequency ablation (RFA) is now extensively done even for multiple lesions, and the short-term results are promising.

Liver Failure

Child–Pugh Score

Child’s classification estimates hepatic reserve in patients with liver failure (see Table 13-3).

Table 13-3. Child–Pugh Score for Liver Failure

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilirubin (mg/dL)</td>
<td>&lt; 2.0</td>
<td>2.0–3.0</td>
<td>&gt; 3.0</td>
</tr>
<tr>
<td>Albumin (mg/dL)</td>
<td>&gt; 3.5</td>
<td>2.8–3.5</td>
<td>&lt; 2.8</td>
</tr>
<tr>
<td>Ascites (clinical evaluation)</td>
<td>None</td>
<td>Easily controlled</td>
<td>Poorly</td>
</tr>
<tr>
<td>Neurologic disorder</td>
<td>None</td>
<td>Minimal</td>
<td>Advanced</td>
</tr>
<tr>
<td>Prothrombin time (sec &lt; control)</td>
<td>&lt; 4.0</td>
<td>4.0–6.0</td>
<td>&gt; 6.0</td>
</tr>
</tbody>
</table>

Correlation with Child’s class: 5–6 points, Class A; 7–9 points, Class B; 10–15 points, Class C.
The Model for End-Stage Liver Disease (MELD) score is used for liver transplant allocation in the United States. The MELD formula is dependent on INR, SCr (serum creatinine level in milligrams per deciliter), and Tbil (total serum bilirubin level in milligrams per deciliter).

MELD Score = 9.57 \ln(SCr) + 3.78 \ln(Tbil) + 11.2 \ln(INR) + 6.43

where \ln represents natural logarithm.

### Portal Hypertension

**Definition**

Portal pressure > 10 mmHg (measure with indirect hepatic vein wedge pressure).

**Causes**

- Prehepatic: Congenital atresia, cyanosis, or portal vein thrombosis.
- Intrahepatic: Cirrhosis, hepatic fibrosis from hemochromatosis, Wilson’s disease, or congenital fibrosis.
- Posthepatic: Budd–Chiari syndrome (thrombosis of the hepatic veins), hypercoagulable state, lymphoreticular malignancy.

**Signs and Symptoms**

- Jaundice
- Splenomegaly
- Palmar erythema
- Spider angiomata
- Ascites
- Truncal obesity with wasting of the extremities
- Asterixis (a flapping hand tremor)
- Hepatic encephalopathy

**Portosystemic Collaterals and their Clinical manifestations**

- Left gastric vein to the esophageal veins—esophageal varices.
- Umbilical vein (via the falciform ligament) to the epigastric veins—caput medusa.
- Superior hemorrhoidal vein to the middle and inferior hemorrhoidal veins—hemorrhoids.
- Veins of Retzius (posterior abdominal wall veins) to the retroperitoneal lumbar veins—retroperitoneal varices.

**Diagnosis**

- Suggestive history and physical examination.
- Duplex Doppler ultrasound: Initial procedure of choice.
  - CT scan of the abdomen—Cirrhosis of the liver, splenomegaly, engorged and tortuous collaterals and varices, prominent abdominal wall and retroperitoneal veins.
- Venous phase of visceral angiography: Defines portal anatomy more precisely.

**Treatment**

- Aimed at reducing portal pressure.
Shunts (see Figure 13-3):

- **Splenorenal (Warren shunt)**: Connects the splenic vein to the left renal vein. Used for patients with esophageal varices and a history of bleeding.
- **End to side**: Connects the end of the portal vein to the side of the IVC. This is considered a total shunt.
- **Side to side**: Connects the side of the portal vein to the side of the IVC. This is considered a partial shunt.
- **Portacaval H graft**: A synthetic graft is attached from the portal vein to the IVC. This is considered a partial graft.
- **Mesocaval H graft**: A synthetic graft is attached from the superior mesenteric vein (SMV) to the IVC.

**Complications**: Increased incidence of hepatic encephalopathy because more toxins are diverted to the systemic circulation (except for the Warren shunt), and death from hepatic failure due to decreased blood flow to the liver.

- **Liver transplant**: Ideal candidate is a young patient with cirrhosis and an episode of bleeding from esophageal varices.
- **Medical treatment**: Beta blockers to reduce portal pressure.

---

**WARD TIP**

Splenomegaly is the most common clinical finding in portal hypertension. Removal is almost never warranted.

---

**Morbidities Resulting from Liver Failure**

**Hepatic Encephalopathy**

**Definition**

- Altered mental status due to hepatic insufficiency.
- Toxins that are normally cleared by the liver are retained in the circulation.
- The exact toxin that causes the central nervous system (CNS) changes is unknown but has been theorized to be ammonia, \(\gamma\)-aminobutyric acid (GABA), mercaptans, or short-chain fatty acids.

**Epidemiology**

- Occurs in all cases of fulminant hepatic failure.
- Occurs in one half of patients with end-stage liver disease requiring transplantation.
- Occurs in one third of patients with cirrhosis.
**Cause**
Precipitating factors include:
- Infection (watch for spontaneous bacterial peritonitis [SBP]).
- Potassium, magnesium, or other electrolyte depletion.
- Use of opiates, sedatives, or other hepatically cleared drugs.
- GI bleed.
- Excess dietary protein.

**Signs and Symptoms**
- Change in level of consciousness, mood disturbance, decreased attention span.
- Lethargy, coma.
- Normal electroencephalogram (EEG).
- Asterixis flapping tremor of hand.

**Diagnosis**
An elevated serum ammonia level along with signs of altered mental status and altered LFT is diagnostic.

**Treatment**
Lactulose and neomycin (PO or PR) to reduce intestinal formation and absorption of ammonia.

---

**Ascites**

**Definition**
- Excess fluid in the peritoneal cavity.
- Sodium and fluid retention by the kidney, low plasma oncotic pressure due to low albumin production by the failing liver, and elevated hydrostatic pressure in the hepatic sinusoids or portal veins cause fluid to be lost into the peritoneal cavity.
- Ascitic fluid is frequently the site of SBP in patients with liver disease.

**Signs and Symptoms**
Distended abdomen, fluid wave, shifting dullness.

**Treatment**
- Reduce sodium intake.
- Potassium-sparing diuretic (e.g., spironolactone).
- Abdominal paracentesis.
- Removing too much ascitic fluid or removing the fluid too quickly will cause intravascular fluid to be drawn into the peritoneal cavity. This leads to a loss of intravascular volume and can cause hypovolemic shock.

---

**Esophageal varices**

**Definition**
Engorged esophageal or gastric veins, usually resulting from increased portal pressure from liver disease.

**Signs and Symptoms**
- Asymptomatic unless rupture occurs.
- With rupture: Upper GI bleeding with hematemesis, melena, and/or hematochezia.

---

**WARD TIP**
In hepatic encephalopathy, the serum ammonia level does not correlate with the degree of encephalopathy.

---

**EXAM TIP**
Al eVe shunt is a peritoneal-jugular shunt used to decrease ascites. One drawback is that it may increase hepatic encephalopathy.
Endoscopically placed esophagogastrroduodenostomy (EGD).

**Treatment of ruptured Varices**

Options include:
- Endoscopic sclerotherapy.
- Vasopressin or somatostatin injection.
- Balloon tamponade (Sengstaken–Blakemore tube).
- Transjugular intrahepatic portacaval shunt (TIPS): This is used more for long-term management, rather than during an acute bleed; it shunts pressure away from the esophageal vessels. Also useful in patients awaiting a liver transplant with advanced cirrhosis and portal hypertension.
- Intraoperative placement of a portacaval shunt.
- Liver transplant.
- \( \beta \)-blockers can be used to decrease portal pressure and reduce the incidence of rupture.

**Prognosis**

Poor, even with treatment. Ruptured esophageal varices have a 50% death rate.

**CapUmedUsa**

**Definition**

Engorged abdominal wall veins—a sign of increased portal pressure.

**Signs and Symptoms**

Mass of veins extending from around the umbilicus, periumbilical bruit (Cruveilhier–Baumgarten bruit).

**The Biliary System**

**Anatomy of the biliary tree**

See Figure 13-4.

- Intrahepatic ducts converge to become the right and left hepatic ducts.
- Right and left hepatic ducts converge, forming the common hepatic duct.
- Cystic duct comes off the gallbladder and joins the common hepatic duct to become the CBD.
- CBD empties into the duodenum via the ampulla of Vater.

**Anatomy of the gallbladder**

- The proximal end of the gallbladder near the cystic duct is called the infundibulum, and the larger distal end of the gallbladder is called the fundus.
- The valves within the cystic duct are called the spiral valves of Heister.
- The gallbladder collects bile directly from the liver via small bile ducts called ducts of Luschka.

**OR TIP**

Calot’s triangle: Inferior border of the liver, common hepatic duct, and cystic duct. The cystic artery runs through it, and the associated lymph node is called Calot’s node. The right hepatic artery is adjacent to the cystic duct in Calot’s triangle and, as such, is susceptible to injury during cholecystectomy.

**WARD TIP**

The infundibulum of the gallbladder is also called Hartman’s pouch.
Conditions of the Gallbladder and the Biliary Tree

**Cholelithiasis**

**Definition**

Stones in the gallbladder formed from an imbalance between the contents of bile—cholesterol, bile salts, and bile pigments. Eighty-five percent of stones are composed primarily of cholesterol, while the remaining 15% are pigmented.

**Incidence**

Approximately 10% of the U.S. population has gallstones.

**Risk Factors**

- The typical ones are: Female, fat, fertile, and forty.
- Other risk factors include Native American race, pregnancy, OCPs, Western diet, inflammatory bowel disease (IBD), hyperlipidemia, ileal resection (due to loss of enterohepatic circulation), and total parenteral nutrition (due to prolonged cholestasis).

**Signs and Symptoms**

- Most patients are asymptomatic.
- Symptomatic patients classically complain of severe RUQ pain that radiates to the back, epigastrium, or left upper quadrant (LUQ) that tends to be worse after eating (especially after fatty foods) and may be associated with nausea and vomiting.
- The symptom complex is called biliary colic and typically resolves over a few hours.

**WARD TIP**

Only 15% of gallstones have enough calcium to be radiopaque. The majority of kidney stones, however, have sufficient calcium to be radiopaque on plain films.

**WARD TIP**

Risk factors for cholelithiasis:

- Female
- Fat
- Fertile
- Forty
- Flatulent
- Familial
- Fibrosis, cystic
- F-Hgb (sickle cell disease)
gnals

Often incidental, as most patients are asymptomatic.

Abdominal plain films pick up 15% of gallstones.

Ultrasound: Procedure of choice; classic findings include an acoustic shadow ("headlight") and gravity-dependent movement of gallstones with patient repositioning (see Figure 13-5).

Asymptomatic cholelithiasis does not require cholecystectomy unless the patient:

- Has a porcelain gallbladder (which has an increased incidence of carcinoma).
- Has sickle cell anemia.
- Has a stone > 2–3 cm.
- Is a pediatric patient.
- Is immunocompromised.

Symptomatic cholelithiasis requires cholecystectomy. A laparoscopic cholecystectomy can be performed on 95% of patients. Medical treatment of cholelithiasis involves chenodeoxycholic acid or ursodeoxycholic acid drugs.

**Hydrops of the gallbladder:**
Complete obstruction of the cystic duct by a gallstone, causing the gallbladder to fill with fluid from the gallbladder mucosa. The fluid is often milky white as the bile pigments are absorbed due to chronic obstruction.

**Acute Cholecystitis**

**Definition**
Inflammation of the gallbladder wall, usually due to obstruction of the cystic duct by gallstones.

**Signs and Symptoms**

- RUQ tenderness and guarding are present, usually for > 3 hours.
- Fever, nausea, vomiting, and anorexia are nonspecific and variable.
- Murphy’s sign: Pain on deep inspiration resulting in inspiratory arrest (positive in about one third of patients) upon palpation of RUQ.
- Sonographic Murphy’s: Pain over RUQ when palpated with ultrasound probe (87% sensitivity).
- One third of patients develop exquisitely tender RUQ mass late in course.

**Diagnosis**

- Labs: Leukocytosis with or without increased alk phos LFTs, amylase, and total bilirubin.
- Ultrasound: Reveals inflammation of the gallbladder wall (> 4 mm), pericholecystic fluid and stones in the gallbladder. Positive predictive value of all three is 90%. Will also see dilation of the CBD if a stone is lodged in CBD.
- HIDA scan (most sensitive): Non-filling of the gallbladder even when the small bowel is visualized is characteristic of acute cholecystitis (see Figures 13-6 and 13-7).

*Figure 13-6. Normal HIDA scan, demonstrating uptake of contrast in intrahepatic bile ducts and gallbladder at 20 minutes and excretion into small bowel at 30 minutes.*
The gallbladder is not visualized even at 4 hours, even though the small bowel is.

**Treatment**
- NPO.
- IV fluids.
- IV antibiotic (broad spectrum).
- IV analgesia.
- Cholecystectomy within 24–48 hours.
- Often done laparoscopically; if inflammation prevents adequate visualization of important structures, convert to open cholecystectomy.

**Emphysematous Cholecystitis**
- Severe variant of cholecystitis caused by gas-forming bacteria.
- Relatively rare.
- Often results in perforation of the gallbladder, high mortality and morbidity.
- Typically affects elderly diabetic men.

**a calculus Cholecystitis**

**Definition**

Acute cholecystitis without evidence of gallstones; thought to be due to biliary stasis and ischemia (low perfusion states).

**Incidence**

Ten percent of cases of acute cholecystitis.
Risk Factors
Most often seen in intensive care unit (ICU) patients with multiorgan system failure, trauma (especially after major surgery), burns, sepsis, and TPN.

Diagnosis
- Labs: Leukocytosis, with or without increased alk phos LFTs, amylase, and total bilirubin.
- Ultrasound: Biliary sludge and inflammation; can also be used to detect complications (e.g., gangrene, empyema, or perforation of the gallbladder).
- HIDA scan: To confirm diagnosis.

Treatment
- Urgent cholecystectomy; percutaneous cholecystostomy is an option in patients with high surgical risk.
- Cholecystectomy:
  - Surgical removal of the gallbladder.
  - May be done open or via laparoscopy.
  - Open is done by Kocher’s incision (right subcostal).
  - Laparoscopic is by 4-port technique (umbilical 10-mm, subxiphoid 10-mm and two 5-mm ports).
  - Safe dissection of the Calot’s triangle is key to demonstrate critical view of safety.
  - If the whole GB cannot be safely removed partial cholecystectomy should be done.

Cholelithiasis

Definition
Stones in the CBD. This can, but does not necessarily, cause cholecystitis.

Incidence
Found in 6–15% of acute calculous cholecystitis and 1–2% of acalculous cholecystitis at surgery.

Signs and Symptoms
Epigastric or RUQ pain and tenderness, jaundice, cholangitis, or recurrent attacks of acute pancreatitis without other known risk factors.

Diagnosis
- Labs: Increased alk phos LFTs, and total and direct bilirubin.
- ERCP: Gold standard for diagnosis of CBD stones; also provides a therapeutic option (see below).
- Endoscopic ultrasound: Less sensitive than ERCP but also less invasive; more sensitive than transabdominal ultrasound.
- Transabdominal ultrasound: Highly specific but not very sensitive for CBD stones.
- CT scan or MRI/MRCP is also very useful for evaluating CBD obstruction and biliary anatomy.

Treatment
- ERCP: Involves endoscopic sphincterotomy with retrieval of the CBD stone(s) with a basket (85–90% successful).
If ERCP fails, the CBD can be opened surgically and the stones removed. Intraoperative cholangiogram has to be done to evaluate for underlying strictures. A T-tube is placed so bile can drain externally. It is removed 2–3 weeks later on an outpatient basis.

**Cholangitis**

**Acute (ascending) Cholangitis**

**Definition**
Bacterial infection of the bile ducts usually associated with obstruction of the CBD—gall stones (most common), strictures, or mass.

**Signs and Symptoms**
- Fever, chills.
- Nausea, vomiting.
- Abdominal pain with or without altered mental status and septic shock.

**Diagnosis**
- Labs: Leukocytosis with increased bilirubin, alk phos and LFTs.
- Ultrasound: Should be the initial study; dilation of common and intrahepatic bile ducts along with gallstones, and a thickened, edematous gallbladder wall.
- CT scan with contrast or MRI/MRCP can be done—shows hyperemic wall of the CBD with ductal dilatation and cut-off point suggesting site of obstruction.
- ERCP/PTCA: Provides a definitive diagnosis; can also be therapeutic.
- Bile cultures: Obtain to facilitate proper antibiotic treatment; offending organisms are usually enteric gram negatives and enterococci.

**Treatment**
- NPO, IV fluids, and broad spectrum IV antibiotics. Close monitoring in ICU.
- If patient is in shock, decompress bile duct and remove obstruction immediately by ERCP/PTC. If unsuccessful, intraoperative decompression with T-tube placement is indicated.
- If the patient is stable, continue conservative management with definitive treatment later.

**Sclerosing Cholangitis**

**Definition**
A chronic, progressive inflammatory process of the biliary tree of unknown etiology that results in strictures and, in most cases, leads to cirrhosis and liver failure. Associated with autoimmune phenomena, particularly ulcerative colitis.

**Incidence**
Two-to-one male predominance with median age of onset at 40 years.

**Risk Factors**
IBD (ulcerative colitis), pancreatitis, diabetes, trauma to the common hepatic duct.

**REYNOLDS’ PENTAD:**
- Charcot’s triad plus
- Central nervous system (CNS) symptoms
- Septic shock

**Common causes of CBD obstruction:**
**SiNGe**
- Stricture
- iatrogenic causes (ERCP/PTC or biliary stent placement)
- Neoplasm
- Gallstones
- extrinsic compression (e.g., pancreatic pseudocyst/pancreatitis)

**WARD TIP**
Seventy percent of patients with sclerosing cholangitis have IBD, whereas 3–7.5% of patients with IBD have sclerosing cholangitis.
Complications of sclerosing cholangitis:
- Cirrhosis
- Cholangitis
- Obstructive jaundice
- Cholangiocarcinoma

**Signs and Symptoms**

Many patients are asymptomatic at the time of diagnosis, but symptoms can include fever, weight loss, fatigue, pruritus, jaundice, hepatomegaly, splenomegaly, and hyperpigmentation.

**Diagnosis**

ERCP/PTC reveal a “beads on a string” appearance of the bile ducts (see Figure 13-10), and alkaline phosphatase is almost always elevated.

**Treatment**

- Balloon dilation with stent placement can be performed for palliative purposes, but definitive treatment varies depending on the location of the strictures.
- Extrahepatic strictures: Hepatoenteric anastomosis with removal of the extrahepatic ducts and T-tube placement for external drainage of bile.
- Intrahepatic strictures: Liver transplant.

**Prognosis**

- Ten percent of patients develop cholangiocarcinoma.
- Ten-year survival is 75%.

**Gallstone ileus**

- Small bowel obstruction caused by a gallstone; the ileocecal valve is the most common site of obstruction.
Most often, a large stone has eroded a hole through the gallbladder wall to the duodenum, causing a cholecystoenteric fistula. A gallstone escapes through this hole into the GI tract and eventually gets stuck in the ileum, causing small bowel obstruction.

**incidence**
Most common in women over 70.

**Signs and Symptoms**
Symptoms of acute cholecystitis followed by signs of small bowel obstruction (nausea, vomiting, abdominal distention, RUQ pain).

**Diagnosis**
- Abdominal plain films: May show the pathognomonic features of pneumatobia, dilated small bowel, and a large gallstone in the right lower quadrant (RLQ).
- Ultrasound: Useful to confirm cholelithiasis; may also identify the fistula.
- Upper and lower GI series: Other diagnostic options that are usually unnecessary.
- CT scan of the abdomen: Shows proximally dilated small bowel with a transition point (stone in the lumen) and collapsed distal loops. The cholecystoenteric fistula can also be visualized.

**Treatment**
Exploratory laparotomy, removal of the gallstone (via proximal longitudinal enterotomy and milking the stone out, with repairing it transversely), and possible small bowel resection. Cholecystectomy and fistula repair can be undertaken if the patient is hemodynamically stable or can be performed in another setting.

**Carcinoma of the gallbladder**

**Definition**
Malignant neoplasm of the gallbladder, the majority of which are adenocarcinomas.

**incidence**
Extremely rare (<1% of patients with cholelithiasis); incidence increases with age with a peak at 75 years; female-to-male ratio 3:1.

**risk Factors**
Include porcelain gallbladder, gallstones, choledochal cysts, gallbladder polyps, and typhoid carriers with chronic inflammation.

**Signs and Symptoms**
Most patients are asymptomatic until late in the course when findings may include abdominal pain, nausea, vomiting, weight loss, RUQ mass, hepatomegaly, or jaundice. Gallbladder carcinoma tends to metastasize earlier in the course.

**Diagnosis**
Ultrasound, CT, MRI, or ERCP/PTC.

---

**OR TIP**
The whole small bowel must be palpated in Gallstone ileus to detect other gallstones in the GI tract.

**WARD TIP**
**Courvoisier's sign:** A palpable, nontender gallbladder often associated with cancer in the head of the pancreas or the gallbladder.
Treatment
- Varies depending on the extent of tumor involvement.
- Tumor confined to gallbladder mucosa: Cholecystectomy.
- Tumor involving muscularis or serosa: Radical cholecystectomy, wedge resection of overlying liver (segment IVb and V), and portal lymph node dissection.
- Tumor involving liver: Consider palliative measures such as decompression of the proximal biliary tree or a bypass procedure of the obstructed duodenum.

benign tumors of the biliary ducts

Definition
Tumors that arise from ductal glandular epithelium most commonly found at the ampulla of Vater; most are adenomas, of polyoid morphology, and < 2 cm in size.

Signs and Symptoms
Intermittent jaundice and RUQ pain.

Diagnosis
Intraoperative cholangiogram, ultrasound, ERCP/PTC.

Treatment
Resection of the tumor with a margin of duct wall either intraoperatively or endoscopically.

choledochal cysts

Abnormal cystic dilatations of intrahepatic and/or extrahepatic bile ducts. Can be congenital, acquired, or familial.
- Incidence: In Western population—1:100,000 to 1:150,000.
- Pathogenesis: Exact etiology is unknown, but an abnormal pancreatobiliary junction appears to play an important role.

presentation
- Can be asymptomatic and be incidentally diagnosed on cross-sectional abdominal imaging.
- Sometimes can be symptomatic—abdominal pain, cholangitis, jaundice. Classic presentation—abdominal pain, jaundice, and a palpable abdominal mass.

Classification
- Todani classification of biliary cysts (Figure 13-11)
- Type I—Fusiform dilatation of the extrahepatic bile duct.
- Type II—Saccular outpouching from the extrahepatic bile duct—bile duct diverticulum.
- Type III—Choledochocele—cystic dilatation of the intraduodenal portion of the bile duct.
- Type IVa—Fusiform dilatation of both extrahepatic and intrahepatic bile ducts.
- Type IVb—Fusiform dilatation of only the extrahepatic bile ducts.
- Type V—Cystic dilatations of intrahepatic ducts only—Caroli’s disease.

Diagnosis
USG, CT, MRI, ERCP.
Patients present with cholangitis—Initial treatment with NPO, IV fluids, and broad spectrum antibiotics, then a definitive surgical treatment.

These cysts carry a risk of malignancy to develop cholangiocarcinoma.

The surgical treatment is based on the type of the choledochal cyst.

Type 1—Extrahepatic bile duct resection with the cyst, cholecystectomy with Roux-en-Y hepaticojejunostomy.

Type 2—Simple cyst excision is an option, but might need bile duct resection with hepaticojejunostomy depending on degree of involvement.

Type 3—Sphincterotony or endoscopic resection.

Type 4—Depending on the degree of involvement—extrahepatic bile duct resection with hepaticojejunostomy could be an option.

Type 5—Liver transplant in ideal candidates. If not medical therapy to control symptoms.

Cholangiocarcinoma

Definition

An uncommon tumor that may occur anywhere along the intrahepatic or extrahepatic biliary tree but is most commonly located at the bifurcation of the right and left hepatic ducts (60–80% of cases). Nearly all are adenocarcinomas.
incidence
- Increases with age with peak at 55–65 years; 1/100,000 people per year.
- No sex predilection.

risk Factors
Choledochal cyst, ulcerative colitis, sclerosing cholangitis, liver flukes, toxins, contrast dye.

Signs and Symptoms
- Jaundice
- Clay-colored stools
- Dark urine
- Pruritus
- Pain
- Malaise
- Weight loss

Diagnosis
- Ultrasound: Shows bile duct dilation.
- CT: Identifies tumors located near the hilum of the liver.
- MRI/MRCP: Useful in localizing the extent and site of obstruction.
- Biopsy via ERCP/PTC under ultrasound guidance.
- Classification of hilar cholangiocarcinoma: Bismuth classification (Figure 13-12)
- Type I—involves the common hepatic duct, spares the bifurcation
- Type II—involves the common hepatic duct and the bifurcation
- Type IIIa—tumor extends to the right branch of the hepatic duct
- Type IIIb—tumor extends to the left branch of the hepatic duct
- Type IV—tumor extends to both right and left branches

WARD TIP
A cholangiocarcinoma that arises at the junction of the right and left hepatic ducts is called a Klatskin tumor.
Treatment
- Varies depending on location of the tumor.
  - Proximal tumors: Resect with a Roux-en-Y hepaticojejunostomy.
  - Distal tumors: Whipple procedure.
  - If both hepatic ducts or the main trunk of the portal vein are extensively involved, the tumor may be unresectable.

Prognosis
Five-year survival rate is 15–20%.
The Pancreas

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Embryology

- During the fourth week of gestation, the pancreas begins development from the duodenal endoderm.
- Two buds form, which rotate and fuse by eighth week.
  - Ventral pancreatic bud: Uncinate process and part of the head.
  - Dorsal pancreatic bud: Remaining part of the head, neck, body, and tail.
- **Heterotopic pancreas**: Pancreatic tissue in an abnormal location such as the stomach, duodenum, or Meckel’s diverticulum.
- **Pancreas divisum**: Due to a failure of the ventral and dorsal ducts to fuse, the majority of pancreatic drainage is accomplished via the accessory papilla and duct of Santorini. This is the most common congenital anomaly of the pancreas (5% of population) but is usually asymptomatic. Rarely, however, chronic pain and recurrent pancreatitis may result from inadequate drainage, requiring papillotomy—ERCP or surgical.
- **Annular pancreas**: Usually presents in infancy with duodenal obstruction (postprandial vomiting). Caused by malrotation of the ventral pancreas leading to a ring of pancreatic tissue around the second portion of the duodenum. Pancreatitis and peptic ulcers may also result.
- **Treatment**: Duodenojejunostomy.

Anatomy

- Location: Retroperitoneal (posterior to stomach, transverse mesocolon, and lesser omentum) at the level of the body of L2.
- Head: Includes uncinate process and abuts the second part of the duodenum.
- Neck: Portion overlying the superior mesenteric vein.
- Body: Lies to the left of the neck, forms posterior floor of lesser sac (omentum bursa).
- Tail: Enters splenorenal ligament, adjacent to splenic hilum; susceptible to injury during splenectomy.
- Ducts: The duct of Wirsung is the main duct; runs entire length of pancreas. It joins the common bile duct and empties into the second part of the duodenum at the ampulla of Vater. The duct of Santorini (small duct) is an accessory duct often joining the duodenum more proximally than the ampulla of Vater.
- Sphincter of Oddi: Smooth muscle around the ampulla of Vater.

Blood Supply and Innervation

See Figure 14-1.

- Head:
  - **Anterior and posterior superior pancreaticoduodenal arteries**—branches of the gastroduodenal artery.
  - **Anterior and posterior inferior pancreaticoduodenal arteries**—branches of the superior mesenteric artery.
- Neck, body, and tail:
  - **Splenic artery and branches (dorsal pancreatic artery).**
- Sympathetics: Pain sensation is provided by the celiac plexus (via the splanchnic nerves).
- Parasympathetics: Islets, acini, and ducts are innervated by branches of the vagus nerve.
### Physiology

#### Exocrine

Secretion of 1–2 L/day of clear, isosmotic, alkaline (pH 7.0–8.3) fluid containing digestive enzymes.

- **Acinar cells:** Secrete enzymes (e.g., chymotrypsin, trypsin, carboxypeptidase, amylase, lipase). These enzymes are secreted as inactive zymogen granules until they are activated intraluminally by enterokinase in the duodenum.
- **Centroacinar and ductal cells:** Secrete water and electrolytes (e.g., Na\(^+\), K\(^+\), HCO\(_3\)-, Cl\(^-\)) in response to secretin stimulation.
- **Phases:**
  2. Gastric phase: Antral distention and ingested protein cause release of gastrin → gastric acid secretion → duodenal acidification → secretin release → pancreatic HCO\(_3\)\(^-\) release.
  3. Intestinal phase: Duodenal acid and bile stimulate secretin. Ingested fat and protein in the duodenum stimulate release of CCK → acinar cells release pancreatic enzymes.

#### Endocrine

Islets of Langerhans make up 2% of pancreas by weight:

- **Insulin:** From beta cells in islets of Langerhans (glucose absorption and storage).
- **Glucagon:** From islet alpha cells (glycogenolysis and release of glucose).
- **Somatostatin:** From islet delta cells (generally causes inhibitory functions of gastrointestinal tract).
Acute Pancreatitis

Definition

Inflammation of the pancreas due to parenchymal autodigestion by proteolytic enzymes.

Etiology

Most common etiologies in the United States:
1. Alcohol abuse (40–50%)
2. Gallstones (40%)
3. Idiopathic (10%)

Other causes of acute pancreatitis:
- Hyperlipidemia
- Hypercalcemia: Secondary to hyperparathyroidism
- Trauma
- Postop and post-ERCP (endoscopic retrograde cholangiopancreatography)
- Pancreatic duct obstruction (e.g., tumor, pancreatic divisum)
- Vasculitis
- Scorpion venom
- Viral infection (e.g., mumps, coxsackie B, cytomegalovirus)
- Drugs (e.g., isoniazid, glucocorticoids, cimetidine)

Signs and Symptoms

- Severe, constant epigastric pain radiating to the back (because pancreas is a retroperitoneal organ and is innervated by the celiac plexus). Pain may be improved by sitting forward or standing.
- Nausea, vomiting.
- Physical exam:
  - Low-grade fever, tachypnea, tachycardia, upper abdominal tenderness with guarding but no rebound. Bowel sounds may be absent due to adynamic ileus. Signs of hypovolemic shock may also be present due to massive retroperitoneal fluid sequestration, dehydration, and systemic inflammatory response syndrome.
  - Cullen’s sign (bluish discoloration of periumbilicus), Grey-Turner’s sign (bluish discoloration of flank), and Fox’s sign (bluish discoloration of inguinal ligament) are indicative of severe, hemorrhagic pancreatitis.

Diagnosis

Laboratory Studies

- Elevated lipase: Only found in gastric and intestinal mucosa and liver, in addition to the pancreas, so is more specific for pancreatitis than amylase.
- Elevated amylase:
  - Also found in salivary glands, small bowel, ovaries, testes, and skeletal muscle, so is not a specific marker for pancreatitis.
  - Although amylase may be persistently elevated in renal insufficiency, a level three times the upper limit of normal is suggestive of pancreatitis.
- Neither amylase nor lipase is a part of Ranson’s criteria.
- Ranson’s criteria listed in Table 14-1.
### Table 14-1: Ranson’s Criteria (Predicts Risk of Mortality in Pancreatitis)

<table>
<thead>
<tr>
<th>On Admission</th>
<th>After 48 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 55</td>
<td>Base deficit &gt; 4</td>
</tr>
<tr>
<td>Blood sugar &gt; 200</td>
<td>Increase in blood urea nitrogen (BUN) &gt; 5</td>
</tr>
<tr>
<td>Serum aspartate aminotransferase (AST) &lt; 250</td>
<td>Fluid deficit &gt; 6 L</td>
</tr>
<tr>
<td>Lactic dehydrogenase (LDH) &gt; 350</td>
<td>Calcium &lt; 8</td>
</tr>
<tr>
<td>White blood count (WBC) &gt; 16,000</td>
<td>Drop in hematocrit &gt; 10%</td>
</tr>
<tr>
<td></td>
<td>PO₂ &lt; 60 mmHg</td>
</tr>
</tbody>
</table>

#### Number of Risk Factors

<table>
<thead>
<tr>
<th>Number of Risk Factors</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3</td>
<td>1%</td>
</tr>
<tr>
<td>3 or 4</td>
<td>16%</td>
</tr>
<tr>
<td>5 or 6</td>
<td>40%</td>
</tr>
<tr>
<td>&gt; 6</td>
<td>70–100%</td>
</tr>
</tbody>
</table>

### Abdominal Imaging

- Abdominal x-ray: Sentinel loop sign and colon cutoff sign.
- Ultrasound: May demonstrate pseudocysts, phlegmon, abscesses, or cholelithiasis.
- Computed tomography (CT) scan: Diagnostic test of choice (90% sensitive and 100% specific).
- Demonstrates
  - the degree of pancreatitis—edematous gland to severe stranding around the pancreas and fluid collections. Helpful in grading of pancreatitis—Balthazar grading system.
  - complications, phlegmon, abscesses, pancreatic necrosis, or pseudocyst formation (see Figure 14-2).

#### Differential Diagnosis

- Pathology of Structures in the Epigastrum, RulQ LowEr mEdiaStInum, and chEst
  - Acute cholecystitis
  - Gastritis/peptic ulcer disease
  - Perforated esophagus/peptic ulcer
  - Myocardial infarction
  - Ruptured abdominal aortic aneurysm
  - Acute mesenteric ischemia
  - Pneumonia

#### Treatment

- Aggressive hydration with electrolyte monitoring to maintain adequate intravascular volume, preferably in intensive care unit (ICU) setting.

### Ward Tip

- **Cullen’s sign:** Think of cULen and Umbilicus.
- **Grey-Turner’s sign:** Think of Turn ing on your side/fank.

### Ward Tip

High amylase levels are also seen in intestinal disease, perforated ulcer, ruptured ectopic pregnancy, salpingitis, salivary gland disorders, renal failure, and diabetic ketoacidosis.
- Nasogastric tube: For severe disease with vomiting.
- Antibiotics: If infection identified especially in necrotizing pancreatitis (CT guided aspiration).
- NPO with nutritional support via post pyloric feeding (enteral feeding is proven to beneficial compared to TPN) or total parenteral nutrition (TPN).
- Avoid use of morphine due to possible spasm of sphincter of Oddi. Use meperidine (Demerol) instead for pain management.
- Surgery indicated for:
  - Complications of pancreatitis—infected necrosis, hemorrhage, pseudocysts.
  - Correction of associated biliary tract disease: Gallstone pancreatitis should be treated with early interval cholecystectomy only after acute pancreatic inflammation has resolved to prevent further attacks of pancreatitis which can be of higher severity. Acutely, ERCP with endoscopic sphincterotomy may be used to relieve biliary obstruction.

**prognosis**

See Table 14-1 for Ranson’s criteria.

**Chronic Pancreatitis**

**Definition**

Chronic inflammation or recurrent acute pancreatitis causes irreversible parenchymal fibrosis, destruction, and calcification, leading to loss of endocrine and exocrine function.
**Etiology**

- Most commonly alcohol abuse (70%).
- Idiopathic (20%).
- Other (10%): Hyperparathyroidism, hypertriglyceridemia, congenital pancreatic anomalies, hereditary, obstruction.

**Signs and Symptoms**

- Recurrent or constant epigastric and/or back pain, bloating, abdominal cramps.
- Malabsorption/malnutrition (exocrine dysfunction).
- Steatorrhea (exocrine dysfunction)—fat-soluble vitamin deficiency.
- Type 1 diabetes mellitus (endocrine dysfunction).
- Polyuria.

**Diagnosis**

- History.
- Fecal fat analysis—both quantitative and qualitative (Sudan stain) stool for elastase levels.
- X-ray (kidneys, ureters, bladder): Pancreatic calcifications.
- ERCP or MRCP: Chain-of-lakes pattern—ductal irregularities with dilation and stenosis.
- CT: Pseudocysts (see Figure 14-3) (use ultrasound for follow-up of pseudocysts). Gland enlargement/atrophy, calcifications, masses also seen on CT.

**Differential Diagnosis**

- Peptic ulcer disease.
- Pancreatic cancer.
- Biliary tract disease (cholecystitis).

---

**Figure 14-3.** Abdominal CT demonstrating calcification involving the entire head of the pancreas, consistent with chronic pancreatitis.
indicationS for Surgery

- Persistent pain
- Gastrointestinal or biliary obstruction
- Pseudocyst infection, hemorrhage, or rupture
- Enlarging pseudocysts

Treatment

- **Nonoperative management**: Includes control of abdominal pain, endocrine and exocrine insufficiency (insulin and pancreatic enzyme therapy).
- **Operative management** (can do preop MRCP or ERCP to evaluate anatomy):
  - Pain relief: Celiac plexus block.
  - Ampullary procedures: ERCP with endoscopic sphincterotomy.
  - Ductal decompression/drainage procedures:
    - Puestow procedure (longitudinal pancreaticojejunostomy) for segmental ductal dilation.
    - Duval procedure (retrograde drainage with distal resection and end-to-end pancreaticojejunostomy).
  - Resective procedures (resection of portions of pancreas):
    - Frey procedure (longitudinal pancreaticojejunostomy with partial resection of the pancreatic head).
    - Whipple procedure (pancreaticoduodenectomy with hepaticojejunostomy, pancreaticojejunostomy, and gastrojejunostomy).
    - Beger procedure—coring out the pancreatic parenchyma in the head of the pancreas (if major involvement is the pancreatic head only)
  - Near-total or total pancreatectomy
  - Pseudocysts: Nonepithelialized, encapsulated pancreatic fluid collections. Up to 30% of peripancreatic fluid collections resolve on their own with bowel rest (TPN and NPO). If after 6 weeks they have not resolved they will tend to have a mature wall and are termed pseudocysts. Pseudocysts larger than 6 cm in size rarely resolve spontaneously and internal drainage of the mature cyst is indicated via cyst gastrostomy or Roux-en-Y cyst jejunostomy.

Other Complications of Pancreatitis

- Pancreatic abscess
- Pancreatic fistulae
- Hemorrhagic pancreatitis
- Pancreatic cancer

Pancreatic Adenocarcinoma

**Etiology**

- Originates in the exocrine pancreas (ductal cells).
- Two thirds occur in the head of the pancreas.

**Risk factors**

- Male gender (2:1 male-to-female).
- African-American heritage (2:1 Black-to-White).
- Tobacco use (2× increased risk).
- Diabetes, alcohol abuse, chronic pancreatitis, and increased age are also associated with increased risk.

**Signs and Symptoms**

- Constitutional symptoms—weight loss and loss of appetite.
- Painless jaundice (due to ampullary obstruction).
- New onset diabetes and steatorrhea (endocrine and exocrine insufficiency).
- Epigastric pain radiating to the back—usually due to underlying chronic pancreatitis.
- Migratory thrombophlebitis: Trousseau’s syndrome is seen especially in tumors of the body or tail.

**Diagnosis**

- Elevated carcinoembryonic antigen (CEA) or CA 19-9 (tumor markers).
- Abdominal CT scan with IV contrast is study of choice. Classic double duct sign—dilated CBD and pancreatic duct, with a hypodense mass in the head of the pancreas.
- MRI/MRCP is also useful.
- Percutaneous transhepatic cholangiography (PTC) and ERCP (useful in peripancreatic lesions.
- Angiography may also be useful.

**Treatment**

- Tumors of the head: The only chance for a cure is the Whipple procedure (pancreaticoduodenectomy), and most tumors are not resectable at the time of diagnosis.
- Tumors of the body/tail: Distal “near-total” pancreatectomy.
- If unresectable (due to liver/peritoneal metastases, nodal metastases beyond the zone of resection, or tumor invasion of the superior mesenteric artery), palliative procedure considered:
  - Relieve biliary obstruction—PTC, ERCP with biliary stenting, cholecystojejunostomy, or choledochojjunostomy
  - Relieve duodenal obstruction—gastrojejunostomy—bypass procedure.
- Double bypass—gastric and biliary bypass procedure.
- Splanchnic nerve block (pain control) or neurolysis with absolute alcohol.
- Postoperative chemoradiation therapy typically includes 5-fluorouracil (5-FU) and external-beam radiation.
- If tumor is borderline resectable invading the adjacent superior mesenteric vein neoadjuvant chemotherapy is an option to make them resectable.

**Prognosis**

- The mean survival for patients with unresectable disease is 4–6 months, with a 5-year survival rate of < 3%.
- The median survival for patients who undergo successful resection is approximately 12–19 months, with a 5-year survival rate of 15–20%.

---

WARD TIP

**Courvoisier’s sign**: Jaundice with a palpable nontender gallbladder.

OR TIP

**Whipple procedure**: Removal of gallbladder, common bile duct (CBD), antrum of stomach, duodenum, proximal jejunum, and head of pancreas (en bloc); Reconstruction with pancreaticojejunoanostomy, hepaticojejunostomy, and gastrojejunostomy.
Cystadenocarcinoma

- Commonly seen in females age 40–60 years.
- In body and tail.
- Less than 2% of all pancreatic exocrine tumors.
- Prognosis better than adenocarcinoma.
- **Treatment**: Surgical resection—distal/total pancreatectomy.

Cystadenoma

- Older and middle-aged women
- Two types:
  - Serous: Benign
  - Mucinous: Generally benign but has potential to be malignant
- **Treatment**: Surgical resection

Endocrine Tumors (Islet Cell Tumors)

**inSulinoma**

**Definition**

Beta cell neoplasm with overproduction of insulin.

**epidemiology**

- Most common islet cell tumor.
- Ninety percent are benign.
- Most are solitary lesions with even distribution in the head, body, and tail of the pancreas.
- If associated with multiple endocrine neoplasia (MEN) I syndrome (< 10% of cases), then multiple insulinomas may be present.

**signs and symptoms**

Most symptoms related to hypoglycemia triggered catecholamine release.

**Differential Diagnosis**

Surreptitious insulin administration.

**Diagnosis**

- Fasting serum insulin level > 10 µU/mL (normal: < 6 µU/mL).
- Fasting insulin-to-glucose ratio > 0.3.
- Proinsulin or C-peptide levels should be measured to rule out surreptitious exogenous insulin administration. Abdominal CT scan or MRI—hypervascular nodules.
- Selective mesenteric angiography can be considered to localize.

**Treatment**

- Surgical enucleation or resection is usually curative (90% of patients).
- Diazoxide can improve hypoglycemic symptoms by inhibiting pancreatic insulin release.
**Gastrinoma**

**Definition**
- Neoplasm associated with overproduction of gastrin.
- Also known as Zollinger–Ellison syndrome.

**Epidemiology**
- Second most frequent islet cell tumor.
- Ninety percent are located in the “gastrinoma triangle” (Figure 14-4) bordered by:
  - Junction of second and third part of the duodenum.
  - The cystic duct.
  - The superior mesenteric artery under the neck of the pancreas.
- Small, slow-growing, multiple, 60% malignant.

**Signs and Symptoms**
- Signs of peptic ulcer disease (especially in patients with recurrent or unusually located ulcers).
- Epigastric pain most prominent after eating.
- Profuse watery diarrhea.

**Diagnosis**
- Fasting serum gastrin level > 500 pg/mL (normal: < 100 pg/mL).
- Secretin stimulation test will cause a paradoxical increase in gastrin in patients with Zollinger–Ellison syndrome (double fasting level or increase of 200 pg/dL over the fasting level).
- Ulcers in unusual locations (e.g., third part of duodenum or jejunum) is highly suggestive.
- Octreotide scan to localize tumor.

---

**Figure 14-4. Gastrinoma triangle—the anatomic triangle in which approximately 90% of gastrinomas are found.**

**Exam Tip**
- Twenty-five percent of gastrinomas are associated with MEN-I.

**Exam Tip**
- Typical scenario: A 40-year-old male complains of chronic epigastric pain shortly following meals and notices needing increasing doses of his anti-ulcer medication. Think Gastrinoma.
Treatment
- Proton pump inhibitor to alleviate symptoms.
- Surgical resection (curative or debulking).
- Chemotherapy.

Vipoma

Definition
Overproduction of vasoactive intestinal peptide (VIP).

epidemiology
- Also known as Verner–Morrison syndrome or WDHA syndrome (Watery Diarrhea, Hypokalemia, Achlorhydria).
- Most are malignant; majority have metastasized to lymph nodes and the liver at time of diagnosis.
- Ten percent are extrapancreatic.

signs and symptoms
- Severe watery diarrhea.
- Signs of hypokalemia—palpitations/arrhythmias, muscle fasciculations/tetany, paresthesias.

Diagnosis
Fasting serum VIP level > 800 pg/mL (normal: < 200 pg/mL) with exclusion of other causes of diarrhea.

Treatment
- Surgical resection, chemotherapy.
- Octreotide (somatostatin analogue)—diarrhea control.

Glucagonoma

Definition
Rare alpha cell neoplasm resulting in overproduction of glucagon.

epidemiology
Most are malignant, large primary tumors that have usually metastasized to lymph nodes and liver at the time of diagnosis.

signs and symptoms
- Mild diabetes (hyperglycemia).
- Anemia.
- Mucositis.
- Weight loss due to low amino acid levels.
- Necrolytic migratory erythema—a red psoriatic-like rash with serpiginous borders over trunk and lower limbs.

Diagnosis
- Fasting serum glucagon level > 1,000 pg/mL (normal: < 200 pg/mL).
- Skin biopsy to confirm presence of necrolytic migratory erythema.
Treatment
- Surgery and chemotherapy.
- Octreotide to inhibit the release of glucagon.

Somatostatinaoma
- Very rare tumor.
- Malignant.
- Treated by surgery and chemotherapy.

Pancreatic Trauma
See Trauma chapter.

Pancreas Transplantation
See Transplant chapter.
## Hormones

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Hormones

**Definition**

A chemical substance secreted into the bloodstream by cells in one part of the body that acts on distant organs or tissues.

**Classes**

- **Steroid:**
  - Adrenal cortical: Cortisol, aldosterone.
  - Ovarian, testicular, and placental hormones (not discussed here).
- **Tyrosine-derived:**
  - Thyroid: Triiodothyronine (T<sub>3</sub>), thyroxine (T<sub>4</sub>.
  - Adrenal medulla: Epinephrine, norepinephrine.
- **Protein/peptide:**
  - Pituitary:
    - Anterior: Growth hormone (GH), adrenocorticotropic hormone (ACTH), thyroxine-stimulating hormone (TSH), follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin (PRL).
    - Posterior: Antidiuretic hormone (ADH), oxytocin.
  - Parathyroid: Parathyroid hormone.
  - Pancreas: Insulin, glucagon, somatostatin.

**Mechanisms of Action**

See Table 15-1.

**WARD TIP**

All protein hormones are produced in locations starting with **P:**
- Pituitary
- Parathyroid
- Pancreas

---

**Table 15-1. Summary of Endocrine Hormones and Their Functions**

<table>
<thead>
<tr>
<th>Endocrine Organ</th>
<th>Hormone (GH)</th>
<th>Functions</th>
<th>Stimulat Ed By</th>
<th>Inhibit Ed By</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior pituitary</td>
<td>Growth hormone (GH)</td>
<td>Opposes insulin, Stimulates amino acid uptake, Stimulates release of fatty acid from storage sites, Mediates immunoglobulin F (IGF) synthesis, Stimulates growth of nearly all tissues</td>
<td>Growth hormone–releasing hormone (GHRH), Hypoglycemia, Arginine, Exercise, l-dopa, Clonidine, Propranolol</td>
<td>Somatostatin</td>
</tr>
<tr>
<td>Adrenocorticotropic hormone (ACTH)</td>
<td>Stimulates secretion of adrenocortical hormones</td>
<td></td>
<td>Corticotropin-releasing hormone (CRH), Stress</td>
<td>Cortisol (negative feedback)</td>
</tr>
<tr>
<td>Thyroid-stimulating hormone (TSH)</td>
<td>Regulates secretion of triiodothyronine (T&lt;sub&gt;3&lt;/sub&gt;) and thyroxine (T&lt;sub&gt;4&lt;/sub&gt;)</td>
<td>Thyrotropin-releasing hormone (TRH)</td>
<td>Negative feedback</td>
<td></td>
</tr>
<tr>
<td>Follicle-stimulating hormone (FSH)</td>
<td>Stimulates ovarian follicular growth (female), Stimulates spermatogenesis and testicular growth (male)</td>
<td>Gonadotropin-releasing hormone (GnRH)</td>
<td>Negative feedback</td>
<td></td>
</tr>
</tbody>
</table>

(continues)
<table>
<thead>
<tr>
<th>Endocrine Organ (continued)</th>
<th>Hormone</th>
<th>Functions</th>
<th>Stimulated By</th>
<th>Inhibited By</th>
</tr>
</thead>
</table>
| **Anterior pituitary**      | Luteinizing hormone (LH) | - Ovulation (female)  
- Latectization of follicle (female)  
- Stimulates production of estrogen and progesterone (female)  
- Promotes production of testosterone (male)  
- Facilitates breast development in preparation for milk production | - GnRH | Negative feedback |
| **Prolactin (PRL)** |       |       | TRH  
- Estrogen  
- Stress  
- Exercise | Bromocriptine |
| **Posterior pituitary**     | Antidiuretic hormone (ADH, vasopressin) | - Promotes water absorption in collecting ducts of kidney  
- Vasodilates peripheral arterioles, increasing blood pressure  
- Increases frequency and strength of uterine contractions  
- Stimulates breast milk ejection | Increased plasma osmolality  
Decreased plasma volume  
Suckling | Vaginal stimulation |
| Oxytocin |       |       | | |
| **Thyroid**                 | T<sub>3</sub>, T<sub>4</sub> | - Increase basal metabolic rate (BMR), oxygen consumption  
- Increase protein synthesis, lipolysis, glycogenolysis, gluconeogenesis  
- Increase heart rate and contractility  
- Increase catecholamine sensitivity  
- Stimulate release of steroid hormones  
- Stimulate erythropoiesis and 2,3-diphosphoglycerate (DPG) production  
- Increase bone turnover  
- Increases serum calcium (by inhibiting osteoclasts)  
- Increases phosphate excretion | TSH | Negative feedback |
| Calcitonin |       |       | High serum calcium | Low serum calcium |
| **Parathyroid**             | Parathyroid hormone (PTH) | - Kidney: Increases calcium resorption in proximal convoluted tubule  
- Rapid  
- Also increased excretion of sodium, potassium, phosphate, and bicarbonate  
- Bone: Increases calcium mobilization  
- Rapid phase: Equilibration with extracellular fluid (ECF)  
- Slow phase: Enzyme activation (promoting bone resorption)  
- GI tract (indirect): Increases absorption via vitamin D | Low serum calcium | High serum calcium |

(continues)
<table>
<thead>
<tr>
<th>Endocrine Organ</th>
<th>Hormone</th>
<th>Functions</th>
<th>Stimulated By</th>
<th>Inhibited By</th>
</tr>
</thead>
</table>
| Pancreas        | Insulin (B cell) | Effects on liver:  
|                 |         | • Glycogenesis, glycolysis, synthesis of protein, triglycerides, cholesterol, very low-density lipoprotein (VLDL)  
|                 |         | • Inhibits glycogenolysis, ketogenesis, gluconeogenesis  
|                 |         | Effects on muscle:  
|                 |         | • Protein synthesis, glycogen synthesis  
|                 |         | Effects on fat:  
|                 |         | • Promotes triglyceride storage | Hyperglycemia | Hypoglycemia |
|                 |         | Metabolic effects:  
|                 |         | • Glycogenolysis, gluconeogenesis, ketogenesis (liver); lipolysis (adipose tissue); insulin secretion  
|                 |         | Effects on gastrointestinal (GI) secretion:  
|                 |         | • Inhibition of gastric acid and pancreatic exocrine secretion  
|                 |         | Effects on GI motility:  
|                 |         | • Inhibition of peristalsis  
|                 |         | Cardiovascular effects:  
|                 |         | • Increase in HR and force of contraction  
| Glucagon (A cell) |         | | Hypoglycemia | Hyperglycemia |
|                 |         | Inhibition of gastric acid, pepsin, pancreatic exocrine secretion  
|                 |         | Inhibition of ion secretion  
|                 |         | Inhibition of motility  
|                 |         | Reduction of splanchnic blood flow  
|                 |         | Inhibition of insulin, glucagons, pancreatic polypeptide secretion  
|                 |         | Function not known, but level rises after a meal (possible inhibition of pancreatic exocrine secretion) |
| Somatostatin (D cell) |         | | | |
| Pancreatic polypeptide (F cell) |         | | | |
| Adrenal cortex | Cortisol (zona fasciculata and reticularis) | • Stimulation of hepatic gluconeogenesis, inhibition of protein synthesis, increased protein catabolism, lipolysis, inhibition of peripheral glucose uptake  
|                 |         | • Inhibition of fibroblast activity, inhibition of bone formation, reduction of GI calcium absorption  
|                 |         | • Inhibition of leukocytes, decreased migration of inflammatory cells to site of injury, decreased production of mediators of inflammation | ACTH circadian rhythm | Stress | Negative feedback |
**TABLE 15-1. Summary of Endocrine Hormones and Their Functions (continued)**

<table>
<thead>
<tr>
<th>Endocrine Organ</th>
<th>Hormone</th>
<th>Functions</th>
<th>Stimulated By</th>
<th>Inhibited By</th>
</tr>
</thead>
<tbody>
<tr>
<td>Androgens (zona fasciculata and reticularis)</td>
<td>Dehydroepiandrosterone (DHEA), DHEA sulfate are converted to testosterone and dihydrotestosterone in the periphery</td>
<td>Adrenal androgens make up &lt;5% of total testosterone production in the normal male</td>
<td>Stress and inhibitors</td>
<td></td>
</tr>
<tr>
<td>Aldosterone (zona glomerulosa)</td>
<td>Stimulates renal tubular sodium absorption in exchange for potassium and hydrogen</td>
<td>Net effect: Fluid reabsorption and intravascular volume expansion</td>
<td>Stress and inhibitors</td>
<td></td>
</tr>
<tr>
<td>Adrenal medulla</td>
<td>Epinephrine and norepinephrine</td>
<td>Increased oxygen consumption, increased heat production, stimulation of glycogenolysis, lipolysis, inhibition of insulin secretion</td>
<td>Stress receptors and inhibitory α1 and β1 receptors</td>
<td>Noncardiac smooth muscle relaxation (vessels, uterus, bronchi)</td>
</tr>
</tbody>
</table>

**Multiple Endocrine Neoplasia (MEN)**

**General**
Autosomal dominant genetic disorder.

**Men I (Wermer’s synDr oMe)**
- Parathyroid hyperplasia (90%).
- Pancreatic (and duodenal) islet cell tumors (50%).
- Pituitary adenomas (25%) (prolactinoma is most common).

**Men IIa (sipple’s synDr oMe)**
- RET oncogene mutation on chromosome 10q11.2; missense mutations on chromosome 1.
- Medullary thyroid carcinoma (100%)—20% of all medullary cancers are due to MEN.
- Pheochromocytoma (33%)—majority are bilateral.
- Parathyroid hyperplasia (50%).

**EXAM TIP**
MEN I tumors are the 3 Ps:
- Pituitary
- Parathyroid
- Pancreas

**EXAM TIP**
MEN IIa, 2Ps and M
- Pheochromocytoma
- Parathyroid
- Medullary thyroid carcinoma
MEN IIb, 1P and 2Ms
- Pheochromocytoma
- Medullary thyroid carcinoma
- Marfanoid habitus
Mucosal neuroma may be earliest sign present (100%)—hypertrophied lips, thickened eyelids.
- Medullary thyroid carcinoma (85%).
- Pheochromocytoma (50%).
- Marfanoid habitus—skeletal abnormalities of spine (e.g., kyphosis), pectus excavatum.

Treatment
- Perform subtotal or total parathyroidectomy with autotransplantation for parathyroid hyperplasia (MEN I and MEN IIA).
- Perform total thyroidectomy with bilateral prophylactic central lymph node dissection for medullary thyroid cancer (MEN II).

The thyroid gland is responsible for the metabolic activity of the body. Dysfunction of the thyroid can result in hyper or hypo states of hormone production. Several different types of cancer can also form in the thyroid gland. These conditions may ultimately require surgical correction.

The thyroid develops at the base of the tongue between the first pair of pharyngeal pouches, in an area called the foramen cecum. The thyroid gland then descends down the midline to its final location overlying the thyroid cartilage, and develops into a bilobed organ with an isthmus between the two lobes. It remains connected to the floor of the pharynx via the thyroglossal duct, which subsequently obliterates around the second month of gestation. However, the thyroglossal duct may fail to obliterate and form a thyroglossal cyst or fistula instead. These are most commonly seen in children and should be surgically excised. A pyramidal lobe can be seen in 50–80% of the population and represents a remnant of the distal thyroglossal tract. The pyramidal lobe extends superiorly from the median isthmus.

Lymphatics ultimately drain to internal jugular nodes. Intraglandular lymphatics connect both lobes, explaining the relatively high frequency of multifocal tumors in the thyroid.
Figure 15-1. **Thyroid anatomy.** (Reproduced, with permission, from Morton DA, Foreman KB, Albertine KH. The Big Picture: Gross Anatomy. New York, NY: McGraw-Hill Education; 2011. Figure 26-3AB)

**vasculature**

See Figure 15-2.
Arterial
- Superior thyroid arteries (on each side).
  - First branch of external carotid artery at the level of the carotid bifurcation.
- Inferior thyroid artery (on each side).
  - From thyrocervical trunk of subclavian artery.
  - Ima (sometimes present).
  - From aortic arch or innominate artery.

Venous
- Superior thyroid vein (on each side).
  - Drains to internal jugular (IJ).
- Middle thyroid vein (on each side).
  - Drains to IJ.
- Inferior thyroid vein (on each side).
  - Drains to brachiocephalic vein.

innervation
- The right recurrent laryngeal nerve (RLN) branches from the right vagus nerve, loops under the right subclavian artery, and ascends to the larynx (posterior to the thyroid) between the trachea and esophagus. It may be anterior or posterior to the inferior thyroid artery. The left RLN branches from the left vagus nerve, loops under the aortic arch, and then ascends along the tracheoesophageal groove to the larynx. Both RLNs innervate the muscles of the true vocal cords.
- Sympathetic: Superior and middle cervical sympathetic ganglia (vasomotor).
- Parasympathetic: From vagus nerves, via branches of laryngeal nerves.

hormones
The thyroid gland produces thyroid hormone (TH) using iodide and tyrosine.

hormone regulation
- TSH causes:
  - Increased formation of TH.
  - The release of TH into circulation within 30 minutes.
The increased TH level in blood then feeds back to the pituitary and results in decreased TSH secretion, by an incompletely understood mechanism.

**Effects of Thyroid Hormone**

- Cardiovascular system: Increased heart rate (HR), cardiac output (CO), blood flow, blood volume, pulse pressure (no change in mean arterial pressure [MAP]).
- Respiratory system: Increased respiratory rate (RR), depth of respiration.
- Gastrointestinal (GI) system: Increased motility.
- Central nervous system (CNS): Nervousness, anxiety.
- Musculoskeletal system: Increased reactivity up to a point, then response is weakened; fine motor tremor.
- Sleep: Constant fatigue but decreased ability to sleep.
- Nutrition: Increased basal metabolic rate (BMR), need for vitamins, metabolism of carbohydrate, lipid, and protein; decreased weight.

**Assessment of Function**

- If \( T_4 \) production is increased, both total \( T_4 \) (t\( T_4 \)) and free \( T_4 \) (f\( T_4 \)) increase.
- If production decreases, both t\( T_4 \) and f\( T_4 \) decrease.
- If amount of thyroid-binding globulin (TBG) changes, only t\( T_4 \) changes, not f\( T_4 \).

**Congenital Anomalies**

- Persistent sinus tract remnant of developing gland: Thyroglossal duct cyst—may occur anywhere along course as a midline structure with thyroid epithelium, usually between the isthmus and the hyoid bone:
  - Most common congenital anomaly.
  - Few symptoms but may become infected.
  - Easier to see when tongue is sticking out.
  - Surgical treatment: Excision of duct remnant and central portion of hyoid bone (Sistrunk’s operation).
- Complete failure to develop.
- Incomplete descent: Lingual or subhyoid position (if gland enlarges, patient will have earlier respiratory symptoms).
- Excessive descent: Subternal thyroid.
- Malformation of branchial pouch.

**Hyperthyroidism**

**Causes**

- Graves’ disease.
- Toxic nodular goiter.
- Toxic thyroid adenoma.
- Subacute thyroiditis.
- Functional metastatic thyroid cancer.
- Struma ovarii (abnormal thyroid tissue in ovary).

**Graves’ disease**

- Most common cause of hyperthyroidism in the United States.
- **Mechanism:** Autoimmune disorder that causes an excess of TH to be produced due to the presence of thyroid-stimulating immunoglobulins that stimulates production of TSH.

**WARD TIP**

Thyroid follicles store enough hormone to last 2–3 months. Thus, there is no need to worry about your postop hypothyroid patients who are NPO for several days. They can resume taking their levothyroxine when they begin a PO diet.

**WARD TIP**

Palpation of the thyroid is easiest if you stand behind the patient and reach your arms around to the front of the neck. Expect the isthmus to be about one fingerbreadth below the cricoid cartilage. In addition, make sure to palpate for cervical lymph nodes.

**WARD TIP**

In 70% of cases of lingual thyroid, it is the patient’s only functioning thyroid tissue. This means that it is important to look for other functioning thyroid tissue prior to removing a lingual thyroid.

**EXAM TIP**

Ten percent of patients will have atrial fibrillation that may be refractory to medical treatment until hyperthyroidism is controlled.
- Affects nearly 2% of American women; six times more common in women.
- Onset age 20–40.
- Families with Graves’ disease have increased risk of other autoimmune disorders (e.g., diabetes, Addison’s) and other thyroid disorders as well.

**Signs and Symptoms**

- Nervousness, increased sweating, tachycardia, goiter, pretibial myxedema, tremor (90%).
- Heat intolerance, palpitations, fatigue, weight loss, dyspnea, weakness, increased appetite, exophthalmos, thyroid bruit (50–90%).
- Other: Amenorrhea, decreased libido and fertility.

**Diagnosis**

- Labs: Thyroid function tests (TFTs)—increased $T_3$ and/or $T_4$ and decreased TSH (negative feedback of ↑ hormone levels).
- Radioactive iodide uptake test (RAIU): Scan shows diffusely increased uptake.

**Treatment**

- Antithyroid drugs.
- Radioiodide ablation with $^{131}$I.
- Subtotal or total thyroidectomy.

**Choosing a treatment:**

- Consider: Age, severity, size of gland, surgical risk, treatment side effects, comorbidities.
- Radioablation is the most common choice in the United States:
  - Indicated for small or medium-sized goiters, if medical therapy has failed, or if other options are contraindicated.
  - Most patients become euthyroid within 2 months.
  - Most patients ultimately require TH replacement (e.g., levothyroxine).
  - Complications include exacerbation of thyroid storm initially.
  - Contraindicated in pregnant patients, women of childbearing age and newborns.

- Surgical treatment:
  - Indicated when radioablation is contraindicated (e.g., young or pregnant patients) or if medical management cannot be used.
  - Patients should be euthyroid prior to excision.
  - Advantage over radioablation is immediate cure.

- Medical therapy:
  - β-blockers provide symptomatic relief.
  - Antithyroid drugs (propylthiouracil [PTU], methimazole) inhibit hormone production and peripheral conversion of $T_4 \rightarrow T_3$.
  - Potassium iodide reduces hormone production (via Wolff–Chaikoff effect), used to shrink gland prior to surgical excision.
  - High recurrence rate with medical therapy.
  - May cause side effects such as rash, fever, or peripheral neuritis.
  - Patients relapse if meds are discontinued.
  - Check TFTs after any treatment to determine if patient is successfully euthyroid or requires hormone replacement.

**Toxic nodular Goiter (Plummer’s disease)**

- Causes hyperthyroidism but without the extrathyroidal symptoms.
- Treatment is surgical since medical therapy and radioablation has a high failure rate.
Solitary nodule: Lobectomy.
Multinodular goiter: Subtotal thyroidectomy.

**Thyroid Storm (Thyrotoxicosis)**
- Life-threatening extreme exacerbation of hyperthyroidism precipitated by surgery on an inadequately prepared patient (i.e., incomplete β-blockade and noneuthyroid patients), infection, labor, iodide administration, or recent radioablation.
- Patient presents with fever, tachycardia, muscle stiffness or tremor, disorientation/altered mental status.
- Fifty percent of patients with thyroid storm develop congestive heart failure (CHF).
- Has a 20–40% mortality rate.
- Best way to treat this is by avoiding it. Prophylaxis includes achieving euthyroid state preop.
- **Treatment:** Fluids, antithyroid medication (thionamide, PTU, methimazole), β-blockers (propanolol, metoprolol), corticosteroids (hydrocortisone), sodium iodide (NaI) or Lugol’s solution (KI), and a cooling blanket.

**Hypothyroidism**

**Causes**
- Autoimmune thyroiditis.
- Iatrogenic: s/p thyroidectomy, s/p radioablation, secondary to antithyroid medications.
- Iodine deficiency.

**Signs and Symptoms**
Differ, depending on age of diagnosis:
- Infants/peds: Characteristic Down’s-like facies, failure to thrive, mental retardation.
- Adolescents/adults (particularly when due to autoimmune thyroiditis):
  - Eighty percent female.
  - Physiologic effects: Bradycardia, decreased CO, hypotension, shortness of breath secondary to effusions.
  - Presentation includes: Fatigue, weight gain, cold intolerance, constipation, menorrhagia, decreased libido and fertility.
  - Less common complaints: Yellow-tinged skin, hair loss, tongue enlargement.

**Diagnosis**
- History and physical exam findings.
- Labs:
  - Decreased T<sub>4</sub>, T<sub>3</sub>.
  - TSH:
    - Increased in primary hypothyroidism.
    - Decreased in secondary hypothyroidism.
  - Confirm with TRH challenge: TSH will not respond in secondary hypothyroidism.
  - Thyroid autoantibodies present in autoimmune thyroiditis.
  - Low hematocrit (Hct).
- Electrocardiogram (ECG): Decreased voltage and flat or inverted T waves.

**Treatment**
Thyroxine PO or IV emergently if patient presents in myxedema coma.
Acute
- **Infectious etiology:** Streptococcus pyogenes, Staphylococcus aureus, Pneumococcus pneumoniae.
- **Risk factors:** Female sex, goiter, thyroglossal duct.
- **Signs and symptoms:** Unilateral neck pain and fever, euthyroid state, dysphagia.
- **Treatment:** IV antibiotics and surgical drainage.

Subacute (de Quervain’s)
- **Etiology:** Post–viral upper respiratory infection (URI).
- **Risk factors:** Female sex.
- **Signs and symptoms:** Fatigue, depression, neck pain, fever, unilateral swelling of thyroid with overlying erythema, firm and tender thyroid, transient hyperthyroidism usually preceding hypothyroid phase.
- **Diagnosis:** Made by history and exam.
- **Treatment:**
  - Usually self-limited disease (within 6 weeks).
  - Manage pain with nonsteroidal anti-inflammatory drugs (NSAIDs).

Chronic (Hashimoto’s Thyroiditis)
- **Etiology:** Autoimmune.
- **Risk factors:** Down’s syndrome, Turner syndrome, familial Alzheimer’s disease, history of radiation therapy as child.
- **Signs and symptoms:** Painless enlargement of thyroid, neck tightness, presence of other autoimmune diseases.
- **Diagnosis:** Made by history, physical, and labs.
  - Labs: Circulating antibodies against microsomal thyroid cell, TH, T₃, T₄, or TSH receptor.
- **Pathology:** Firm, symmetrical, enlargement; follicular and Hürthle cell hyperplasia; lymphocytic and plasma cell infiltrates.
- **Treatment:**
  - Thyroid hormone (usually results in regression of goiter).
  - With failure of medical therapy, partial thyroidectomy is indicated.

Riedel’s Fibrosing Thyroiditis
- **Rare.**
- **Fibrosis replaces both lobes and isthmus.**
- **Risk factors:** Associated with other fibrosing conditions, like retroperitoneal fibrosis, sclerosing cholangitis.
- **Signs and symptoms:** Usually remain euthyroid; neck pain, possible airway compromise; firm, nontender, enlarged thyroid.
- **Diagnosis:** Open biopsy required to rule out carcinoma or lymphoma.
- **Pathology:** Dense, invasive fibrosis of both lobes and isthmus. May also involve adjacent structures.
- **Treatment:**
  - With airway compromise: Isthmectomy.
  - Without airway compromise: Medical treatment with steroids.

Workup of a Mass
- Fifteen percent of solitary thyroid nodules are malignant.
- If multinodular thyroid gland, risk of malignancy is only 5%.
- Ninety to 95% present as well-differentiated cancer.
- Lateral aberrant thyroid: Usually well-differentiated papillary cancer metastatic to cervical lymph nodes.
- **Risk factors:** History of radiation, family history of thyroid cancer, age, gender.
**Signs and symptoms:**
- Voice and/or airway symptoms, sudden enlargement of nodule.
- History of head and neck radiation therapy leads to 40% risk of developing thyroid cancer, so if patient presents with this history, proceed directly to surgery.
- Exam: Check size, mobility, quality, adherence of mass, and presence of lymphadenopathy. Concerning findings include hard, fixed gland or palpable cervical lymph nodes.

**Diagnosis**
Fine-needle aspiration (FNA) is the standard of care for thyroid nodule workup (see Figure 15-3).

- **Benign (65%):**
  - Ultrasound (US) for sizing and to differentiate nodules and cysts.
  - Obtain thyroglobulin level and follow over time. No need for surgery.
  - Suspicious or nondiagnostic (15%): Usually follicular (20% will be malignant).
  - Obtain 123I scan:
    - Eighty-five percent are “cold” nodules with a 10–25% chance of malignancy.
    - Five percent are “hot” nodules with only a 1% chance of malignancy.
    - Surgery is indicated if serial T4 levels fail to regress and future biopsies are worrisome.
- **Malignant (15%):** Surgery.
- **Cyst:** Drain completely (curative in 75% of cases). If cyst is > 4 cm, complex, or recurring even after three aspirations, send to OR for removal.

See Table 15-2.
### TABLE 15-2. Thyroid Cancer

<table>
<thead>
<tr>
<th></th>
<th>Papillary</th>
<th>Follicular</th>
<th>Medullary</th>
<th>Anaplastic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Most common type of thyroid cancer</strong></td>
<td>Not common in populations that do not have iodine deficiency</td>
<td>Rarest but worst prognosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Percent</strong></td>
<td>80–85 (75% of pediatric thyroid cancer)</td>
<td>5–20%</td>
<td>5–10%</td>
<td>1–5%</td>
</tr>
<tr>
<td><strong>Risk factors</strong></td>
<td>Radiation</td>
<td>Dyshormonogenesis</td>
<td>MEN II in 30–40%</td>
<td>Prior diagnosis of well-differentiated thyroid cancer, iodine deficiency</td>
</tr>
<tr>
<td><strong>Age group</strong></td>
<td>30–40</td>
<td>40–50</td>
<td>50–60</td>
<td>60–70</td>
</tr>
<tr>
<td><strong>Sex (F/M)</strong></td>
<td>2/1</td>
<td>3/1</td>
<td>1.5/1</td>
<td>1.5/1</td>
</tr>
<tr>
<td><strong>Signs and symptoms</strong></td>
<td>Painless mass, Dysphagia, Dyspnea, Hoarseness, Euthyroid</td>
<td>Painless mass, Rarely hyperfunctional</td>
<td>Painful mass, Palpable lymph node (LN) (15–20%), Dysphonia, Dyspnea, Hoarseness</td>
<td>Rapidly enlarging neck mass (large mass at presentation), Neck pain, Dysphagia, Hard, fixed LN (50%)</td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td>FNA, CT or MRI (to assess local invasion)</td>
<td>FNA, CT or MRI (to assess local invasion)</td>
<td>FNA, Presence of amyloid is diagnostic, Check for calcitonin</td>
<td>FNA</td>
</tr>
<tr>
<td><strong>Metastases</strong></td>
<td>Lymphatic (5% at time of presentation)</td>
<td>Hematogenous</td>
<td>Lymphatic (local neck and mediastinal nodes), Local (into trachea and esophagus)</td>
<td>Aggressive local disease, 30–50% have synchronous pulmonary mets at time of diagnosis</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Minimal cancer (&lt; 1.5 cm): Limited lobectomy and isthmectomy, Other: Total or near-total thyroidectomy for cancers &gt; 1.5 cm, For + LN, modified radical neck dissection, ^131^I ablation or thyroid suppression (with thyroid hormone) for patients with residual thyroid tissue or LN mets</td>
<td>Minimal cancer (&lt; 4 cm): Lobectomy and isthmectomy, Other: Total or near-total thyroidectomy for cancers &gt; 4 cm, For + LN, modified radical neck dissection, ^131^I ablation for patients with residual thyroid tissue or LN mets</td>
<td>Sporadic (80%): Total thyroidectomy, Familial (20%): Total thyroidectomy and central neck node dissection, No value for ^131^I ablation, Follow patients with calcitonin levels</td>
<td>Debulking resection of thyroid gland and adjacent structures, External radiation therapy (XRT), Doxorubicin-based chemotherapy</td>
</tr>
</tbody>
</table>

(continues)
TABLE 15-2.  Thyroid cancer (continued)

<table>
<thead>
<tr>
<th>Papillary</th>
<th>Follicular</th>
<th>Medullary</th>
<th>Anaplastic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prognosis</td>
<td>Worse for older</td>
<td>Worse for older</td>
<td>Poor prognosis</td>
</tr>
<tr>
<td></td>
<td>patients and those with distant mets</td>
<td>patients, those with distant mets, tumor size &gt; 4 cm, high tumor grade</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Presence of + LN not strongly correlated with overall survival</td>
<td>Presence of + LN not strongly correlated with overall survival</td>
<td></td>
</tr>
</tbody>
</table>

Parathyroid

Development

- Superior parathyroid glands (2) develop from fourth pharyngeal pouch.
- Inferior parathyroid glands (2) develop from third pharyngeal pouch.

Anatomy

- Weight < 50 mg/gland.
- 3 × 3 × 3 mm.
- Adult position of superior gland constant and next to the superior lobes of thyroid.
- Inferior glands have more variable position (posterior/lateral to thyroid and below inferior thyroid artery).
- Not uncommon for one of the inferior glands to be “missing.”
- Most common location in thymus. Other sites include intravagal, groove in carotid sheath.
- Yellow-brown tissue similar to surrounding fatty tissue.
- Histology: Normal gland contains mainly chief cells (produce parathyroid hormone [PTH]), with occasional oxyphils.
- Vasculature: Inferior thyroid arteries; superior, middle, and inferior thyroid veins.

Physiology/Ca l C i u M h oM eost asis

- **Primary function**: Regulation of calcium and phosphate metabolism by PTH, vitamin D, and calcitonin.
- Primary organ systems involved: GI tract, bone, kidney.

PTH

- Synthesized by the parathyroid glands (see Figure 15-4).
- Serum calcium levels regulate secretion of cleaved PTH by negative feedback mechanism.
- Bone:
  - Stimulates osteoclasts (increased bone resorption).

WARD TIP

FNA is less reliable if patient has history of irradiation, and initial OR biopsy may be appropriate.

EXAM TIP

Of the half of serum calcium in an ionized, active form, 80% is bound to albumin, and 20% is found in a citrate complex.
Osteoclast Activation

Transport

Ca++ Reabsorption

Ca++ and PO₄ Absorption

↑Serum Calcium

Ca++ and PO₄ Absorption

↑1,25 Vitamin D

↑Ca++

Ca++ Reabsorption

Serum Calcium

EXAM TIP
Calcitonin tones down Ca²⁺ levels.

WARD TIP
Hypercalcemia (calcium > 13 mg/dL and symptomatic): Treat with saline, furosemide, bisphosphonates, and, if needed, antiarrhythmic agents.

EXAM TIP
Hyperparathyroidism = ↑Ca²⁺. Think: Stones, bones, groans, and moans.

FIGURE 15-4. Actions of parathyroid hormone.

Hyperparathyroidism = ↑Ca²⁺. Think: Stones, bones, groans, and moans.

Primary

Due to overproduction of PTH, causing increased absorption of calcium from intestines, increased vitamin D₃ production, and decreased renal calcium excretion, thereby raising the overall serum level of calcium and lowering the amount of phosphorus.

Incidence: 1/4,000 in the United States.

Risk factors: MEN I, MEN IIA, history of radiation.

Signs and symptoms:
- “Stones”: Kidney stones.
- “Bones”: Bone pain, pathologic fractures, subperiosteal resorption.
- “Groans”: Nausea, vomiting, muscle pain, constipation, pancreatitis, peptic ulcer disease.
- “Moans”: Lethargy, confusion, depression, paranoia.

Etiology:
- Solitary adenoma: 85–90%
- Four-gland hyperplasia: 10%
- Cancer: < 1%
Preop imaging: Ultrasound, Sestamibi scan, CT/magnetic resonance imaging (MRI), operative exploration.

**Diagnosis:** Elevation of plasma PTH, with inappropriately high serum calcium. Check urine for calcium to rule out diagnosis of familial hypocalciuric hypercalcemia (will be low if familial disease, and high if primary hyperparathyroidism).

**Treatment:**
- Solitary adenoma: Solitary parathyroidectomy with neck exploration to identify and possibly biopsy 3 remaining glands. If preop sestamibi scan is done to localize area, no need for neck exploration.
- Multiple gland hyperplasia: Remove three glands, or all four with reimplantation of at least 30 g of parathyroid tissue in forearm or other accessible site to retain function (this makes it easier to resect additional parathyroid gland if hyperparathyroid state persists).

**Outcome:**
- First operation has 98% success rate.
- Reoperation has 90% success rate if remaining gland is localized preop.

**Secondary**
Increased PTH due to hypocalcemia that is the result of chronic renal failure (phosphate retention → low calcium), GI malabsorption, osteomalacia, or rickets.

**Signs and symptoms:**
- Bone pain from renal osteodystrophy and pruritus.
- Patients are often asymptomatic.

**Diagnosis:** Made by labs in asymptomatic patient; usually due to four-gland hyperplasia.

**Treatment:**
- Nonsurgical: In renal failure patients, correct calcium and phosphate.
  - Restrict phosphorus intake, treat with phosphorus-binding agents and calcium/vitamin D supplementation. Adjust dialysate to maximize calcium and minimize aluminum.
- Surgical: Indicated for intractable bone pain or pruritus, or pathologic fractures, with failure of medical therapy. No role for parathyroid surgery in secondary HPTH.
  - Perform renal transplant if necessary.

**Tertiary**
Due to persistent hyperparathyroidism after treatment for secondary hyperparathyroidism. Due to autonomously functioning parathyroid glands that are resistant to negative feedback from high calcium levels. Usually s/p renal transplant.

- Usually a short-lived phenomenon.
- If persistent, surgery is indicated (3½-gland parathyroidectomy).

**Hyperparathyroidism**
- Uncommon.

**Etiology**
- Surgically induced: Following total thyroidectomy; usually transient and treated if symptoms develop.
- Congenital absence of all four glands.
- DiGeorge syndrome: Absence of parathyroid and thymus.
- Functional: Chronic hypomagnesemia.

---

**EXAM TIP**

**Osteitis Fibrosa Cystica**
- Caused by hyperparathyroidism, characterized by painful, cyst-like brown tumor infiltrating the bone. Tumors regress with management of hyperparathyroidism.

---

**WARD TIP**

Not all patients with hypercalcemia have hyperparathyroidism. Hypercalcemia of malignancy (due to tumor-secreted PTH-related protein) must be ruled out. Malignancies commonly implicated include colon, lung, breast, prostate, head, neck, and multiple myeloma.

---

**EXAM TIP**

Patients with hereditary hyperparathyroidism (i.e., MEN Ia/b) have a high recurrence rate; total parathyroidectomy with forearm reimplantation is indicated to facilitate potential reoperation if HPTH persists. Patients with sporadic four-gland hyperplasia may undergo total parathyroidectomy with reimplantation or three-gland excision.
**Signs and Symptoms**
- Numbness and tingling of circumoral area, fingers, toes.
- Anxiety, confusion.
- May progress to tetany, hyperventilation, seizures, heart block.

**Treatment**
- Supplementation with PO calcium and vitamin D (to help GI absorption).
- Pseudohypoparathyroidism: Familial disease causing resistance of PTH at target tissue. Patients remain hypocalcemic and hyperphosphatemic despite bone resorption from elevated PTH. Treatment consists of calcium and vitamin D supplementation.

---

**Parathyroid Cancer**

**Signs and Symptoms**
- Forty to 50% present with firm, fixed mass that is palpable.
- Extremely high calcium and PTH levels. Usually has high levels of human chorionic gonadotropin (hCG—tumor marker).
- Neck pain, voice change (due to lesion in RLN).

**Treatment**
- En bloc surgical resection of mass and surrounding structures, along with ipsilateral thyroid lobectomy, and ipsilateral lymph node dissection.
- Postop external radiation therapy (XRT) and chemotherapy are not usually beneficial.
- Postop complications: RLN damage, severe hypocalcemia (hungry bone syndrome).
- Five-year survival: 70%.

---

**Adrenal Gland**

**Anatomy**
- Bilateral retroperitoneal organs, anterior and medial to superior pole of kidneys.
- At level of T11.
- Size: 3–6 g, 5 × 2.5 cm.
- Vasculature: Branches of aorta, inferior phrenic, and renal arteries.
- Venous drainage: Right side drains to inferior vena cava (IVC), and left side drains to the left renal vein.

**Histology**
- Cortex.
- Glomerulosa: 15%; aldosterone synthesis.
- Fasciculata: 75%; steroids and cortisol synthesis.
- Reticularis: 10%; cortisol, androgen, and estrogen secretion.

**Physiology**

See section on endocrine organs and Figure 15-5.
hyperplasia

Area of cortex expands and becomes hyperfunctioning.

aDenoMa

- Most are unilateral.
- If adrenal gland < 6 cm, usually observe unless hormonally active (↑ cortisol, ↑ ACTH) or increasing in size.
- If adrenal gland > 6 cm, surgically resect due to increased risk of adrenocortical carcinoma.

aDrenal Cortal CaringoMa

definition

Very rare adrenal tumor.

epidemiology

Affects women more than men, peak < 5 years old or 30–40 years old.

Signs and Symptoms

- Vague abdominal complaints (due to enlarging retroperitoneal mass).
- Symptoms related to overproduction of a steroid hormone (most tumors are functional).

diagnosis

- Twenty-four-hour urine collection for cortisol, aldosterone, catecholamines, metanephrine, vanillylmandelic acid (VMA), 17-OH corticosteroids, 17-ketosteroids.
- CT (lesions > 7 mm) or MRI (especially for assessing IVC invasion).
- Chest x-ray (CXR) to rule out pulmonary metastases.

Treatment

- Radical en bloc resection, but only one third of adrenal carcinomas are operable.
- If resection cannot be completed, debulk to reduce amount of cortisol-secreting tissue.
Bone metastases should be palliated with XRT.
- No role for chemotherapy.
- Monitor steroid hormone levels postop.
- Recurrence also warrants resection.
- Recurrence: Lungs, lymph nodes, liver, peritoneum, bone.
  - In 10% of bilateral adrenalectomies, patients may develop Nelson’s syndrome—excess production of ACTH from pituitary adenoma. Pituitary adenoma often causes visual disturbances (due to mass effect on chiasm), hyperpigmentation (↑ melanocyte-stimulating hormone [MSH]), and amenorrhea as well.

**Prognosis**
- Seventy percent present in stage III or IV.
- Five-year survival 40% for complete resection.
- If local invasion, median survival time is 2–3 years.

**Cushing’s syndrome**

**Definition**
Excessive cortisol production.

**Causes**
- Iatrogenic administration of corticosteroids most common cause!
- Pituitary tumor that secretes ACTH (i.e., Cushing’s disease).
- Ectopic ACTH secretion by tumor elsewhere stimulates adrenal cortisol production.
- Adrenal tumor that secretes cortisol.

**Signs and Symptoms**

**Appearance**
- Weight gain
- Truncal obesity
- Extremity wasting
- Buffalo hump
- Moon facies
- Acne
- Purple striae
- Hirsutism

**Physiologic**
- Mild glucose intolerance
- Amenorrhea
- Decreased libido
- Depression
- Impaired memory
- Muscle weakness

**Diagnosis**
See Table 15-3.
- Confirm presence of hypercortisolism:
  - Low-dose dexamethasone test.
  - Twenty-four-hour urinary cortisol looking for cortisol or its metabolites (e.g., 17-hydroxycorticosteroids).
  - Direct measurement of serum cortisol.

---

**Typical scenario:** A young male presents with weight gain, especially in the trunk; loss of muscle mass; and a buffalo hump. He has recently been noted to be mildly glucose intolerant. His past medical history is significant for severe asthma, for which he is chronically on steroids. Think Cushing’s syndrome secondary to exogenous administration of steroids.
### TABLE 15-3. Assays in the Workup of Cushing’s Syndrome

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Interpretation</th>
</tr>
</thead>
</table>
| 24-hour urinary free cortisol or single dose dexamethasone suppression test | Normal | Hypercortisolism can be ruled out:  
- Low-dose dexamethasone (2 mg) normally decreases urinary cortisol levels in normal patients. Lack of suppression confirms hypercortisolism  
- High-dose dexamethasone (8 mg) will only decrease urinary cortisol if pituitary-dependent cause of ↑ACTH (Cushing’s disease), but will not suppress cortisol at all if cause is either ectopic ACTH production or a primary adrenal tumor |
| Plasma ACTH                                                          | Very high/intermediate value/low or undetectable | Ectopic ACTH production/Pituitary tumor/Adrenal source: Either adenoma, hyperplasia, or, very rarely, cancer |
| Urinary 17-ketosteroids                                             | < 10 mg/day/60 mg/day/in between | Adenoma/Cancer/Likely hyperplasia |
| Metyrapone test                                                      | Increased ACTH | Pituitary cause |
| Petrosal sinus sampling                                             | Sinus/plasma ACTH ratio > 3 after corticotropin-releasing hormone (CRH) administration | Identifies Cushing’s disease with 100% sensitivity |

- Determine whether cortisol production is pituitary dependent or independent: High-dose dexamethasone test.
- CT (adrenals): Can distinguish cortical hyperplasia from tumor with sensitivity > 95%, but lacks high specificity.
- MRI (sella).
- Petrosal sinus sampling (will see elevated ACTH if pituitary tumor).
- Adrenal cortisol production: High cortisol, low plasma ACTH (due to negative feedback from cortisol production), and no suppression of cortisol with high-dose dexamethasone suppression test.
- Ectopic ACTH production: Increased plasma cortisol and ACTH, and no suppression of cortisol (because only ACTH from pituitary, and not ectopic source, is capable of negative feedback).
- Pituitary tumor (Cushing’s disease): Mild elevation of ACTH and cortisol is successfully suppressed with high-dose dexamethasone (negative feedback on pituitary).

### Treatment
- Cushing’s disease: Transsphenoidal resection of pituitary adenoma.
- Adrenal adenoma: Adrenalectomy.
- Adrenal carcinoma: Adrenalectomy.
- Ectopic ACTH: Resection of primary lesion (usually in lung).
- Unresectable lesions and recurrence should be debulked for palliation.
- Medical treatment to suppress cortisol production: Metyrapone (inhibits cortisol production), aminoglutethimide, mitotane, ketoconazole.

### Exam Tip
Most common cause of ectopic ACTH production is small cell lung cancer (a.k.a. oat cell carcinoma), followed by carcinoid tumors.

### Ward Tip
Low-dose dexamethasone suppression test: Single dose of steroid at 11 p.m., followed by measurement of serum and urinary cortisol levels at 8 a.m.  
Normal: < 5 µg/dL (because evening dose suppresses further release).  
Abnormal: > 5 µg/dL high-dose dexamethasone (8 mg) distinguishes pituitary cause (suppression) from adrenal or ectopic cause (no suppression).
**Addison’s Disease**

**Definition**

**Adrenal insufficiency:**
- Primary: Due to destruction of adrenal cortex with sparing of medulla.
- Secondary: Failure due to hypothalamic or pituitary abnormalities.

**Etiology**

**Primary**
- Post-adrenalectomy.
- Autoimmune adrenalitis.
- Tuberculosis (TB).
- Fungal infection.
- Acquired immune deficiency syndrome (AIDS).
- Metastatic cancer.
- Familial glucocorticoid deficiency.

**Secondary**
- Exogenous steroids.
- Craniopharyngioma.
- Pituitary surgery or irradiation.
- Empty sella syndrome.

**Addisonian Crisis**
- Acute situation due to some extrinsic stressor (i.e., infection, surgery).

**Signs and Symptoms**
- Nausea.
- Vomiting.
- Abdominal pain.
- Tachycardia.
- Weight loss.
- Weakness.
- Fatigue.
- Lethargy.
- Hyperpigmentation (low levels of cortisol cause ↑ pituitary production of proopiomelanocortin [POMC], which is precursor to both ACTH as well as MSH).
- Fever and hypovolemic shock in Addisonian crisis.

**Diagnosis**
- Hyponatremia, hyperkalemia (due to ↓ adosterone, which is normally produced in adrenals).
- ACTH stimulation test: Give ACTH and measure cortisol level after 30 minutes. If adrenal failure is present, there will be no increase in cortisol.
- Baseline ACTH level is elevated in patients with primary failure due to absence of negative feedback.

**Treatment**
- Glucocorticoid therapy for primary and secondary causes.
- Additional mineralocorticoid therapy for primary cause.
- Addisonian crisis: Volume (D₃NS) and glucocorticoids IV.
**Hyperaldosteronism**

**Definition**
- Hyperaldosteronism
- Primary
- Secondary

**Etiology**
- Primary (due to excessive aldosterone secretion): Conn’s syndrome:
  - Aldosterone-secreting tumor (66%)
  - Idiopathic adrenocortical hyperplasia (30%)
- Secondary (due to elevated renin → elevated aldosterone):
  - Renal artery stenosis
  - Cirrhosis
  - CHF
  - Normal pregnancy

**Signs and Symptoms**
- Hypertension
- Muscle weakness and cramping
- Headache
- Polyuria
- Polydipsia
- Hypokalemia

**Diagnosis**
- **Primary/Conn’s syndrome:**
  - Diastolic hypertension without edema.
  - Elevated plasma aldosterone.
  - Normals or low plasma renin.
  - Hypokalemia, elevated urinary potassium (off antihypertensive medications).
  - Post-captopril plasma aldosterone:
    - Normally results in decreased aldosterone.
    - Diagnostic of hyperaldosteronism if ratio > 50.
  - Imaging:
    - CT picks up tumors > 1 cm. If there is an aldosteronoma, opposite adrenal appears atrophied.
    - Iodocholesterol scan: Picks up 90% of aldosteronomas and shows how functional they are. Hyperplasia will present as bilateral hyperfunction versus unilateral (for tumor).
    - If all imaging is nondiagnostic, then sample adrenal vein for aldosterone and cortisol pre- and post-ACTH.
    - Unilateral elevation of aldosterone or aldosterone/cortisol ratio indicates aldosterone-secreting adenoma.
    - Bilateral elevation of aldosterone is consistent with hyperplasia.

**Treatment**
- Hyperplasia: Medical treatment with spironolactone, nifedipine, amiloride, and/or other antihypertensive. **No surgery!**
- Adenoma: Laparoscopic adrenalectomy.
- Outcome:
  - Most patients become normotensive and normokalemic with treatment.
  - Twenty to 30% have recurrent hypertension (HTN) in 2–3 years, but for unknown reason.
- For secondary hyperaldosterone, treat underlying cause.

---

**WARD TIP**

Renin is produced in the juxtaglomerular (JG) cells of the kidney when blood pressure is low, and stimulates conversion of angiotensinogen to angiotensin I in the kidney. Angiotensin I is converted to angiotensin II in the lung. Angiotensin II causes adrenals to produce aldosterone.

---

**WARD TIP**

In working up a patient for suspected Conn's syndrome, make sure it isn't just a patient with uncontrolled hypertension on potassium-wasting diuretics.
**hypoadrenalism**

**Definition**
Decreased aldosterone without a change in cortisol production.

**Etiology**
- Congenital error of aldosterone synthesis.
- Failure of zona glomerulosa (autoimmune).
- Status post adrenalectomy.
- Drug inhibition.

**Signs and Symptoms**
- Postural hypotension
- Persistent severe hyperkalemia
- Muscle weakness
- Arrhythmia

**Treatment**
Mineralocorticoid therapy.

---

**Medullary Tumors**

**Neuroblastoma**

**Definition**
Embryonal neural crest tumor occurring primarily in children (small round blue cell tumor).
- Fourth most common pediatric malignancy.
- Can occur anywhere along sympathetic chain—50% in adrenal, 25% in paraspinal ganglia, 20% in thorax, 5% in pelvis.
- May spontaneously differentiate and regress.
- Aggressive tumor that commonly presents with distant metastases, in 50% of infants and 66% of older children (to lymph node, bone, liver, subcutaneous tissue).

**Associated Diseases**
- Neurofibromatosis
- Beckwith–Wiedemann syndrome
- Trisomy 18

**Signs and Symptoms**
- Abdominal or flank mass.
- Respiratory distress.
- Subcutaneous blue tumor nodules (blueberry muffin sign).

**Diagnosis**
- Imaging: CT for staging, MRI.
- Urinary tumor markers: Elevated 24-hour levels of homovanillic acid (HVA), VMA, and metanephrines.

---

**Exam Tip**

**Typical Scenario:** A 50-year-old male has an abdominal CT scan. Appendicitis is confirmed, and the patient undergoes a successful appendectomy and uneventful recovery. The CT scan also revealed a 2-cm adrenal mass. What’s your next step? Think Test for functionality. If the mass is functional, it should be resected regardless of its small size.
Treatment

- Localized disease (stages I and II): Surgical resection.
- Nonlocalized disease (stage II): Surgical resection with chemotherapy +/− radiation.
- Metastatic disease (stage IV): Surgical resection with chemotherapy +/− radiation.
- Lymph node metastasis warrants XRT.

Prognosis

- Mortality lower when diagnosed within first year of life.
- Five-year survival 90% for disease confined to primary site; 20–40% for disseminated disease.

Pheochromocytoma

Definition

Chromaffin cell tumor that is most commonly in the adrenal medulla (> 90%), but may be anywhere along the sympathetic chain. Most common site for extra-adrenal pheochromocytoma is the organ of Zuckerkandl.

Associated risk Factors

- MEN IIA and IIB (usually result in bilateral adrenal tumors)
- Von Hippel–Lindau disease
- Neurofibromatosis
- Family history

Signs and Symptoms

- HTN: May be sustained elevation, normal with paroxysmal HTN, or sustained HTN with acute elevations. Most common presenting symptom.
- Headaches.
- Anxiety, palpitations, pallor, diaphoresis.

Diagnosis

- Twenty-four-hour urine collection for catecholamine by-products: VMA, metanephrine, normetanephrine.
- Serum epinephrine and norepinephrine (note: if elevated epinephrine, must be adrenal tumor).
- Clonidine test: Will suppress plasma catecholamine concentrations in normal patients but not in patients with pheochromocytoma.
- CT or MRI, positron emission tomography (PET), or nuclear scan ([^31]I-metaiodobenzylguanidine).

Treatment

- Preop α-adrenergic blockade (phenoxybenzamine) followed by β-blockers for persistent tachycardia.
- Intraoperative arterial blood pressure monitoring is essential because extreme changes in blood pressure may occur with manipulation.
- Important to ligate veins first to prevent unintentional release of catecholamines that may result from manipulation of adrenal gland.
- If malignant:
  - Resect recurrences and metastases when they occur.
  - Treat with catecholamine blockade.
  - Use XRT for bony mets.
  - Chemotherapy has a 60% response rate.

**WARD TIP**

Most common extra-adrenal location is organ of Zuckerkandl (to left of aortic bifurcation at inferior mesenteric artery).

**EXAM TIP**

Pheochromocytoma

- If extra-adrenal, then more likely malignant
- If bilateral, more likely familial

**WARD TIP**

10% rule for pheochromocytoma:
- 10% malignant
- 10% familial
- 10% extra-adrenal
- 10% bilateral
- 10% in children

**WARD TIP**

Alpha blockade must precede beta blockade in patients with pheochromocytoma. The use of β-blockers will cause negative inotropic effects and result in unopposed α-induced vasoconstriction, which may precipitate malignant hypertension and cardiac failure.

**WARD TIP**

10% rule for pheochromocytoma:
- 10% malignant
- 10% familial
- 10% extra-adrenal
- 10% bilateral
- 10% in children
Prognosis
For malignant tumors: 5-year survival 36–60%.

Pituitary

anatomy

See Figure 15-6.
- Ectodermal origin.
- Relations:
  - Within sella turcica (floor of sella is roof of sphenoid sinus).
  - Surrounded by dura.
  - Optic chiasm superior and anterior to pituitary stalk.
  - Lateral cavernous sinuses.
  - Superior: Diaphragma sella (meningeal tissue).
  - Size: 12 × 9 × 6 mm, 500–600 mg.
- Parts:
  - Adenohypophysis: Anterior lobe.
  - Neurohypophysis: Posterior lobe.
- Blood supply:
  - Anterior lobe lacks a direct supply. Portal channels from the hypothalamus and posterior pituitary supply it.
  - Posterior pituitary is supplied by middle and inferior hypophyseal arteries, branches of the internal carotid artery.
  - Drains via cavernous sinuses to petrosal sinuses to jugular veins.
- For hormone actions, see section on endocrine organs.

adenoma

- Benign tumor arising from anterior lobe.
- Divided into two types based on size:
  - Macroadenoma: > 1 cm diameter.
  - Microadenoma: < 1 cm diameter.
**Signs and Symptoms**

- **Macroadenomas:** Visual loss (bilateral hemianopsia), hypopituitarism, headache, hyperprolactinemia due to compression of surrounding structures.
- **Microadenomas:** Signs and symptoms depend on type of hormone overproduced.
- **Prolactinoma (most common):** Secondary amenorrhea, galactorrhea.
- **Growth hormone-producing tumor:** Gigantism or acromegaly depending on age of patient; also, coarse facial features, thick finger and heel pads, cardiomegaly, hepatomegaly, enlarged mandible, increased teeth spaces, neuropathy, arthropathy, osteoporosis, HTN, diabetes mellitus (DM), goiter.
- **ACTH:** See Adrenal section.
- Multihormonal.

**Diagnosis**

Imaging and clinical presentation.

**Treatment**

- Preoperative: Complete endocrine assessment; electrolytes to look for borderline diabetes insipidus.
- Surgery:
  - Transsphenoidal approach: Results in improved function of remaining pituitary gland.
  - Transcranial approach: When transsphenoidal not possible due to location of carotid arteries, extrasellar tumor.
  - Perioperative glucocorticoids, serial visual field assessment, repeat endocrine assessment.
  - Postop XRT: For large lesions.
- Primary radiation therapy: Consider when surgery contraindicated for other reasons in nonfunctioning tumor as primary therapy may worsen preexisting hypopituitarism.
- Medical treatment:
  - For prolactinomas: Bromocriptine.
  - For GH-secreting adenomas: Somatostatin, which decreases tumor size in 20–50%, normalizes GH in 50%, and normalizes IGF-1 in 40–80%.

**Complications**

- Death due to direct hypothalamic injury (<1%).
- Delayed mortality: Cerebrospinal fluid (CSF) leak, vascular injury.
- Morbidity: Diabetes insipidus (2–17%), cerebrovascular accident (CVA), meningitis.

**Sheehan’s Syndrome**

Postpartum infarction and necrosis of pituitary leading to hormonal failure.

**Etiology**

Pituitary ischemia due to hemorrhage, hypovolemic shock, pituitary portal venous thrombosis.

**Signs and Symptoms**

- Failure of lactation.
- Amenorrhea.
- Progressive decreased adrenal function and thyroid function.
posterior pituitary Disorders

**Syndrome of inappropriate Antidiuretic hormone (SIADH)**
- Occurs in 15% of hospital patients.
- Impaired water secretion.
- Hypersecretion of ADH results in increased urinary sodium with elevation of urine osmolality.
- Causes: CNS injury, cancer, trauma, drugs.

**diabetes insipidus**
- Decreased ADH secretion.
- Impaired water conservation; large volumes of urine, leads to increased plasma osmolality and thirst.
- One third idiopathic; two thirds due to tumor or trauma.
# The Spleen

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<tr>
<td><strong>Complications</strong></td>
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**Description**

The spleen is an immunologic organ without distinct lobes or segments that weighs about 100–175 g. It is responsible for the removal of old red blood cells and bacteria from the blood circulation.

**Anatomic Boundaries**

Left upper quadrant (LUQ) of the abdomen, between the 8th and 11th ribs (see Figure 16-1).

- Superior: Left diaphragm leaf.
- Inferior: Colon, splenic flexure, and phrenocolic ligament.
- Medial: Pancreas (tail) and stomach.
- Lateral: Rib cage.
- Anterior: Rib cage, stomach.
- Posterior: Rib cage.

**Splenectomy**

See Table 16-1 for indications.

**Types**

- Laparoscopic
- Open

**Complications**

- Overwhelming postsplenectomy sepsis.
- Atelectasis (not taking deep breaths due to pain)/pneumonia (due to atelectasis sequestering bacteria).
- Pleural effusion (usually on the left).
- Subphrenic abscess.
- Injury to pancreas (because tail of pancreas “hugs” spleen).
- Postoperative hemorrhage.
- Thrombocytosis—many of the platelets that were sequestered in the spleen are now out in the circulation.

**Tumors of the Spleen**

**Benign**

- Hemangioma/lymphangioma
- Hamartomas
- Primary cyst/echinococcal cyst

**Malignant**

- Either lymphomas or myeloproliferative diseases.
- Rare site for solid tumor metastatic disease.
- A common site for metastases especially in lung and breast. However, it is rarely clinically significant and usually an autopsy finding.
**Figure 16-1.** The spleen and its relationships. (Reproduced, with permission, from Morton DA, Foreman K, Albertini K. The Big Picture: Gross Anatomy. New York, NY: McGraw-Hill Education; 2011. Figure 9-3.)
<table>
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<th>Disease/Condition</th>
<th>Indications for Splenectomy</th>
<th>Response to Splenectomy</th>
</tr>
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<tr>
<td>Hereditary spherocytosis</td>
<td>Hemolytic anemia, recurrent transfusions, intractable leg ulcers</td>
<td>Improves or eliminates anemia</td>
</tr>
<tr>
<td>Hereditary elliptocytosis</td>
<td>Limited role for splenectomy</td>
<td>—</td>
</tr>
<tr>
<td>Pyruvate kinase deficiency</td>
<td>Only in severe cases, recurrent transfusions</td>
<td>Decreased transfusion requirement, palliative only</td>
</tr>
<tr>
<td>Glucose-6-phosphate dehydrogenase deficiency</td>
<td>None</td>
<td>—</td>
</tr>
<tr>
<td>Warm-antibody autoimmune hemolytic anemia</td>
<td>Failure of medical (steroid) therapy</td>
<td>60–80% response rate, recurrences common</td>
</tr>
<tr>
<td>Sickle cell disease</td>
<td>History of acute sequestration crisis, splenic symptoms, or infarction (consider concomitant cholecystectomy)</td>
<td>Palliative, variable response</td>
</tr>
<tr>
<td>Thalassemia</td>
<td>Excessive transfusion requirements, symptomatic splenomegaly, or infarction</td>
<td>Diminished transfusion requirements, relief of symptoms</td>
</tr>
<tr>
<td>Acute myeloid leukemia (AML)</td>
<td>Intolerable symptomatic splenomegaly</td>
<td>Relief of abdominal pain and early satiety</td>
</tr>
<tr>
<td>Chronic myeloid leukemia</td>
<td>Symptomatic splenomegaly</td>
<td>Relief of abdominal pain and early satiety</td>
</tr>
<tr>
<td>Chronic myelomonocytic leukemia</td>
<td>Symptomatic splenomegaly</td>
<td>Relief of abdominal pain and early satiety</td>
</tr>
<tr>
<td>Essential thrombocythemia</td>
<td>Only for advanced disease (i.e., transformation to myeloid metaplasia or AML) with severe symptomatic splenomegaly</td>
<td>Relief of abdominal pain and early satiety</td>
</tr>
<tr>
<td>Polycythemia vera</td>
<td>Only for advanced disease (i.e., transformation to myeloid metaplasia or AML) with severe symptomatic splenomegaly</td>
<td>Relief of abdominal pain and early satiety</td>
</tr>
<tr>
<td>Myelofibrosis (agnogenic myeloid metaplasia)</td>
<td>Severe symptomatic splenomegaly</td>
<td>76% clinical response at 1 year, high risk of hemorrhagic, thrombotic, and infectious complications (26%)</td>
</tr>
<tr>
<td>Chronic lymphocytic leukemia</td>
<td>Cytopenias and anemia</td>
<td>75% response rate</td>
</tr>
<tr>
<td>Hodgkin’s disease</td>
<td>Surgical staging in selected cases</td>
<td>—</td>
</tr>
<tr>
<td>Non-Hodgkin’s lymphoma</td>
<td>Cytopenias, symptomatic splenomegaly</td>
<td>Improved complete blood count values, relief of symptoms</td>
</tr>
<tr>
<td>Khiopathic thrombocytopenic purpura</td>
<td>Failure of medical therapy, recurrent disease</td>
<td>75–85% rate of long-term response</td>
</tr>
</tbody>
</table>
Sp e n i c I n j u r y

**s i g n s a n d s y m p t o m s**

- History: Check for preexisting diseases that cause splenomegaly (these patients are more vulnerable to splenic injury), details of injury mechanism.
- Exam: Look for peritoneal irritation, Kehr’s sign, left-sided lower rib fractures, external signs of injury.

**T r e a t m e n t**

**I n i t i a l**

- Airway, breathing, circulation (ABCs).
- Patients who are stable or who stabilize with fluid resuscitation may be considered for conservative management.
- Further diagnostic tools:
  - Computed tomographic (CT) scan: Able to define injury precisely.
  - Ultrasound (US): May be used for initial assessment to detect hemoperitoneum as a part of focused abdominal sonography for trauma (FAST) exam.
  - Diagnostic peritoneal lavage (DPL): Not specific for splenic injury but will show hemoperitoneum.
  - Angiogram: May be able to use therapeutically in the stable patient (embolization of CT-identified injury).

**D e f i n i t i v e**

- Nonoperative management criteria:
  - Stable.
  - Injury grade I or II (see Table 16-2).
  - No evidence of injury to other intra-abdominal organs.
  - Consists of bed rest, nasogastric tube (NGT) decompression, monitored setting, serial exam, and hematocrits.

---

**Table 16-1. Indications for and expected Response to Splenectomy in Various Diseases and Conditions** (continued)

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<tr>
<th>Disease/Condition</th>
<th>Indications for Splenectomy</th>
<th>Response to Splenectomy</th>
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<td>Thrombotic thrombocytopenic purpura</td>
<td>Excessive plasma exchange requirement</td>
<td>Typically curative</td>
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<tr>
<td>Abscesses of the spleen</td>
<td>Therapy of choice</td>
<td>Curative</td>
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<tr>
<td>Symptomatic parasitic cysts</td>
<td>Therapy of choice</td>
<td>Curative; exercise caution not to spill cyst contents</td>
</tr>
<tr>
<td>Symptomatic nonparasitic cysts</td>
<td>Partial splenectomy for small cysts; unroofing for large cysts</td>
<td>Curative</td>
</tr>
<tr>
<td>Gaucher's disease</td>
<td>Hypersplenism</td>
<td>Improves cytopenias; does not correct underlying disease</td>
</tr>
<tr>
<td>Niemann–Pick disease</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


---

**WARD TIP**

Transfusing platelets in TTP is thought to “fuel the fire” and exacerbate consumption of platelets and clotting factors, resulting in more thrombi in the microvasculature. Plasmapheresis is the treatment of choice.

---

**EXAM TIP**

The spleen is the most commonly injured organ in blunt abdominal trauma, and trauma is the most common reason for splenectomy.
Causes of splenic rupture:
- Trauma: Rib fractures on the left (especially the 9th and 10th, which make up 20% of cases).
- Spontaneous rupture (associated with mononucleosis).

Thirty percent of patients with splenic injury will present with hypotensive shock due to hemorrhage.

Radiographic signs of splenic injury:
- CT: Low-density mass or intrasplenic accumulation of contrast.
- US: Perisplenic fluid, enlarged spleen, irregular borders, abnormal position, increase in size over time.

Patients with a vascular blush on CT scan are likely to fail nonoperative management.

Operative management indications:
- Signs and symptoms of ongoing hemorrhage.
- Injury ≥ grade III.
- Failure of nonoperative management.

Exploratory laparotomy:
- Perform splenectomy if the spleen is the primary source of exsanguinating hemorrhage.
- If not, pack the area and search for other, more life-threatening injuries; address those first.
- Subsequently, return to inspection of spleen. Mobilize fully unless the only injury is a minor nonbleeding one.

Capsular bleeding and most grade II injuries: Apply direct pressure ± topical hemostatic agent.

Persistently bleeding grade II or III injuries: Suture lacerations.

Multiple injuries: Consider mesh splenorrhaphy for splenic preservation (especially in children).

Complex fractures: Perform anatomic resection if possible, based on demarcation after segmental artery ligation.

Perform splenectomy (see indications).

**Splanic Abscess**

**Causes**
- Sepsis seeding
- Infection from adjacent structures
• Trauma
• Hematoma
• IV drug use

**Signs and Symptoms**

• Fever, chills.
• LUQ tenderness and guarding.
• Spleen may or may not be palpable.

**Diagnosis**

• US: Enlarged spleen with areas of lucency contained within.
• CT: Abscess will show lower attenuation than surrounding spleen parenchyma. Defines abscess better than US.

**Treatment**

• Splenectomy for most cases.
• Percutaneous drainage for a large, solitary juxtacapsular abscess.

**Complications**

• Spontaneous rupture
• Peritonitis
• Sepsis

---

**WARD TIP**

Patients who fail nonoperative management usually do so within 48–72 hours. Delayed bleeding can occur up to several weeks after the injury.

**WARD TIP**

Pneumococcal, Haemophilus influenzae, and Neisseria meningitidis vaccines will be needed for patients undergoing splenectomy. It may be given 2 weeks postoperatively, or on the day of hospital discharge.
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Basic Anatomy

The breast is a modified sebaceous gland composed of glandular, fibrous, and adipose tissue (see Figure 17-1).

- Lies within layers of superficial pectoral fascia, anterior to the deep fascia of pectoralis major.
- Each mammary gland consists of approximately 15–20 lobules, each of which has a lactiferous duct that opens on the areola.
- Structural support provided by ligaments that extend from the deep pectoral fascia to the superficial dermal fascia, called Cooper's ligaments.
- Breast tissue frequently extends into axilla as the axillary tail of Spence.
- Is partitioned into quadrants by vertical and horizontal lines across the nipple: Upper inner quadrant (UIQ), lower inner quadrant (LIQ), upper outer quadrant (UOQ), and lower outer quadrant (LOQ).

Breast Development:
- Derived from ectoderm milk streak
- Estrogen: duct development
- Progesterone: lobular development

Blood Supply

- Arterial: Axillary artery via the lateral thoracic and thoracoacromial branches, internal mammary artery via its perforating branches, and adjacent intercostal arteries.

FIGURE 17-1. Cut-away diagram of a mature resting breast. The breast lies cushioned in fat between the overlying skin and the pectoralis major muscle. Both the skin and the retromammary space under the breast are rich with lymphatic channels. Cooper's ligaments, the suspensory ligaments of the breast, fuse with the overlying superficial fascia just under the dermis, coalesce as the interlobular fascia in the breast parenchyma, and then join with the deep fascia of breast over the pectoralis muscle. The system of ducts in the breast is configured like an inverted tree, with the largest ducts just under the nipple and successively smaller ducts in the periphery. After several branching generations, small ducts at the periphery enter the breast lobule, which is the milk-forming glandular unit of the breast. (Reproduced, with permission, from Peart O. Lange Q&A Mammography Examination. 2nd ed. New York: McGraw-Hill; 2009: 124.)
- Venous: Follows arterial supply; axillary, internal mammary, and intercostal veins; axillary vein responsible for majority of venous drainage.

**lymphatic drainage**

See Figure 17-2.

- Level I: Lateral to lateral border of pectoralis minor.
- Level II: Deep to pectoralis minor.
- Level III: Medial to medial border of pectoralis minor.
- **Rotter's nodes** lie between the pectoralis major and pectoralis minor muscles.
- Lymphatic drainage from nipple, areola, and lobules all drain in a sub-areolar lymphatic plexus.
- Ninety-seven percent drain to the axillary lymph nodes
- Two percent drain to the internal mammary nodes
  - Any quadrant can drain into the internal mammary nodes

**nerveS**

See Table 17-1.

---

**WARD TIP**

Venous drainage is largely responsible for metastases to the spine through the **paravertebral plexus of Batson**.

**WARD TIP**

Lymph node involvement by tumor tends to progress from level I to III. The higher the level, the worse the prognosis.

**OR TIP**

The medial pectoral nerve is actually lateral to the lateral pectoral nerve. The nerves are named according to their origin from the brachial plexus, not by their relation to one another on the chest wall.

**OR TIP**

The anesthesiologist should not paralyze the patient at surgery because the major nerves (long thoracic and thoracodorsal) are identified by observing muscle contraction when stimulating them with a forceps.

---

**Figure 17-2. Contents of the axilla.** In this figure there are five named and contiguous groupings of lymph nodes in the full axilla. Complete axillary dissection, as done in the historical radical mastectomy, removes all these nodes. However, note that the subclavicular nodes in the axilla are continuous with the supraclavicular nodes in the neck and nodes between the pectoralis major and minor muscles, named the interpectoral nodes or Rotter's lymph nodes. The internal mammary nodes probably drain independently from the breast. The sentinel lymph node, located in modern sentinel biopsy, is functionally the first and lowest node in the axillary chain. Anatomically, the sentinel lymph node is usually found in the external mammary group. (Reproduced from Zollinger RM Jr, Zollinger RM Sr. Zollinger's Atlas of Surgical Operations. 8th ed. New York, NY: McGraw-Hill; 2003: 397.)
**Boundaries for Mastectomy**

- Superior: Clavicle
- Inferior: Inframammary fold
- Medial: Sternum
- Lateral: Latissimus dorsi

**Boundaries for Axillary Dissection**

See Figure 17-2.

- Superior: Axillary vein.
- Posterior: Long thoracic nerve.
- Medial: Either lateral to, underneath, or medial to the medial border of the pectoralis minor muscle, depending on the level of nodes taken.
- Lateral: Latissimus dorsi muscle.

**Initial Evaluation of Patients with Possible Breast Disease**

- Complete medical history, including risk factors for breast cancer (see next page). Be sure to inquire about any history of nipple discharge or any changes in the size, shape, symmetry, or contour of the breasts.
- Physical examination (see Figure 17-3).
- Inspection: Note color, symmetry, size, shape, and contour, and check for dimpling, erythema, edema, or thickening of skin with a porous appearance (*peau d’orange*).
- Palpation: Palpate all four quadrants and the nipple–areolar complex for any discharge.
- Axillary nodes are palpated along the lateral border of anterior and posterior axillary fold, the medial and lateral wall of the axilla and the apex of the axilla.

**Table 17-1. Neural Structures Encountered During Major Breast Surgery**

<table>
<thead>
<tr>
<th>Nerves</th>
<th>Muscle(s)/Area Supplied</th>
<th>Functional Deficit if Injured</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long thoracic nerve (of Bell)</td>
<td>Serratus anterior</td>
<td>Winging of scapula</td>
</tr>
<tr>
<td>Thoracodorsal nerve</td>
<td>Latissimus dorsi</td>
<td>Cannot push oneself up from a sitting position, weak upper extremity adduction</td>
</tr>
<tr>
<td>Medial and lateral pectoral nerves</td>
<td>Pectoralis major and minor</td>
<td>Weakness of pectoralis muscles</td>
</tr>
<tr>
<td>Intercostobrachial nerve</td>
<td>Crosses axilla transversely to supply inner aspect of arm</td>
<td>Area of anesthesia on inner aspect of arm</td>
</tr>
</tbody>
</table>

**Exam Tip**

Typical scenario: A female with one or more risk factors for breast cancer presents with a mass in the upper outer quadrant of the breast. Think: She's at risk for cancer, and 50% of breast cancers occur in the upper outer quadrant. Therefore, the mass is likely to be malignant.

**WARD TIP**

Perform breast self-examination at same time each month (1 week after menstrual period is ideal).

**WARD TIP**

Palpable breast masses should have a core needle biopsy (as opposed to an FNA).
Evaluating a Palpable Breast Mass

**Approach**

See Figures 17-4 and 17-5. If age < 30, serial physical examination with observation for 2–4 weeks or until next menstrual period is an option.

**Differential Diagnosis**

- Infectious/inflammatory: Mastitis, fat necrosis, Mondor’s disease.
- Benign lesions: Fibroadenoma, fibrocystic changes, mammary duct ectasia, cystosarcoma phyllodes (occasionally malignant), intraductal papilloma, gynecomastia.

**Algorithm for Diagnosis**

```
ALGORITHM FOR DIAGNOSIS

Dominant Mass

Suspicious Not suspicious

Mammogram

Suspicious Not suspicious

Biopsy

Suspicious

Fine-needle Aspiration

Not suspicious

Consider Observation
```


**WARD TIP**

All persistent breast masses require evaluation.

**WARD TIP**

Only presentations nonsuspicious of cancer:

- Lactating woman with focal erythematous warm swelling.
- Cyclical changing mass in young woman with clear aspirate. Everything else is extensively worked up.

**EXAM TIP**

Typical scenario: A 42-year-old woman presents with an undiagnosed breast mass. Think: Evaluate without delay. Observation is not an option if age > 30.
Premalignant disease: Ductal carcinoma in situ (DCIS), lobular carcinoma in situ (LCIS).

Malignant tumors: Infiltrating ductal, infiltrating lobular, and inflammatory carcinoma; Paget’s disease; and other less common histologic types of breast cancer.

Infectious/Inflammatory Conditions

Mastitis

Usual etiologic agent: Staphylococcus aureus or Streptococcus spp.

Most commonly occurs during early weeks of breast-feeding.

Physical exam: Focal tenderness with erythema and warmth of overlying skin, fluctuant mass occasionally palpable.

Diagnosis: Ultrasound can be used to localize an abscess; if abscess is present, aspirate fluid for Gram stain and culture.

Treatment: Prevent engorgement of breast. Continue breast-feeding and recommend use of breast pump as an alternative.

Cellulitis: Wound care and antibiotics.

Abscess: Incision and drainage followed by antibiotics.

Hidradenitis Suppurativa

A chronic inflammatory condition of the accessory areolar glands of Montgomery; also affects the axilla.

Women with acne are predisposed to develop hidradenitis.

May mimic other diseases (Paget’s, invasive carcinoma).

Skin may be involved contiguously or multifocally.

Treatment: Antibiotics with incision and drainage.

Rarely, skin loss may require skin grafts.

Fat Necrosis

Presentation: Firm, irregular mass of varying tenderness.

History of local trauma elicited in 50% of patients.

Predisposing factors: Chest wall or breast trauma, previous radiation.
Physical exam: Irregular mass without discrete borders that may or may not be tender; later, collagenous scars predominate.

Often indistinguishable from carcinoma by clinical exam or mammography.

Diagnosis and treatment: Excisional biopsy with pathologic evaluation for carcinoma.

---

**Benign Disease**

See Table 17-2.

**Fibroadenoma**

- **Definition:** Fibrous stroma surrounds ductlike epithelium and forms a benign tumor that is grossly smooth, white, and well circumscribed.
- **Risk factors:** More common in black women than in white women. Most common breast lesion in adolescents and young females.
- **Incidence:** Typically occurs in late teens to early 30s; estrogen sensitive (increased tenderness during pregnancy).
- **Signs and symptoms:** Smooth, discrete, circular, mobile mass.
- **Diagnosis:** FNA.

---

**Table 17-2. A NDI Classification of Benign Breast Disorders**

<table>
<thead>
<tr>
<th>Normal</th>
<th>Disorder</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early reproductive years (age 15–25 years)</td>
<td>Lobular development</td>
<td>Fibroadenoma</td>
</tr>
<tr>
<td></td>
<td>Stromal development</td>
<td>Adolescent hypertrophy</td>
</tr>
<tr>
<td></td>
<td>Nipple eversion</td>
<td>Nipple inversion</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Later reproductive years (age 25–40 years)</td>
<td>Cyclical changes of menstruation</td>
<td>Cyclical mastalgia</td>
</tr>
<tr>
<td></td>
<td>Epithelial hyperplasia of pregnancy</td>
<td>Nodularity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bloody nipple discharge</td>
</tr>
<tr>
<td>Involvement (age 35–55 years)</td>
<td>Lobular involution</td>
<td>Macrocysts</td>
</tr>
<tr>
<td></td>
<td>Duct involution</td>
<td>Sclerosing lesions</td>
</tr>
<tr>
<td></td>
<td>Dilatation</td>
<td>Duct ectasia</td>
</tr>
<tr>
<td></td>
<td>Sclerosis</td>
<td>Nipple retraction</td>
</tr>
<tr>
<td></td>
<td>Epithelial turnover</td>
<td>Epithelial hyperplasia</td>
</tr>
</tbody>
</table>

ANDI = aberrations of normal development and involution.

**Treatment:**
- If FNA is diagnostic for fibroadenoma, may observe if asymptomatic and size < 2 cm. If > 2 cm, symptomatic offer surgical excision.
- If FNA is nondiagnostic or not synchronous with imaging, must excise mass.
- In patients > 40 years old, recommend excising mass to ensure diagnosis. The mass is well encapsulated and can be shelled out easily at surgery.

**Mondor's Disease**

**Definition:** Superficial thrombophlebitis of lateral thoracic or thoracoepigastric vein.

**Predisposing factors:** Local trauma, surgery, infection, repetitive movements of upper extremity.

**Presentation:** Acute pain in axilla or superior aspect of lateral breast.

**Physical exam:** Palpation of tender cord.

**Diagnosis:** Confirm with ultrasound.

**Treatment:**
- Clear diagnosis by ultrasound: Salicylates, warm compresses, limit motion of affected upper extremity. Usually resolves within 2–6 weeks.
- If persistent, surgery to divide the vein above and below the site of thrombosis or resect the affected segment.
- Ultrasound nondiagnostic or an associated mass present: Excisional biopsy.

**Fibrocystic Changes**

- Usually diagnosed in 20s–40s.
- **Presentation:** Breast swelling (often bilateral), tenderness, and/or pain.
- **Physical exam:** Discrete areas of nodularity within fibrous breast tissue.
- **Evaluation:** Serial physical examination with documentation of the fluctuating nature of the symptoms is usually sufficient unless a persistent discrete mass is identified; definitive diagnosis requires aspiration or biopsy with pathologic evaluation.
- Symptoms thought to be of hormonal etiology and tend to fluctuate with the menstrual cycle.
- Associated with a group of characteristic histologic findings, each of which has a variable relative risk for the development of cancer.
- Not associated with an increased risk for breast cancer unless biopsy reveals lobular or ductal hyperplasia with atypia.

**Treatment:**
- For cases with a classic history or absence of a persistent mass: Conservative management; options include nonsteroidal anti-inflammatory drugs (NSAIDs), oral contraceptive pills (OCPs), danazol, or tamoxifen; advise patient to avoid products that contain xanthine (e.g., caffeine, tobacco, cola drinks).
- If single dominant cyst, aspirate fluid; may discard if green or cloudy but must send to cytology and excise cyst if bloody.
- Atypical ductal or lobular hyperplasia: must be excised.

**Mammary Duct Ectasia**

**Definition:** Inflammation and dilation of mammary ducts.

- Most commonly occurs in the perimenopausal years.
- **Presentation:** Noncyclical breast pain with lumps under nipple/areola with or without a nipple discharge.
- **Exam:** Palpable lumps under areola, possible nipple discharge.
- **Diagnosis:** Based on exam; excision biopsy required to rule out cancer.
- **Treatment:** Excision of affected ducts.
Phyllodes Tumor (Cystosarcoma Phyllodes)

- A variant of fibroadenoma.
- Characterized as benign, intermediate, or malignant phyllodes. Malignant phyllodes are very rare (<10%).
- Patients tend to present later than those with fibroadenoma (>30 years).
- Characteristics: Indistinguishable from fibroadenoma by ultrasound or mammogram. The distinction between the two entities can be made on the basis of their histologic features (phyllodes tumors have more mitotic activity).
- Exam: Large, freely movable mass with overlying skin changes.
- Diagnosis: Definitive diagnosis requires biopsy with pathologic evaluation.
- Treatment:
  - Smaller tumors: Wide local excision with at least a 1-cm margin.
  - Larger tumors: Simple mastectomy.
  - No need for sentinel lymph node biopsy.

Intraductal Papilloma

- Definition: A benign local proliferation of ductal epithelial cells.
- Characteristics: Unilateral serosanguineous or bloody nipple discharge.
- Presentation: Subareolar mass and/or spontaneous nipple discharge.
- Evaluation: Radially compress breast to determine which lactiferous duct expresses fluid; mammography.
- Diagnosis: Definitive diagnosis by pathologic evaluation of resected specimen.
- Treatment: Excise affected duct.

Sclerosing Adenosis

- A sclerosing disorder causing palpable mass in breast.
- Mimics cancer on palpation, mammogram, and gross pathology.
- Excision and histologic exam is diagnostic.

Gynecomastia

- Definition: Development of female-like breast tissue in males.
- May be physiologic (neonatal, adolescent, and senescent) or pathologic.
  - Pathologies to consider: may be associated with medication use (spironolactone, cimetidine), inappropriately elevated hormonal levels (check prolactin, estrogen, testosterone). If hormones are elevated, evaluate further cause.
- At least 2 cm of excess subareolar breast tissue is required to make the diagnosis.
- Treatment: Treat underlying cause if specific cause identified; if normal physiology is responsible, only surgical excision (subareolar mastectomy) may be effective. Surgery is recommended only for progressive gynecomastia or for cosmetic reasons.

Nipple Discharge

- Most nipple discharge is benign
- Evaluation includes history, breast examination, mammography, +/- ultrasound
- Worrisome characteristics: unilateral, spontaneous, serous
Green discharge: if cyclical and nonspontaneous, likely due to fibrocystic breast
Bloody discharge: if unilateral, most likely due to intraductal papilloma
Bilateral discharge: if milky, consider intracranial etiology, obtain prolactin level

Premalignant Disease
See Table 17-3.

Malignant Tumors

infiltrating ductal carcinoma
- Most common invasive breast cancer (80% of cases).
- Most common in perimenopausal and postmenopausal women.
- Ductal cells invade stroma in various histologic forms described as scirrhous, medullary, comedo, colloid, papillary, or tubular.
- Metastatic to axilla, bones, lungs, liver, brain.

infiltrating lobular carcinoma
- Second most common type of invasive breast cancer (10% of cases).
- Originates from terminal duct cells and, like LCIS, has a high likelihood of being bilateral.
- Presents as an ill-defined thickening of the breast.
- Like LCIS, lacks microcalcifications and is often multicentric.
- Tends to metastasize to the axilla, meninges, and serosal surfaces.

Paget’s Disease (of the nipple)
- Two percent of all invasive breast cancers.
- Usually associated with underlying LCIS or ductal carcinoma extending within the epithelium of main excretory ducts to skin of nipple and areola.
- Presentation: Tender, itchy nipple with or without a bloody discharge with or without a subareolar palpable mass.
- Diagnosis: Biopsy shows Paget’s cells.
- Treatment: Usually requires a modified radical mastectomy.

inflammatory carcinoma
- Two to 3% of all invasive breast cancers.
- Most lethal breast cancer.
- Frequently presents as erythema, “peau d’orange,” and nipple retraction.
- Inflammatory picture is due to the blockage of the efferent lymphatic ducts causing edema—“peau d’orange.”
- Diagnosis: Skin biopsy shows dermal lymphatic invasion seen at pathologic evaluation.
- Treatment: Consists of chemotherapy followed by surgery and/or radiation, depending on response to chemotherapy.

Causes of gynecomastia:
- Increased estrogen (tumors, endocrine disorders, liver failure, obesity).
- Decreased testosterone (aging, primary or secondary testicular failure, Klinefelter’s, renal failure).
- Drugs (e.g., spironolactone).

Twenty percent of infiltrating lobular breast carcinoma have simultaneous contralateral breast cancer.
**Table 17-3. Premalignant Disease**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Disease</th>
<th>Lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell of origin</td>
<td>Inner layer of epithelial cells in major ducts</td>
<td>Cells of terminal duct–lobular unit</td>
</tr>
<tr>
<td>Definition</td>
<td>Proliferation of ductal cells that spread through the ductal system but lack the ability to invade the basement membrane</td>
<td>A multifocal proliferation of acinar and terminal ductal cells. Is NOT precancerous in and of itself</td>
</tr>
<tr>
<td>Age and sex</td>
<td>More than half of cases occur after menopause 5% of male cancer</td>
<td>Vast majority of cases occur prior to menopause Never seen in males</td>
</tr>
<tr>
<td>Palpable mass</td>
<td>Sometimes</td>
<td>Never</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Clustered microcalcifications on mammogram, malignant epithelial cells in breast duct on biopsy</td>
<td>Typically a clinically occult lesion; undetectable by mammogram and incidental on biopsy</td>
</tr>
<tr>
<td>Lymphatic invasion</td>
<td>&lt; 1%</td>
<td>Rare</td>
</tr>
<tr>
<td>Risk of invasive</td>
<td>Considered the anatomic precursor of breast carcinoma. Increased risk in ipsilateral breast, usually same quadrant; infiltrating ductal carcinoma most common histologic type; comedo type has the worst prognosis</td>
<td>Considered as a marker of breast carcinoma. Equally increased risk in either breast, infiltrating ductal carcinoma also most common histologic type (counterintuitive); associated with simultaneous LCIS in the contralateral breast in over half of cases</td>
</tr>
<tr>
<td>Treatment</td>
<td>If small (&lt; 2 cm): Lumpectomy with either close follow-up or radiation  If large (&gt; 2 cm): Lumpectomy with 1-cm margins and radiation  If breast diffusely involved: Simple mastectomy</td>
<td>None; bilateral mastectomy an option if patient is high risk</td>
</tr>
</tbody>
</table>

---

**Breast Cancer**

### Epidemiology

- One in eight women will develop breast cancer in their lifetime.
- Second most common cause of cancer death among women overall (lung cancer is number 1).
- Incidence increases with increasing age.
- One percent of breast cancers occur in men.

---

**EXAM TIP**

**Typical scenario:** A 65-year-old female presents with a pruritic, scaly rash of her nipple–areolar complex and a bloody nipple discharge. Think: Paget’s disease. Biopsy and pathologic exam required to confirm diagnosis.

---

**WARD TIP**

Greater than 75% of patients have axillary metastases at time of diagnosis of inflammatory breast carcinoma, and distant metastases are common.

---

**WARD TIP**

Paget’s disease of vagina is a similar disease, presenting as a scaly, pruritic rash of the vagina.

---

**EXAM TIP**

**Typical scenario:** A 45-year-old female presents with enlargement of her left breast with nipple retraction, erythema, warmth, and induration. Think: Inflammatory breast carcinoma.

---

**WARD TIP**

Fibrocystic changes of the breast alone are not a risk factor for breast cancer.


**Risk Factors**

- Any change that causes increased exposure to estrogen without the protective effects of progesterone.
- Early menarche (<12).
- Late menopause (>55).
- Nulliparity or first pregnancy >30 years.
- White race.
- Old age.
- History of breast cancer in mother or sister (especially if bilateral or premenopausal).
- Genetic predisposition (BRCA1 or BRCA2 positive, Li–Fraumeni syndrome).
- Prior personal history of breast cancer.
- Previous breast biopsy.
- DCIS or LCIS.
- Atypical ductal or lobular hyperplasia.
- Postmenopausal estrogen replacement (unopposed by progesterone).
- Radiation exposure.

**Genetic predisposition**

- Five to 10% of breast cancers are associated with an inherited mutation.
- Both BRCA1 and BRCA2 function as tumor-suppressor genes, and for each gene, loss of both alleles is required for the initiation of cancer.
- BRCA1 and BRCA2 both are inherited in an autosomal dominant fashion with varying penetrance.
- BRCA1: On 17q, also associated with ovarian cancer. Sixty percent lifetime risk of breast cancer.
- BRCA2: On chromosome 13q, also associated with male breast cancer. Sixty percent lifetime risk of breast cancer.
- Somatic mutation of p53 in 50% and of Rb in 20% of breast cancers.

**Screening recommendations (from the American Cancer Society)**

See Table 17-4. Screening reduces mortality by 30–40%.

---

**Table 17-4. American Cancer society recommendations for Breast Cancer Detection in a Symptomatic Woman**

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Examination</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>20–39</td>
<td>Breast self-examination</td>
<td>Monthly</td>
</tr>
<tr>
<td></td>
<td>Clinical breast examination</td>
<td>Every 3 years</td>
</tr>
<tr>
<td>40 and older</td>
<td>Breast self-examination</td>
<td>Monthly</td>
</tr>
<tr>
<td></td>
<td>Clinical breast examination</td>
<td>Annually</td>
</tr>
<tr>
<td></td>
<td>Mammography</td>
<td>Annually</td>
</tr>
</tbody>
</table>

Diagnostic Options

- **Mammography:** See Figure 17-6.
  - Identifies 5 cancers/1,000 women.
  - Sensitivity 85–90%.
  - False positive 10%, false negative 6–8%.
  - If cancer is first detected by mammogram, 80% have negative lymph nodes (vs. 45% when detected clinically). Mammography picks up early disease and hence is a good screening test.
  - Suspicious finding: Stellate, speculated mass with associated microcalcifications.

- **Reporting mammogram results (Breast Imaging Reporting and Data System—BIRADS):**
  0: Assessment incomplete
  1: Negative
  2: Benign finding
  3: Probably benign finding
  4: Suspicious for cancer
  5: Highly suspicious for cancer
  6: Known biopsy-proven cancer, treatment pending

- **Ultrasound:**
  - (+ +) No ionizing radiation.
  - (+) Good for identifying cystic disease and can also assist in therapeutic aspiration.
  - (+) Results easily reproducible.
  - (–) Resolution inferior to mammogram. Used as adjunct if concerning mass is found on mammography.
  - (–) Will not identify lesions < 1 cm.

- **FNA (aspiration of tumor cells with small-gauge needle):**
  - Advantages: Low morbidity, cheap, only 1–2% false-positive rate.
  - Disadvantages: False-negative rate up to 10%, requires a skilled pathologist.

- **Needle localization biopsy:** Locates occult cancer in > 90%.

**WARD TIP**

Start yearly mammograms 10 years before the age at which first-degree relative was diagnosed with breast cancer.

**WARD TIP**

Mammography is more useful if age > 30 because the large proportion of fibrous tissue (‘dense tissue’) in younger women’s breasts make mammograms more difficult to interpret.

**WARD TIP**

Benign cysts should not be bloody. A bloody aspirate usually indicates malignancy.

**WARD TIP**

Five to 10% of palpable masses have a negative mammogram.

Core biopsy: Chance of sampling error.

Stereotactic core biopsy:
- Fewer complications compared to needle localization biopsy.
- Less chance of sampling error than core biopsy alone.
- No breast deformity.

Treatment decisions

See Table 17-5.

Early Invasive Breast Cancer (Stages I and II)
- For early stages (I and II): Currently, mastectomy with axillary sentinel lymph node biopsy (which evaluates the status of axillary lymph nodes) and breast conservation (lumpectomy, sentinel lymph node biopsy, and adjuvant radiation therapy) are considered equivalent treatments for stage I and II breast cancer (see Table 17-6).
- Adjuvant chemotherapy for early invasive breast cancer is considered for all node-positive cancers, all cancers that are larger than 1 cm in size, and node-negative cancers larger than 0.5 cm in size when blood vessel or lymph vessel invasion, high nuclear grade, high histologic grade, HER2/neu overexpression, and negative hormone receptor status is present (see Table 17-7).
- Tamoxifen (estrogen blocker) therapy is considered for hormone receptor-positive women with cancers that are larger than 1 cm in size.

Locally Advanced Breast Cancer (Stage III)
See Figure 17-7.
- If proceeding with surgery first, would expect mastectomy with axillary lymph node dissection for pathologically confirmed nodal disease.
- Consideration can be made for neoadjuvant chemotherapy, for assessment of tumor responsiveness or to shrink tumor for ease of surgery. At time of surgery, still expect to proceed with axillary lymph node dissection.

Advanced Breast Cancer (Stage IV)
- Systemic and palliative treatment.
- Some patients need mastectomy for palliation and relief from foul discharge and pain.

Types of operations

- Simple mastectomy: Removal of all breast tissue.
- Modified radical mastectomy: Resection of all breast tissue and axillary nodes (level I).
- Radical mastectomy: Resection of all breast tissue, axillary nodes, and pectoralis major and minor muscles (rarely performed nowadays due to increased morbidity without advantage).
- Breast-conserving surgery:
  - Lumpectomy and axillary node dissection: Resection of mass with rim of normal tissue and axillary node dissection—good cosmetic result. Axillary dissection is generally carried out in levels I and II. Zone III is explored only if nodes are palpable.
  - Quadrantectomy and axillary radiotherapy (QUART).
- Breast conservation is considered for all patients with stage I or II cancer because of the important cosmetic advantages. Relative contraindications to breast-conservation therapy include:
  - Prior radiation therapy to the breast or chest wall.
  - Involved surgical margins or unknown margin status following reexcision.

WARD TIP

Prognosis depends more on stage than on histologic type of breast cancer. Node positivity is the most important factor.

WARD TIP

Lumpectomy with sentinel node biopsy and postoperative radiation is a viable treatment option only in stages I and II. Mastectomy is not required and has no additional survival benefit.
### Table 17-5. TNM Staging for Breast Cancer

#### Primary Tumor (T)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>T0</td>
<td>No evidence of tumor</td>
</tr>
<tr>
<td>Tis</td>
<td>Carcinoma in situ</td>
</tr>
<tr>
<td>DCIS</td>
<td>Ductal carcinoma in situ</td>
</tr>
<tr>
<td>LCIS</td>
<td>Lobular carcinoma in situ</td>
</tr>
<tr>
<td>T1</td>
<td>&lt; 2 cm</td>
</tr>
<tr>
<td>T2</td>
<td>&gt; 2 cm but &lt; 5 cm</td>
</tr>
<tr>
<td>T3</td>
<td>&gt; 5 cm</td>
</tr>
<tr>
<td>T4</td>
<td>Any size tumor with direct extension to the chest wall and/or to the skin (including inflammatory breast cancer or &quot;peu d’orange&quot;)</td>
</tr>
</tbody>
</table>

#### Clinical Nodes (N)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nx</td>
<td>Regional lymph nodes cannot be assessed</td>
</tr>
<tr>
<td>N0</td>
<td>No regional lymph node metastasis</td>
</tr>
<tr>
<td>N1</td>
<td>Metastasis to movable ipsilateral level I, II axillary lymph nodes</td>
</tr>
<tr>
<td>N2</td>
<td>Metastasis in ipsilateral level I, II axillary lymph nodes that are clinically matted or fixed</td>
</tr>
<tr>
<td>N3</td>
<td>Metastasis in ipsilateral infraclavicular or supraclavicular lymph nodes with or without level I, II involvement; or in clinically detected ipsilateral internal mammary lymph nodes with clinically evident level I, II axillary lymph nodes</td>
</tr>
</tbody>
</table>

#### Distant Metastasis (M)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>M0</td>
<td>No clinical or radiographic evidence of distant metastasis</td>
</tr>
<tr>
<td>M1</td>
<td>Distant detectable metastasis as determined by classic clinical and radiographic means and/or histologically proven larger than 0.2 mm</td>
</tr>
</tbody>
</table>

#### Anatomic Staging

<table>
<thead>
<tr>
<th>Stage</th>
<th>T</th>
<th>N</th>
<th>M</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0</td>
<td>Tis</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>Stage I A</td>
<td>T1</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>Stage I B</td>
<td>T0</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T1</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td>Stage II A</td>
<td>T0</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T1</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>Stage II B</td>
<td>T2</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td>Stage III A</td>
<td>T0</td>
<td>N2</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T1</td>
<td>N2</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>N2</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>N2</td>
<td>M0</td>
</tr>
<tr>
<td>Stage III B</td>
<td>T4</td>
<td>N0</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T4</td>
<td>N1</td>
<td>M0</td>
</tr>
<tr>
<td></td>
<td>T4</td>
<td>N2</td>
<td>M0</td>
</tr>
<tr>
<td>Stage III C</td>
<td>Any T</td>
<td>N3</td>
<td>M0</td>
</tr>
<tr>
<td>Stage IV</td>
<td>Any T</td>
<td>Any N</td>
<td>M1</td>
</tr>
</tbody>
</table>
Table 17-6. **Staging System for Breast Cancer**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0</td>
<td>DCIS or LCIS</td>
</tr>
<tr>
<td>Stage I</td>
<td>Invasive carcinoma ≤ 2 cm in size (including carcinoma in situ with micro invasion) without nodal involvement and no distant metastases</td>
</tr>
<tr>
<td>Stage II</td>
<td>Invasive carcinoma ≤ 5 cm in size with involved but movable axillary nodes and no distant metastases, or a tumor &gt; 5 cm without nodal involvement or distant metastases</td>
</tr>
<tr>
<td>Stage III</td>
<td>Breast cancers &gt; 5 cm in size with nodal involvement; or any breast cancer with fixed axillary nodes; or any breast cancer with involvement of the ipsilateral internal mammary lymph nodes; or any breast cancer with skin involvement, pectoral and chest wall fixation, edema, or clinical inflammatory carcinoma, if distant metastases are absent</td>
</tr>
<tr>
<td>Stage IV</td>
<td>Any form of breast cancer with distant metastases</td>
</tr>
</tbody>
</table>

Table 17-7. **Hormone Receptor Status Response to Therapy**

<table>
<thead>
<tr>
<th>Hormone Receptor Status</th>
<th>Response to Hormonal Therapy (Estrogen Blockers or Anti-Male Inhibitors)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ER+/PR+</td>
<td>80%</td>
</tr>
<tr>
<td>ER-/PR+</td>
<td>45%</td>
</tr>
<tr>
<td>ER+/PR-</td>
<td>35%</td>
</tr>
<tr>
<td>ER/PR-</td>
<td>10%</td>
</tr>
</tbody>
</table>

- Multicentric disease.
- Scleroderma or other connective-tissue disease.
- Pregnancy

**Sentinel node biopsy:** Recently developed alternative to complete axillary node dissection:
- Done only if there are no palpable nodes.
- Based on the principle that metastatic tumor cells migrate in an orderly fashion to first draining lymph node(s).
- Lymph nodes are identified on preoperative nuclear scintigraphy and blue dye is injected in the periareolar area.
- Axilla is opened and inspected for blue and/or “hot” nodes identified by a gamma probe.
- When sentinel node(s) is positive, an axillary dissection is completed.
- When sentinel node(s) is negative, an axillary dissection is not performed unless axillary lymphadenopathy is identified.

**Hormonal therapy: tamoxifen**

- Selective estrogen receptor modulator (SERM) that blocks the uptake of estrogen by target tissues.
- Side effects will simulate menopause: Hot flashes, irregular menses, thromboembolism and increased risk for endometrial cancer due to selective hormone agonist action.
- Therapy of choice for postmenopausal women with positive receptors. Survival benefit for pre- and postmenopausal women, but benefit greater for ER-positive patients.
- May get additional benefit by combining tamoxifen with chemotherapy.

**Chemotherapy**

- Any number of regimens are acceptable. Adriamycin, 5-fluorouracil, methotrexate, cyclophosphamide, and paclitaxel (Taxol) are some of the agents used.
- Herceptin is used for HER2/neu-positive patients.

**Reconstruction**

- Done in patients after mastectomy.
- Can use either prosthetic or autologous implants. May also use an expander and implant at a later date.
- Prosthetic implant may be either saline or silicone based.
- Autologous tissue can be either rectus muscle or latissimus dorsi muscle myocutaneous flap.

**Recurrence**

- Five to 10% local recurrence at 10 years.
- Metastases in < 10% of cases.
- Local chest wall recurrence most common within 2–3 years, if at all.

---

WARD TIP

Raloxifene is another SERM that has been used for breast cancer prevention. It does not increase uterine cancer risk.
Median survival 2 years.
Palliative therapy indicated.

The 5-year survival rate is:
- Stage I: 94%
- Stage II: 70–85%
- Stage III: 48–52%
- Stage IV: 18%

Breast cancer in pregnant and lactating Women
- Three breast cancers are diagnosed per 10,000 pregnancies.
- A fine-needle aspiration should be performed. If it identifies a solid mass, then it should be followed by biopsy.
- Mammography is possible as long as proper shielding is used.
- Radiation is not advisable for the pregnant woman. Thus, for stage I or II cancer, a modified radical mastectomy should be done rather than a lumpectomy with axillary node dissection and postoperative radiation. If diagnosed in the third trimester, only then can lumpectomy and adjuvant radiation be considered. Radiation would be given postpartum.
- If lymph nodes are positive, delay chemotherapy until the second trimester.

Breast cancer in males
- Predisposing factors: Klinefelter’s syndrome, estrogen therapy, elevated endogenous estrogen, previous irradiation, and trauma.
- Infiltrating ductal carcinoma most common histologic type (men lack breast lobules).
- Diagnosis tends to be late, when the patient presents with a mass, nipple retraction, and skin changes.
- Stage by stage, survival is the same as it is in women. However, more men are diagnosed at a later stage.
- Treatment for early-stage cancer involves a modified radical mastectomy and postoperative radiation.
- Sentinel node biopsy and estrogen receptor status may help to stage disease.
# Burns

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<td>Increase in Fluid Requirements</td>
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<td>Fasciectomy</td>
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<tr>
<td>Debridement And Skin Grafting</td>
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<td>Infection</td>
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<td>Treatment</td>
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<td>Inhalational Injury and Carbon Monoxide Poisoning</td>
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<td>Diagnosis</td>
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<td>Treatment</td>
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<td>Electrical Injury</td>
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<td>Definition</td>
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<td>Mechanism</td>
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<td>Signs And Symptoms</td>
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<td>Treatment</td>
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<td>Late Complications</td>
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<tr>
<td>Chemical Injury</td>
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<td>Mechanism</td>
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<td>Severity</td>
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<td>Priorities in Treatment</td>
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<td>Nutrition of Burn Patients</td>
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<td>Burn Scar Cancer</td>
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<tr>
<td>Operations</td>
<td>287</td>
</tr>
<tr>
<td>Esch Arotomy</td>
<td>287</td>
</tr>
<tr>
<td>Fasciectomy</td>
<td>288</td>
</tr>
<tr>
<td>Debridement And Skin Grafting</td>
<td>288</td>
</tr>
<tr>
<td>Infection</td>
<td>288</td>
</tr>
<tr>
<td>Treatment</td>
<td>288</td>
</tr>
</tbody>
</table>
Incidence of burns is higher during winter months.

Seventy-five percent of burn-related deaths can be attributed to house fires.

Typical scenario: A 4-year-old child is brought to your emergency department (ED) with a 14% total body surface area (TBSA) burn, including both second and third degree. It looks as if he had been seated in scalding water. Where should this child be cared for? Think: This patient is under 10 years old, with > 10% TBSA burned, including the perineum and genitalia, and may require special social assistance (due to likelihood of child abuse). He should be transferred to a burn center.

Treatment Outline

1. Airway, breathing, circulation (ABC); intubate patient if indicated. Start fluid resuscitation with 1 L lactated Ringer's (LR) solution bolus in adults, and 20 mL/kg for children in the ED.
2. If carbon monoxide inhalation is suspected, administer 100% oxygen by nonrebreather mask (accelerates dissociation).
3. Assess area of burn (second and third degree).
4. All burn patients need good analgesia (IV morphine). Apply cold saline soaks for analgesia if burns are < 25% BSA (watch for hypothermia).
5. Cover burns with silver sulfadiazine/clean sheet and then warm blanket.
6. Elevate burned areas when possible to minimize edema.
7. Basic laboratories: Arterial blood gas (ABG), complete blood count (CBC), electrolytes, carboxyhemoglobin.
8. Weigh patient.
9. Continue fluid resuscitation as per the Parkland formula.
10. Insert Foley catheter in patients requiring fluid resuscitation or with significant perineal burns.
11. Electrocardiogram (ECG).

Patients with high-voltage electrical injury require cardiac monitor, as do any intubated or otherwise unstable patients.

Assessment of Extent and severity of Burns

- Consider only second- and third-degree burns when stating %BSA burned.
- The Rule of Nines may be used to estimate the area burned (see Figure 18-1).
- The palm of the patient’s hand is roughly equivalent to 1% BSA.
Percent BSA burned is used to determine need for fluid resuscitation (see below).

Patients not requiring admission to any hospital should receive appropriate management and follow-up, including cleansing of burned areas; debridement of loose, nonviable skin; application of topical antimicrobial/nonadherent dressing or of biologic dressing (described below).

Assess need for escharotomy or fasciotomy (see below).

### Deciding Who May Be Treated as an Outpatient

- Most first-degree burns.
- Superficial and intermediate second-degree burns of < 10% BSA (excluding most burns of face, eyes, hands, perineum).
- Patients with acceptable social situations amenable to providing a safe and helpful environment at home.

## Fluid Resuscitation

### Adults

#### Parkland Formula

For first 24 hours:

- LR at rate of 4 mL/kg/%BSA burn.
- Give half of 24-hour requirement in first 8 hours from the time of burn, and the remainder over the next 16 hours.

### Pediatric Patients

#### Galveston Formula

\[
5,000 \text{ mL/m}^2 \text{ burned area} + 1,500 \text{ mL/m}^2 \text{ total area}
\]

**monitoring fluid status**

- Resuscitation is adequate when urine output is 30–50 cc/hr in adults and 1 cc/kg/hr in children < 30 kg.

**EXAM TIP**

*Typical scenario:* An adult male is brought to the ED with second-degree burns on his chest and abdominal wall, anterior right leg, and perineum. What percentage TBSA does he have? Think: Rule of Nines says 18% for anterior torso, 9% for anterior leg, and 1% for perineum = 28%
- Adjust fluids when urine output is more than 33% different (in either direction) from recommended over 2–3 hours.
- Monitor daily weights.

**Increase in fluid requirements**
- High-voltage electrical injury
- Inhalational injury
- Delayed resuscitation
- Intoxicated at time of injury

### Burn Care

**Daily**
- ABC, physiologic resuscitation.
- Cleanse wounds, trim nonviable skin.
- Topical antimicrobial agents for burn wound care (see Table 18-1).
- Daily burn care in shower or tank if possible; otherwise, at bedside.

#### TABLE 18-1. Topical Antimicrobial Agents for Burn Wound Care

<table>
<thead>
<tr>
<th>Silver Nitrate</th>
<th>Mafenide Acetate</th>
<th>Silver Sulfadiazine</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Active component</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.5% in aqueous solution</td>
<td>11.1% in water-miscible base</td>
<td>1.0% in water-miscible base</td>
</tr>
<tr>
<td><strong>Spectrum of antimicrobial activity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gram-negative—good</td>
<td>Gram-negative—good</td>
<td>Gram-negative—variable</td>
</tr>
<tr>
<td>Gram-positive—good</td>
<td>Gram-positive—good</td>
<td>Gram-positive—good</td>
</tr>
<tr>
<td>Yeast—good</td>
<td>Yeast—poor</td>
<td>Yeast—good</td>
</tr>
<tr>
<td><strong>Method of wound care</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occlusive dressings</td>
<td>Exposure</td>
<td>Exposure or single-layer dressings</td>
</tr>
</tbody>
</table>

**Advantages**
- Painless
- No hypersensitivity reaction
- No gram-negative resistance
- Dressings reduce evaporative heat loss
- Greater effectiveness against yeasts
- Penetrates eschar
- Wound appearance readily monitored
- Joint motion unrestricted
- No gram-negative resistance
- Painless
- Wound appearance readily monitored
- When exposure method used
- Easily applied
- Joint motion unrestricted when exposure method used
- Greater effectiveness against yeasts

**Disadvantages**
- Deficits of sodium, potassium, calcium, and chloride
- No eschar penetration
- Limitation of joint motion by dressings
- Methemoglobinemia—rare
- Argyria—rare
- Staining of environment and equipment
- Painful on partial-thickness burns
- Susceptibility to acidosis as a result of carbonic anhydrase inhibition
- Hypersensitivity reactions in 7% of patients
- Neutropenia and thrombocytopenia
- Hypersensitivity—infrequent
- Limited eschar penetration

For definitive nonexcisional therapy, continue daily cleansing and debridement until granulation tissue is present with minimal or no necrotic debris, at which time a biologic dressing is applied. For operative therapy, see Operations section.

EXAM TIP
Hypovolemia and hemocoagulation can lead to an elevated hematocrit and decreased left ventricular end-diastolic volume that result in decreased cardiac output and low-flow state.

EXAM TIP
Burn patients are susceptible to Curling's ulcer, which is due to lack of the normal mucosal barrier.

Operations

Escharotomy

- Areas of concern: Circumferential burns of extremities (including penis) or thorax (see Figure 18-2).
- Indications:
  - Impairment of circulation in extremities
  - Impaired ventilation
  - No anesthesia is needed.

**Figure 18-2. Locations for escharotomies.** The incisions are placed along the mid-medial lines of the extremities and the thorax (dashed lines). The skin is especially tight along major joints, and decompression at these sites must be complete (solid lines). Neck and digital escharotomies are rarely necessary. (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz’s Principles of Surgery, 8th ed. New York: McGraw-Hill, 2004: 194).
Escharotomy may fail, especially when the burn is from high-voltage electrical injury or is associated with soft tissue, bone, or vascular injury. If compartment syndrome persists after escharotomy, incision of the fascia is also required. General anesthesia is required.

**Debridement and Skin Grafting**

- Excisional treatment is indicated for most deep second- and third-degree burns once the patient is stabilized. 
  - Advantages: Decreased length of stay, earlier return to work, decreased incidence of infection, decreased complications, improved survival.
  - Technique:
    - Full-thickness burns require debridement to the investing fascial layer using the scalpel and bovie.
    - Tangential excision may be used for deep partial burns of < 20% BSA and for staged excision of more extensive partial- and full-thickness burns.
    - A Goulian knife, which takes off sequential thin layers, can be used for partial- and some full-thickness excisions; requires debridement until uniform capillary bleeding.
    - Once debridement is complete, the wound is covered with split-thickness skin graft (STSG), full-thickness graft, or biologic dressing.
    - Wound closure.
    - STSG may be applied when the burn is excised or there is no residual nonviable tissue, no pooled secretions, and surface bacterial count is < 10^5/cm^2.
    - Autograft should be 0.010–0.015 inches thick:
      - Donor sites may be reharvested (after 2–3 weeks when reepithelialization is complete), but the quality decreases each time as dermis is thinner.
      - Grafts may be meshed at a ratio of 1.5:1 to increase coverage, unless burns are on face or joints.
      - If the risk of mortality is anticipated to be < 50%, first graft hands, feet, face, and joints. If > 50%, graft fat surfaces first to decrease uncovered surface area.
      - Biologic dressings: Bilaminate; outer layer with pores to permit water vapor but not liquid or bacterial passage, and inner layer that permits ingrowth of fibrovascular tissue from wound surface.

**Infection**

- Infection of a burn wound causes an increase in the depth (thus converts a second-degree burn to a third-degree).
- Biopsy of the wound is the most definitive way to diagnose burn wound infection.
- Burn patients are very prone to pneumonia and catheter-related infections.

**Treatment**

- For invasive infection, use topical antimicrobials (see Table 18-1) and start systemic antibiotics.
For pseudomonal or pediatric infections, infuse subeschar piperacillin, and plan for emergent operative debridement within 12 hours.

For candidal infections, start antifungal creams; if that treatment fails, start systemic therapy with amphotericin B.

**Inhalational Injury and Carbon Monoxide Poisoning**

- In closed space burns.
- Carbon monoxide impairs tissue oxygenation by decreasing oxygen-carrying capacity of blood, shifting oxygen-hemoglobin dissociation curve to the left, binding myoglobin and terminal cytochrome oxidase.
- Symptoms do not correlate well with carboxyhemoglobin levels, but levels up to 10% are typically asymptomatic.

**signs And symptoms**

Patients may have hoarse voice, cough, wheeze, bronchorrhea, hypoxemia, carbonaceous sputum, head and neck burns, singed nose hairs.

**diagnosis**

- Often delayed but is made with the use of chest x-ray (CXR), bronchoscopy, CT scan, and ventilation-perfusion (V/Q) scan.
- Measure carboxyhemoglobin with ABG.
- Bronchoscopy reveals edema and ulceration.
- V/Q scanning will demonstrate carbon particle deposition on endobronchial mucosa.

**treatment**

- Half-life of carbon monoxide decreased from 4 hours on room air to 45–60 minutes with 100% oxygen and 15–20 minutes at 3 atmospheric pressure (hyperbaric oxygen).
- Mild injury: Use warm humidified oxygen and an incentive spirometer.
- Moderate: Repeated bronchoscopy when there is continued mucosal sloughing and the patient is unable to clear it.
- Severe, with progressive hypoxemia: Intubation.
  - Nebulized bronchodilators and N-acetylcysteine are useful adjunct treatments.

**complications**

Most commonly, pneumonia.

**Electrical Injury**

**definition**

- Extent of injury depends on voltage of current:
  - Up to 1,000 volts—increased resistance limits further passage of current and heating of tissue.

**WARD TIP**

Infection is more likely in patients with > 30% BSA burn without complete excision or grafting. Apparently infected wounds need to be examined daily and biopsied, including eschar and underlying unburned tissue.

**EXAM TIP**

Patients with evidence of inhalation injury (soot around nares or mouth) should be intubated.

**WARD TIP**

Mortality of burns with inhalational injury is increased compared to burns without inhalational injury.

**EXAM TIP**

Carbon monoxide is a colorless, tasteless, odorless gas and has an affinity for hemoglobin 200 times that of oxygen.
More than 1,000 volts—passage of current is not limited and tissue injury can continue.
- Deeper tissue may be more severely injured because it cools more slowly.

**Mechanism**

- Tissue damaged via conversion to thermal energy.
- Damage occurs in skin and underlying tissues along the course of current.
- Skin at the point of contact is often severely charred.

**Signs and Symptoms**

- Charring at point of contact.
- Myoglobinuria (with muscle damage).
- Hyperkalemia (due to tissue necrosis).
- High-voltage or lightning injury may cause cardiac arrest.
- Neuropathy (immediately following injury, likely to resolve over time).
- Compartment syndrome: Swelling of injured extremity with pain, paresthesia, pallor, pulselessness, poikilothermia.

**Treatment**

- Patients with loss of consciousness or abnormal ECG require cardiac monitoring for 48 hours even if there are no further arrhythmias or ECG changes.
- Electrical injury is more likely than other types of burn injury to necessitate fasciotomy because deep tissue edema may be extensive, causing compartment syndrome.

**Late Complications**

- Delayed hemorrhage because of inadequate wound exploration, debridement, or exposure of vessel.
- Cataracts.

**Chemical Injury**

**Mechanism**

- General: Protein denaturation and precipitation, with release of thermal energy.
- Liquefaction necrosis (alkali).
- Delipidation (petroleum products).
- Vesicle formation (vesicant gas).

**Severity**

Determined by:

- Concentration
- Amount of agent in contact.
- Duration of contact (tissue injury continues to occur as long as the agent remains in contact with the skin).
Priorities in Treatment

- Remove all clothing to prevent further contact.
- Copious water lavage: Irrigation should continue for at least 30 minutes for acid burns, and longer for alkali burns (because they penetrate deeper into the tissue).
- Check pulmonary status (for edema, mucosal desquamation, bronchospasm).

Nutrition of Burn Patients

- Enteral route preferred (total parenteral nutrition only if enteral is not possible).
- Early feeding recommended.
- High calorie (30–35 kcal/kg/day) and high protein (1.5–2 g/kg/day) requirements.

Burn Scar Cancer

- Rare long-term complication of burn scars (can occur in scars of any origin).
- Called Marjolin’s ulcer.
- Usually squamous cell carcinoma, which metastasizes via lymph nodes.
- Diagnosis made by biopsy.
- **Treatment:** Wide excision.

WARD TIP
Chemical wound irrigation should be with water only. Attempts to neutralize the agent may cause further release of heat, thereby causing further damage.
## Section iii

### High-Yield Facts For Surgical electives

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<thead>
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Malignant Hyperthermia

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General Anesthesia

**Definition**
- Drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation.
- Ability to independently maintain ventilatory function is often impaired; therefore, airway assistance by way of intubation, laryngeal mask airway, or other adjunct is usually necessary.

**Stages of Anesthesia**
Anesthetic drug effects can be divided into four stages of increasing depth of central nervous system (CNS) depression:
- Stage I: Analgesia stage
- Stage II: Excitement stage
- Stage III: Surgical anesthesia stage
- Stage IV: Medullary depression stage

**Asa Classification of Surgical Patients**
See Table 19-1.

**Induction**
Induction anesthetics can be both IV and inhaled. See Table 19-2.

**Neuromuscular Blockade**
- It is important to realize that muscle relaxation does not ensure unconsciousness, amnesia, or analgesia.
- Postsynaptic receptors: Located in the neuromuscular junction, they are the site of action of neuromuscular blockers, blocking acetylcholine (ACh) transmission.

**Table 19-1. asa Physical Status Classification System and Expected Mortality**

<table>
<thead>
<tr>
<th>ASA Classification</th>
<th>Description</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>A normal healthy patient</td>
<td>0.1%</td>
</tr>
<tr>
<td>P2</td>
<td>A patient with mild systemic disease</td>
<td>0.2%</td>
</tr>
<tr>
<td>P3</td>
<td>A patient with severe systemic disease</td>
<td>1.8%</td>
</tr>
<tr>
<td>P4</td>
<td>A patient with severe systemic disease that is a constant threat to life</td>
<td>7.8%</td>
</tr>
<tr>
<td>P5</td>
<td>A moribund patient who is not expected to survive without the operation</td>
<td>9.4%</td>
</tr>
<tr>
<td>P6</td>
<td>A declared brain-dead patient whose organs are being removed for donor purposes</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Effect</th>
<th>Monitor</th>
<th>iv Drugs</th>
<th>Potent gases</th>
<th>Weak gas</th>
<th>Local Anesthetics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unconscious (amnesia) (anxiolysis)</td>
<td>EEG and/or clinical signs</td>
<td>Benzodiazepines, Midazolam, Lorazepam, Diazepam, Barbital, Etomidate, Ketamine</td>
<td>Sevoflurane, Desflurane, Isoflurane, Enflurane, Halothane</td>
<td>Nitrous oxide</td>
<td>Amides, Ethers</td>
</tr>
<tr>
<td>Analgesia</td>
<td>Heart rate, Blood pressure, Respiratory rate</td>
<td>Opioids, Fentanyl, Morphine, Hydromorphone, Nonopioid, Ketamine, Parecoxib, Dexmedetomidine, Acetaminophen IV</td>
<td>Sevoflurane, Desflurane, Isoflurane, Enflurane, Halothane</td>
<td>Nitrous oxide</td>
<td>Amides, Ethers, Cocaine, Procaine, Chloroprocaine, Tetracaine, Benzocaine</td>
</tr>
<tr>
<td>Muscle relaxation (paralysis)</td>
<td>Nerve stimulator</td>
<td>Depolarizer, Succinylcholine, Pancuronium, Vecuronium, Rocuronium, Atracurium</td>
<td>Sevoflurane, Desflurane, Isoflurane, Enflurane, Halothane</td>
<td></td>
<td>Peripheral nerve blocks (brachial plexus, femoral, etc.)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Central nerve blocks (spinal, epidural)</td>
</tr>
</tbody>
</table>

EEG = electroencephalogram; IV = intravenous.

Note that the IV agents are quite specific in their effects, except for ketamine, which has both amnestic and analgesic qualities.

The potent inhalational anesthetics contribute to all three components of anesthesia, but nitrous oxide has weak amnestic and analgesic properties and provides no muscle relaxation at all.

The local anesthetics produce excellent analgesia and muscle relaxation but contribute nothing to amnesia or anxiolysis; these anesthetics must be supplemented with an IV sedative.


- Depolarizing muscle relaxants (succinylcholine) act as ACh receptor agonists, whereas nondepolarizing muscle relaxants (pancuronium, vecuronium, atracurium) function as competitive antagonists.
- Atracurium and cis-atracurium undergo degradation in plasma at physiological pH and temperature by organ-independent Hofmann elimination and so can be used in patients with hepatic and renal dysfunction.
- Anticholinesterases prevent breakdown of ACh, thus allowing ACh to compete more effectively with the paralyzing agents. Can be used to “reverse” neuromuscular blockade of nondepolarizing agents (at end of surgery).
Involves use of inhalational agents (e.g., nitrous oxide, isoflurane, sevoflurane, desflurane) and IV agents (e.g., opiates, ketamine, propofol).

**Emergency**
- Return of patient to an awake state.
- Patient should have full muscle strength and protective airway reflexes before extubation.

Airway: Due to anatomy of pediatric airway, intubation can be more challenging (see Table 19-3).

**Anatomy**
- Subarachnoid space lies between arachnoid and pia mater, which contains cerebrospinal fluid (CSF).
- Spinal cord ends at L1–L2.
- Iliac crests are at level of L4.

**Spinal anesthesia**
- For surgeries of lower extremities, lower abdomen, genitourinary, and anal region.
- Insert needle at L3–L4 or L4–L5 (at this level cauda equina is present and spinal cord has already ended).

**Epidural anesthesia**
- Indicated for same surgeries as with spinal anesthesia.
- differs from spinal anesthesia in that needle is placed in epidural space (outside of CSF); commonly an indwelling catheter is left in place.
- With catheter, can have continuous anesthesia (can periodically bolus the catheter for prolonged surgeries).
- Takes longer to obtain effect and requires higher dosages than spinal anesthesia.
- Technically more difficult and requires more time to place than spinal anesthesia.

**TAP (Transversus Abdominis Plane) Block**
- Local anesthesia is used for postoperative pain relief in abdominal surgeries. Best done with ultrasound guidance.

---

**Table 19-3. Ways Pediatric airway differs from adult**

<table>
<thead>
<tr>
<th>Description</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Larger occiput</td>
<td></td>
</tr>
<tr>
<td>Large tongue</td>
<td></td>
</tr>
<tr>
<td>Larynx is higher up (c3 vs. c4–5) and funnel shaped so narrowest portion of airway is beyond what you see!</td>
<td></td>
</tr>
<tr>
<td>Vocal cords are at a slant and more anterior</td>
<td></td>
</tr>
</tbody>
</table>
Local Anesthesia

- Used for local infiltration of operative site, during spinal or epidural anesthesia, and peripheral nerve blocks.
- Classification: Ester or amide anesthetic.
- Mechanism of action: Through blockade of sodium channels.
- Myelinated fibers: More susceptible to blockade.
- Sensory fibers: More readily blocked than motor nerves (dose needed for motor blockade is usually double than that needed for sensory block).
- Nonionized form: Needed to cross nerve sheath while the ionized form is the active form.
- Acidosis: Local tissue acidosis as from infection increases ionized drug form and limits anesthetic activity of the drug. Therefore, local infiltration into an area of infection may not produce adequate analgesia.
- Epinephrine: Adding this to the local anesthetic prolongs duration of action by vasoconstriction. Avoid epinephrine in areas with lack of collateral blood flow.
- Toxicity: Initially CNS effects (tinnitus, restlessness, vertigo, seizures), then cardiovascular effects (hypotension, PR prolongation, QRS widening, dysrhythmias).

Complications

See Table 19-4.

**Table 19-4. Complications of Spinal and Epidural Anesthesia**

<table>
<thead>
<tr>
<th>Complication</th>
<th>Spinal</th>
<th>Epidural</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension (from sympathetic nervous system blockade)</td>
<td>More common</td>
<td>Less common</td>
</tr>
<tr>
<td>Nausea (from unopposed parasympathetic activity)</td>
<td>More common</td>
<td>Less common</td>
</tr>
<tr>
<td>Postspinal headache (due to CSF leak)</td>
<td>Less common</td>
<td>Occurs only with inadvertent dural puncture during epidural placement</td>
</tr>
<tr>
<td>Urinary retention</td>
<td>Common</td>
<td>Common</td>
</tr>
<tr>
<td>Backache</td>
<td>Common</td>
<td>Common</td>
</tr>
<tr>
<td>Permanent neurologic injury</td>
<td>Very rare</td>
<td>Very rare</td>
</tr>
<tr>
<td>Epidural abscess or hematoma</td>
<td>N/A</td>
<td>Rare</td>
</tr>
</tbody>
</table>

Malignant Hyperthermia

**Definition**

Autosomal dominant inherited hypermetabolic syndrome occurring after exposure to an anesthetic agent (very rare but life threatening).
Impaired reuptake of calcium by sarcoplasmic reticulum in muscles.

**Signs and Symptoms**

- Tachycardia, ventricular dysrhythmias
- Hyperthermia
- Hypercarbia, acidosis
- Hypoxemia
- Muscle rigidity

**Treatment**

- Discontinue anesthetics.
- Benzodiazepines (work fastest to control hypermetabolic state).
- Dantrolene sodium (a calcium channel blocker considered more definitive treatment, but onset of action takes about 30 minutes).

**Exam Tip**

Amide anesthetics have ‘T’ before ‘caine’ (e.g., prilocaine, lidocaine, bupivacaine).

**OR Tip**

Do not use epinephrine in these areas:

- Scrotum
- Penis
- Fingers
- Toes
- Ears
- Nose

**High-Yield Facts in Anesthesia**
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Anatomy

Layers of arterial Wall

- **Intima**: One layer of endothelial cells overlying a matrix of collagen and elastin.
- **Media**: Thick layer of smooth muscle cells, collagen, and elastic fibers.
- **Adventitia**: Collagen and elastin, important component to wall strength.

Types of arteries

- **Elastic**: Major vessels, including aorta, subclavian, carotid, pulmonary arteries.
- **Muscular**: Branches from elastic arteries: Radial, femoral, coronary, cerebral.
- **Arterioles**: Terminal branches to capillary beds.

Venous anatomy

- Also three layers, but adventitia is most prominent, and intima and media are generally thin.
- Valves prevent reflux.

Vascular Physical Exam

**Inspection**

Signs of vascular insufficiency:

- Hairless, shiny skin.
- Change of skin color—darkening, mottling, reddening, blanching.
- Nail changes.
- Presence of ulcers or gangrene.
- Edema, erythema.

**Auscultation**

- Auscultation for bruits: Carotid, abdomen, common femoral artery (CFA).

**Palpation**

- Temperature: Cool extremities worrisome for poor circulation.
- Edema.

**Pulsatile Masses**

- Pulsations of normal aorta are palpable in thin people.
- Examine abdomen with both hands around epigastrium and periumbilical area for abdominal aortic aneurysm (AAA).
- Easily palpable pulses in either lower quadrant indicate distal aortic or common iliac aneurysm.

**Extremity Pulses**

- Include brachial, radial, ulnar, femoral, popliteal, dorsalis pedis (DP), posterior tibial.
- Patient should be reclining in supine position, with full exposure of abdomen and legs.
- Legs should be in gentle extension with feet supported.
- To examine pedal pulses, sit at foot of table facing patient.
- Common femoral artery: Found halfway from pubic tubercle to anterior superior iliac spine. (Remember the mnemonic “NAVEL” from lateral to medial: nerves, artery, vein, (empty), lymphatics.)
- Popliteal artery: Place both hands in the middle of the fossa, with fingertips parallel longways (fossa: area between pes anserinus tendon laterally, medial head of gastrocnemius and biceps tendon medially, and biceps and lateral head of gastrocnemius laterally).
- Pedal pulses:
  - DP found between proximal first and second metatarsal (slight dorsiflexion may facilitate).
  - Posterior tibial found posterior to medial malleolus.

### Diagnostic Tests

**Doppler**
- Relates average flow velocity to frequency shift.

**Duplex**
- Combines real-time ultrasound (US) with Doppler analysis.

**Pletysmography**

Measures volume change in organ or body region:
- Pulse volume recording (PVR): Used with Doppler to assess perfusion of distal extremities, assuming change in volume corresponds to change in arterial pressure; useful to predict healing of ulcers and amputations.

**Segmental Blood Pressure Measurement**

- **Ankle-brachial index (ABI):** Systolic blood pressure (SBP) ankle (via Doppler)/highest SBP in upper extremities.
  - ≥ 1: Normal
  - 0.5–0.7: Claudication, up to moderate arterial disease
  - ≤ 0.5: Severe arterial disease
  - ≤ 0.3: Ischemic rest pain, gangrene
- Misleading results associated with:
  - Diabetic patients, as calcification of vessels makes them less compressible, thereby elevating results falsely.
  - Collateral flow in long-standing insufficiency may artificially improve results.

**Arteriography**
- Use of contrast dye with fluoroscopy to delineate arteries.
- Risks: Hemorrhage, allergic reaction to dye, thrombosis of puncture site, embolization of clot, renal dysfunction.
- Dose-independent reactions to dye: Asthma, laryngeal edema, spasm, cardiovascular (CV) collapse.

### Ward Tip

“Scoring” of pulses:
- 0 = no palpable pulse
- 1+ = present, but barely palpable
- 2+ = normal
- 3+ = normal, strong
- 4+ = hyperdynamic, abnormally strong

### Ward Tip

If you are not sure whether pulse is palpable, count out pulses with second person palpating an easier artery such as the radial artery. If pulsations are simultaneous, you are likely palpating pulse accurately.

### Ward Tip

Contrast contains iodine and is renally excreted. Therefore, you should use < 200 cc of dye, and use with caution in patients with renal dysfunction. Ask patients about iodine and shellfish allergies. Hydrate patients prior to and after contrast use.

### Exam Tip

A pseudoaneurysm does not contain all three layers of the arterial wall.
### Complications:
- Neurologic deficits, secondary to emboli.
- Bleeding: Hematoma, hemorrhage at site.
- Decreased pulses compared to pre-angiography: If resolves within 1 hour, may be due to spasm; otherwise, consider arterial injury, clot.

### Postprocedure:
- Maintain patient supine for at least 6 hours.
- Check puncture site for hematoma or false aneurysm.
- Follow neurovascular exam, including mental status.
- Maintain well-hydrated state.

### Digital Subtraction Angiography (DSA)
- Dye is injected into vein or artery, and computerized fluoroscopy subtracts bone and soft tissue so that only the arterial system is visible.

### Spiral Computed Tomography (CT)
- Especially useful for AAA.

### Magnetic Resonance Angiography (MRA)
- Allows good visualization of patent distal vessels with minimal flow; also useful for evaluation of carotid bifurcation and abdominal aorta.
- Advantage: No contrast used. Gadolinium causes allergic reactions in fewer patients.
- Disadvantage: Cost, limited ability to visualize arterial calcification.

### Acute Arterial Occlusion

#### Etiology
- Embolization: From heart or any proximal artery.
- Trauma: Posterior knee dislocation, long-bone fracture, penetrating trauma.
- Iatrogenic (catheter-related).
- Thrombosis: Atherosclerosis, aneurysm.

#### Signs and Symptoms of Acute Arterial Insufficiency
- Paresthesia and paralysis are most important signs because nerves are most sensitive to ischemia. Secondarily, there will be loss of muscle function.
- Pain may not exist in diabetics with neuropathy or if rapidly progressive ischemia occurs with anesthesis.
- Earliest sign in lower extremity will be along distribution of peroneal nerve, with hypesthesia, no great toe dorsiflexion, foot drop.
- Progression in muscle damage: Muscles soft, then doughy, then stiff/hard.
- Once skin is mottled and no longer blanches, tissue ischemia is irreversible. Color change indicates extravasation of blood from capillaries into dermis.

### Differential Diagnosis
- Nerve root compression
- Deep venous thrombosis (DVT)
- Infection
**Diagnosis**

- If diagnosis is certain (i.e., cold, newly pulseless, painful extremity), no further workup is required. Patient should be promptly taken to OR.
- Typically, ABI and/or angiography are performed to confirm the site of the lesion. Currently, on-table angiography is an option in some centers.

**Embolism**

- Must find source of embolism and reason.
- Patient may have chronic atrial fibrillation or history of myocardial infarction (MI) or valvular disease (mitral stenosis, with valvular vegetation or mural thrombus).
- Atheroembolism: From aorta, iliac, or femoral vessels to distal vessels.
- “Blue toe” syndrome.
- Likely to have palpable pedal pulses anyway because at least one proximal vessel is likely still patent.
- Suspect atheroembolism in patients with no history of peripheral vascular disease (PVD), with digital ischemia and palpable pulses.

**Treatment**

- See below.
- Address primary problem (i.e., atrial fibrillation).

**Thrombosis**

- Usually underlying stenosis from atherosclerosis.
- May occur in hypercoagulable states, especially with repeated mild trauma.
- Effects of thrombus vary greatly from no symptoms to severe ischemia depending on extent of collateral circulation.
- All patients should have an angiogram unless ischemia is severe and rapidly progressive.
- Cause suggested by history of PVD, popliteal, or aortic aneurysm.
- On exam, patient may have evidence of PVD such as skin changes or lack of distal pulses on contralateral side.

**Treatment**

- Thrombectomy or grafting (most common).
- Thrombolytic therapy.
- Success rate: 60–80%.
- Mechanism: Activation of plasmin system causes fibrinolysis and clot dissolution.
- Agents:
  - Streptokinase: Bacterial origin (antigenic), binds plasminogen to make plasmin, not often used.
  - Urokinase: From renal parenchyma, directly activates plasminogen, not currently available in the United States.
  - Tissue plasminogen activator (tPA): From vascular endothelium, directly activates plasminogen.
  - Reteplase: Recombinant tPA, catalyzes cleavage of endogenous plasminogen to form plasmin.
- Indications: Acute occlusion of native vessel or graft.
- Contraindications: History of gastrointestinal (GI) or intracerebral lesion, pregnancy, any contraindication to angiography.
- Method: Catheter placed proximally, diagnostic angiogram performed, catheter advanced into clot, thrombolytic agent administered.
complications
- Intracerebral bleed, catheter site bleed (both infrequent).
- Patient may ultimately require operation to fix underlying problem.

Chronic Ischemia

presentation
- Claudication.
- Rest pain.
- Gangrene.
- Most commonly affects infrarenal aorta, iliac arteries, SFA at adductor canal.

risk factors
- Systolic hypertension (HTN)
- Cigarette smoking
- Hyperlipidemia
- Diabetes

aortoiliac Disease

Types
- Localized disease of aorta and common iliac disease (10%).
- Localized disease of external iliac artery (25%).
- Multisegmental disease, including infrainguinal (65%).

Differential Diagnosis
- Nerve root compression (lumbosacral), secondary to disc herniation or spinal stenosis.

Diagnostic Tests
- Femoral-brachial pressure ratio.
- Angiogram.
- Axillofemoral has better patency rates and is often used for unilateral disease.
- Indicated when patient is extremely at high risk for medical complications following intra-abdominal surgery or if graft from prior operation is infected.

Treatment
- Aortofemoral bypass:
  - Five-year patency 90%, 10-year patency 75%.
  - Better patency rates than aortoiliac bypass.
- Aortoiliac endarterectomy: Acceptable for localized atherosclerotic disease when distal vessel is normal.
- Extra-anatomic bypass:
  - Best option: Femoral-femoral bypass (5-year patency 70%).
  - Other options: Axillofemoral, axillofemoral—used in high-risk patients for intra-abdominal surgery (elderly, hostile abdomen, abdominal infection).

WARD TIP
Thrombolytic therapy for acute thrombotic occlusion is not done if severe ischemia is present, as it takes time to dissolve clot.

EXAM TIP
If chronic ischemia occurs at locations other than infrarenal aorta, iliacs, or SFA, the patient is likely to have a comorbid disease, such as diabetes (increased risk at profunda femoris and tibial vessels) or an inflammatory disorder (increased risk at axillary arteries).

EXAM TIP
Leriche syndrome is indicative of aortoiliac disease and presents with claudication, impotence (decreased flow from the internal iliacs), and decreased (or absent) femoral pulses.
Percutaneous transluminal angioplasty (PTA): Indicated for short, nonoccluding lesions with less extensive atherosclerosis. Five-year patency rate is 90% with secondary interventions.

Complications
- Hemorrhage.
- Thrombosis of reconstruction.
- Distal embolization.
- Ischemic colitis.
- Paraplegia from lumbar vessel ischemia.
- Sexual dysfunction (if operation involved abdominal aorta).

Infragenital Disease
- Most commonly affects SFA at adductor canal.
- Patients have decreased life expectancy regardless of treatment and outcome.
- Ulcers:
  - Arterial insufficiency: Lateral ankle and foot, pale, no granulation tissue.
  - Venous insufficiency: Medial malleolus, pink, granulation tissue, other stigmata of venous disease.

Diagnostic Tests
- Doppler
- ABI
- PVRs
- Angiogram

Management
- Nonoperative:
  - Smoking cessation, increase exercise tolerance, medication.
  - Pentoxifylline: Decreases viscosity, increases flow; unpredictable relief of symptoms but proven to increase microvascular flow.
  - Cilostazol: Type III phosphodiesterase inhibitor, reduces platelet aggregation and increases vasodilation. Can be more effective than pentoxifylline.
- Operative:
  - Revascularization requires adequate inflow, outflow, and conduit.
  - Bypass (femoral-popliteal): Uses polytetrafluoroethylene (PTFE) or saphenous vein, reversed or in situ—reversed saphenous vein graft (RSVG) best patency.
  - Endarterectomy: Limited to short lesions of SFA at adductor canal or origin of profunda.

Upper Extremity Disease
- Compared to lower extremity, more often due to vasospasm or arteritis.
- Palpate pulses: Axillary, radial, ulnar, brachial.
- Subclavian steal syndrome.
- Non hemispheric cerebrovascular symptoms with mild arm claudication due to decreased flow to posterior cerebral artery (PCA) when blood flows retrograde through vertebral artery to SCA.
- If patient has neurologic symptoms, then carotid stenosis is present as well.
- Cause is proximal SCA lesion.
- Angiogram shows flow reversal in vertebral artery.

WARD TIP
Patients presenting with rest pain, ulcers, or gangrene most likely have multisegmental disease.

WARD TIP
ABI is likely to be artificially elevated in a diabetic patient because the vessels are generally less compressible than normal. PVRs are more useful because results are not affected by compressibility of vessels.
- Operate for:
  - Incapacitating claudication.
  - Emboli to hand or to posterior cerebral circulation.
  - Symptoms of subclavian steal (if also carotid symptoms, fix carotid first).

**carotid artery stenosis**

**general**
- Differentiate based on asymptomatic or symptomatic.
- Asymptomatic: No history of transient ischemic attack (TIA)-like symptoms.
- Symptomatic history of:
  - Amaurosis fugax—transient blindness due to occlusion of the ophthalmic artery (favorite question of surgeons!).
  - Middle cerebral artery (MCA) syndrome.
  - Anterior cerebral artery (ACA) syndrome.
- Syncope is not considered “symptomatic” because unilateral carotid occlusion rarely results in impairment of consciousness.

**evaluation**
- **Duplex Doppler Ultrasound**
  - Detects increased flow in stenotic lesions.
  - Not reliable in very high-grade stenoses.
  - Near-complete stenosis results in “trickle” flow.
  - May be mistaken for complete occlusion.
  - Cannot assess carotids above the mandible.
- **MRA**
  - Detects functional flow.
  - Can estimate plaque thickness.
  - Can evaluate intracranial vasculature.
  - May not resolve trickle flow.
- **Carotid Angiogram**
  - Gold standard.
  - Reveals trickle flow.
  - Cannot discern thickness of plaque.

**Treatment**
- Medical therapy: Aspirin, aspirin plus dipyridamole, clopidogrel.
- Control of underlying medical conditions (HTN, diabetes mellitus, hyperlipidemia).
- Smoking cessation.
- Surgical therapy—carotid endarterectomy (CEA):
  - Indications: Ipsilateral neurologic symptoms with carotid artery stenosis or asymptomatic with > 70% carotid stenosis.
  - CEA has been shown to be more effective than aspirin alone in preventing subsequent strokes in all symptomatic patients. It is superior to medical management alone in asymptomatic females with > 70% stenosis and in asymptomatic males with > 60% stenosis.
  - Emergency CEA may be indicated in symptomatic cases of recent onset with angiographically proven occlusion or loss of a known previous bruit.
Procedure:
- Patient is maintained on aspirin preoperatively.
- Timing is generally 4–6 weeks after CVA.
- Intraoperative electroencephalogram (EEG) and sensory evoked potentials routinely monitored in case ischemia develops.
- The surgeon must ensure complete dissection of the plaque in order to prevent remnant plaque rupture with subsequent embolic stroke.
- After intraoperative ICA occlusion, measurement of retrograde flow from opposite circulation ("stump pressure") should yield mean arterial pressure (MAP) > 50 mmHg. ICA shunt may be indicated if the stump pressure is < 50 mmHg or if the contralateral side also has a tight stenosis.

Risks/complications:
- Operative mortality: 1–5%.
- Vagus is the most common nerve injured (during clamping of the common carotid).
- Hoarseness—recurrent laryngeal nerve injury.
- Horner syndrome—injury to sympathetic plexus coursing along with carotids.
- Partial tongue paresis (hypoglossal nerve injury).
- Hematoma causing airway compromise.
- Cerebral hyperperfusion syndrome.
- Unilateral headache due to poor autoregulation.
- Seizures.
- Carotid occlusion (100% stenosis) of chronic nature usually has little benefit from surgical intervention. Typically, slowly progressive carotid stenosis affords development of alternative collateralization to the anterior cerebral circulation (e.g., through external carotid artery [ECA] anastomoses).

Amputations

Indications
- Nonviable extremity that has become infected.
- Irreparable vascular injury with irreversible ischemia (traumatic or atraumatic).
- Cancer.
- Elderly patient with infection who is not a candidate for surgical revascularization.

Types of amputations
- Toe: For gangrene or osteomyelitis distal to proximal interphalangeal joint (PIP) without proximal cellulitis, necrosis, or edema.
- Transmetatarsal: For necrosis between level of transmetatarsal incision and PIP, often interdigital crease necrosis.
- Syme’s: Amputation at base of tibia and fibula. For terminal arterial disease of distal foot—not done very often, requires palpable posterior tibialis pulse.
- Below-knee amputation (BKA) (most common level for nonviable foot): For ischemia up to malleoli.
  - Likely to adapt well to prosthesis.
  - Contraindicated if gangrene more proximal than ankle, or if patient has hip or knee contractures.
  - Likely to fail when there is no femoral pulse.
- Above-knee amputation (AKA): For gangrene above below-knee level, for contractures at knee or hip, and for elderly nonambulatory patients.

OR TIP
Only superficial layers should be closed primarily after CEA. Closure of deep fascia creates an enclosed space capable of retaining hematoma under high pressures. Should an arterial leak develop, the airway may be rapidly compromised.

WARD TIP
Carotid artery dissection must always be in your differential in a patient presenting with neurological deficits of the head after a traumatic episode.
Best chance of healing, particularly in PVD patients.
- Hip disarticulation: For extensive lower extremity ischemia, proximal gangrene, tumor, extensive trauma.
- Poor outcome when performed for PVD.
- Upper extremity amputations: More commonly performed for trauma or tumor.
  - Digital, forearm, upper arm.
  - Leave as much as possible, unless for tumor.

**Basic principles**

1. Patient should be medically stabilized first when possible.
2. Select level of amputation for PVD:
   - Use PVRs and ABI to help predict chance of healing.
   - At proposed level, infection is controlled, skin looks good, without proximal dependent rubor, proximal pain, and with a venous filling time < 20 seconds.
   - If for cancer, wide excision necessary.
   - If status post trauma or for PVD, remaining tissue level must be acceptably healthy.
   - Consider length of stump in regard to ease of fitting/using prosthesis.
   - Consider overall condition of patient.
3. Determination of closure:
   - Standard: Flaps constructed to close over bone.
   - Guillotine (in situation of sepsis): Level is lower than that eventually desired. Stump left open for dressing changes until sepsis is resolved, at which time completion amputation is performed.
4. Postoperative care:
   - Dressing left in place for at least 3 days unless patient becomes febrile or has profuse bleeding through dressing.
   - Rehabilitation.

**Chronic Venous Disease: Obstruction and Incompetence**

**Basics**

- Postphlebitic syndrome: After DVT, many patients (50%) get chronic venous insufficiency due to valvular incompetence of recanalized veins.
- Up to 50% of patients with signs and symptoms of venous insufficiency have no documented history of DVT.

**Presentation**

- Pain and swelling in affected leg.
- Findings on exam include induration, swelling, skin darkening, and possibly ulcers.

**Treatment**

- Goals:
  - Alleviate symptoms.
  - Heal any ulcers that exist.
  - Prevent development of new ulcers.

**WARD TIP**

If patient has popliteal pulse, BKA has 90-97% success rate. Without palpable popliteal pulse, success rate is about 82%.

Longer stump is more functional.

When the patient is compliant and therapy is properly done, 85% of ulcers heal, but may recur.
- Nonoperative:
  - Compression stockings.
  - Leg elevation.
  - Ulcer treatment: Bed rest, antibiotics when cellulitis present, thromboembolism deterrents (TEDs), Unna’s boots, dressings.
  - Generalized skin care, with antifungal creams when appropriate, moisturizer for eczema.
- Operative:
  - Indications: Severe symptoms, failure of conservative measurements, recurrent ulcers.
  - Options:
    - **Ligation** of responsible perforators (usually around medial malleolus).
    - Valvuloplasty (leads to symptomatic improvement in 60–80%).
    - Venous reconstruction with grafting (not popular).

### Aneurysm

**Definition**

- An irreversible dilatation of an artery to at least 1.5 times its normal caliber.
- True aneurysm: Involves all layers of arterial wall.
- False aneurysm: Involves only a portion of wall, or involves surrounding tissue.

**Varieties of aneurysm**

- Degenerative—due to atherosclerosis, the most common type of aneurysm:
  - Intima replaced by fibrin.
  - Media fragmented with decreased elasticity.
  - Imbalance in elastin metabolism, between elastase and $\alpha_1$-antithrombin.
- Traumatic—due to iatrogenic, catheter-related injury, or to penetrating trauma:
  - Results in focal defect in wall; hemorrhage controlled by surrounding tissue.
  - Formation of fibrous capsule.
- Poststenotic—from Bernoulli’s principle, occurs distal to cervical rib in thoracic outlet syndrome, distal to coarctation of aorta, or to aortic or pulmonary valvular stenosis.
- Dissecting—blood travels through an intimal defect, creating a false passegway between the intima and the inner two thirds of the media:
  - If ruptures externally, patient exsanguinates.
- Mycotic—infected.
  - #1 agent Salmonella, followed by Staphylococcus.
- Anastomotic—separation between graft and native artery, with formation of sac and fibrous capsule, often in CFA status post aortofemoral bypass:
  - Painless, pulsatile, groin mass.
  - Diagnose with duplex for peripheral aneurysms, and CT for abdominal aneurysms, or angiogram.

WARD TIP

Unna boots: A wrap for the leg which has been soaked in zinc oxide and calamine lotion. The leg is wrapped from the base of the toes to the tibial tuberosity and left on for a week; it provides compression, healing properties from the zinc, and moisturization. It is used for the treatment of venous stasis ulcers.
aBDominal aortic aneurysm (AAA)

- Fusiform dilatation of abdominal aorta more than 1.5 times its normal diameter.
- Normal aorta 2–3 cm.
- Due to degeneration of the medial layer.
- Incidence: 2% of elderly population (nine times more common in males than females).
- Seventy-five percent are asymptomatic at diagnosis.
- If symptomatic, can present with rupture, distal embolization, compression of nearby organs.
- Risk factors for rupture: Diastolic HTN, initially large size at diagnosis, chronic obstructive pulmonary disease (COPD)

**Screening**

- Abdominal US one time for males age 65–75 with history of smoking.

**Indications for Repair**

- Size at which the risk of rupture exceeds the risk of mortality from the operation (generally about 2–5%).
- Increasing size: > 1 cm per year; monitor with CT/US every 6 months.
- Consider factors that increase risk of rupture (diastolic HTN, COPD).
- Symptoms, including pain over AAA site.

**Diagnosis**

- Exam: Periumbilical palpable pulsatile mass.
- CT: Provides information about character, wall thickness, and location with respect to renal arteries, presence of leak or rupture.
- Magnetic resonance imaging (MRI): Provides more details than CT or US about the lumen, surface anatomy, neck, relationship to renal arteries.
- US: Used more frequently to follow aneurysm size over time than to assess in acute phase (see Figure 20-1).
- Angiogram: Defines vascular anatomy and is especially important in cases of mesenteric ischemia, HTN, renal dysfunction, horseshoe kidney, claudication.

**Preoperative Assessment**

- Includes assessment of carotid disease and cardiac, pulmonary, renal, and hepatic systems.

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**WARD TIP**

Size at which AAA is considered for surgical repair: 5.5 cm.

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**Figure 20-1.** AAA on ultrasonography. Aortic diameter is 5.40 cm.
Cardiac workup and optimization:
- No history of coronary artery disease (CAD): 1–3% chance of MI, pulmonary edema, or cardiac death perioperatively.
- Known CAD, risks increase to 5–10%.
- Steps to minimize risk:
  - No cardiac history, normal electrocardiogram (ECG): No further workup.
  - Known but stable CAD: Echocardiogram/dipyridimole or thallium.
  - If abnormal, consider preoperative revascularization.
  - Clinically severe CAD: Definite need for revascularization.
  - If percutaneous, may proceed immediately with AAA repair.
  - If coronary artery bypass graft (CABG), wait 4–6 weeks.

Operative Plan (elective)
- Open repair:
  - Approach: Transperitoneal or retroperitoneal.
  - Retroperitoneal approach: Avoids formation of intra-abdominal adhesions, does not interfere with any GI or genitourinary (GU) stoma that may exist, is better tolerated in COPD patients, and provides better suprarenal exposure.
- Endovascular repair (EVAR):
  - Endovascular system consists of the graft, stents, and a delivery mechanism.
  - Problems include immediate thrombosis and leakage at anatomic sites.
  - Should be considered in patients who are elderly and/or have high operative risk due to a multitude of medical comorbidities.
  - Benefits compared to open repair: Shorter procedural time, hospital stay, and recovery time; recent evidence suggests improved mortality.

Emergent repair of ruptured AAA
- Higher-risk operation.
- Signs and symptoms: Abdominal pain, severe back or flank pain, CV collapse.
- Differential diagnosis: Perforated peptic ulcer, renal or biliary colic, ruptured intervertebral disk.
- Treatment:
  - In an unstable patient with known AAA: Proceed to OR immediately—“mobilize the team.”
  - In a stable patient, without history of AAA: CT first, then OR as needed.
  - OR:
    1. Team scrubs and preps/drapes.
    2. Only then is anesthesia induced, as surgeon has knife in hand. (Anesthesia causes vasodilatation, which may worsen hypotension.)
    3. Right angle retractor used to compress supraceliac aorta against vertebrae so patient can be resuscitated by anesthesia.
    4. Retractor replaced with supraceliac clamp.
    5. AAA repaired.

Complications of AAA repair
- Renal failure: 6% elective, 75% ruptured—acute tubular necrosis (ruptured) or atheroemboli (elective).
- Ischemic colitis (5%): Bloody diarrhea, elevated white blood count (WBC), peritonitis.
- Spinal cord ischemia: Disruption of artery of Adamkiewicz.
- EVAR: 20–25% will develop endoleak (flow outside graft lumen but inside aneurysm sac).
Other Aneurysms

Iliac Artery
- Common iliac artery (CIA), 90%; hypogastric artery, 10%.
- Hypogastric artery aneurysms may cause a pulsatile mass palpable on digital rectal examination.
- Resection indicated when > 3 cm, otherwise 6-month follow-up indicated.
- CIA aneurysm is treated with interposition graft.

Popliteal Artery
- Most common peripheral aneurysm.
- Fifty to 75% of cases are bilateral aneurysms.
- Untreated, 60% will lead to thrombosis or distal embolization, and 20% to amputation.
- Diagnosis established on exam, and using US and angiography.
- Operation indicated if no serious comorbidity: Excision and interposition graft with RSVG or ligation and bypass.

Femoral Artery
- Uncommon.
- On exam, mass, local pain, venous obstruction, embolism, thrombosis.
- Whether symptomatic or not, resection and graft indicated once > 2.5 cm.

Diabetic Disease

Location
- Usually spares aortoiliac vessels.
- Affects distal profunda femoris, popliteal, tibial, and digital arteries of the foot.
- Small vessels affected: Microangiopathy occurs with intimal and basement membrane thickening.
- Large vessels affected with atherosclerosis and calcification of media.

Problems
- Diabetic neuropathy: Causes motor and sensory deficits as well as high arch deformity in the foot that increases pressure on tarsal heads (atrophy of intrinsic muscles).
- Arteriopathy.
- Infection: Likely to be worse—gram negatives, gram positives, and anaerobes all potential culprits, in particular Peptococcus, Proteus, Bacteroides.

Treatment
- Revascularization indicated when ulcers occur if there are significant vessels available to use in repair. (See above information for further details that apply to diabetic and nondiabetic patients.)

Venous Thrombotic Disease

Anatomy
- Superficial: Greater and lesser saphenous veins.
Deep: Veins follow arteries and have same names (popliteal, superficial femoral vein [SFV], distal femoral vein [DFV], common femoral vein [CFV], external iliac vein [EIV]).
Perforators: Connect deep and superficial.

**Physiology**

- Systemic veins contain two thirds of circulating volume.
- Further from heart, there are more valves.
- Venous return depends on respiration: Inspiration causes descent of diaphragm, increased intra-abdominal pressure, and decreased venous return.
- Venous return generated by the relationship among contraction of the heart, static filling pressure, and gravity.
- Virchow’s triad: Stasis, blood abnormality, vessel endothelium injury (factors allowing thrombosis to occur).

**Deep Venous Thrombosis (DVT)**

- Stasis leads to thrombin formation, which leads to platelet aggregation.
- Endothelial injury and/or a hypercoagulable state may contribute.
- Screening warranted for younger patients or those with repeated occurrences.
- Check prothrombin time (PT), partial thromboplastin time (PTT), WBC, hematocrit (Hct), erythrocyte sedimentation rate (ESR), platelets.
- For extremely high-risk patients, also check homocysteine level, antiphospholipid (APL) antibodies, proteins C and S, antithrombin III (AT III), activated protein C resistance (APC-rt), platelet aggregation, mutant factor V Leiden, prothrombin gene mutation (factor II).
- Also check CT scan of the chest, abdomen, and pelvis to look for malignancy if patient has no other risk factors for DVT.

**Risk Factors**

- Central venous line placement.
- Congestive heart failure (CHF) and MI (20–40%): Passive congestion and increased blood viscosity.
- Joint replacement operation (15–30%): Femoral vein trauma, long OR, postop immobility.
- Fractures of hip, pelvis, proximal femur (35–60%): Endothelial damage.
- Laparoscopic surgery (7–10%): Intra-abdominal pressure > pressure of venous return from legs.
- Prior DVT (five times): Scarring, valvular damage, decreased muscle pumping, stasis.
- Hormone replacement therapy (HRT) (two to six times): Increases coagulation factors, and decreases protein S, AT III, and fibrinolytic activity.
- Other: Trauma patients, malignancy, obesity, pregnancy, sepsis, prolonged immobility.

**Congenital**

- Antiphospholipid antibody syndrome (APS): Antibodies change normal endothelial function.
- AT III deficiency.
- Plasminogen deficiency: Decreased fibrinolysis.
- Dysfibrinogenemia: Fibrin resistance to plasmin proteolysis, abnormal fibrinogen, defective thrombin binding, increased blood viscosity.
- Factor V Leiden/APC-r: Factor Va resistant to degradation by APC; hyper-viscosity syndrome.
- Lupus anticoagulant: Exact mechanism unknown.
- Protein C and S deficiency: Factors V and VIII are not appropriately inactivated.
- Prothrombin gene mutation (factor II).

**Diagnosis**
- Exam: Calf tenderness, swelling, Homan’s sign (not very reliable).
- Duplex: Demonstrates thrombus, assesses compressibility of veins, analyzes venous flow.

**Prophylaxis**
- TEDs (compression stockings): Increase velocity of venous flow, reduce venous wall distention, and enhance valvular function.
- Sequential compression devices: Reduce incidence of DVT by up to 75% when used properly.
- Pharmacologic: Subcutaneous heparin, low-molecular-weight heparin.

**Treatment**
- Bed rest until pain and swelling subside.
- Lower extremity elevation.
- Anticoagulation (period of 6 months for first DVT).
- Elastic stockings once ambulating.
- Consider fibrinolytics for large thrombi.

**Long-Term Outcome**
Recurrent DVT: 18% at 2 years, 30% at 8 years.

**Postphlebitic Syndrome**
- Occurs in 24%.
- Risk of recurrent DVT then increased six times.
- Symptoms: Edema, pain, aches, fatigue, skin discoloration, scarring, ulcers.

**Superficial Venous Thrombosis (SVT)**
- Usually a noninfected, localized inflammatory reaction.
- **Signs and symptoms:** Swelling, pain, erythema, tenderness.
- **Diagnosis:** Duplex ultrasonography to rule out associated DVT.
- **Treatment:**
  - Nonsteroidal anti-inflammatory drugs (NSAIDs) as needed until symptoms resolve (about 2–3 weeks).
  - Use elastic stockings.
  - Consider anticoagulation for involvement of saphenofemoral junction.

**Vascular Trauma**

**Acute Compartment Syndrome (ACS)**
- Definition: Increased pressure within a compartment with subsequent compromise to the circulation and tissue function.
Trauma-related etiologies: Fractures (69%), crush injuries, thermal burns, constrictive dressing, injury to vascular structures.

It is important to remember that non-trauma causes also exist. These involve anything that may increase compartment pressure. (IV fluid extravasation, anticoagulation leading to intramuscular hemorrhage, thrombosis, any ischemic-reperfusion injury.)

Muscle groups of limbs are divided into compartments by unyielding fascial membranes.

Signs and symptoms:
- The 5 “Ps”:
- Pain out of proportion
- Pallor
- Pulselessness
- Paresthesia
- Paralysis of muscle associated with that compartment. Ex: ACS of posterior thigh compartment leads to inability to flex the knee (late finding)

Diagnosis: ACS is a clinical diagnosis. Compartment pressures for ancillary information.
- 0–8 mmHg: Normal pressure
- 25–30 mmHg: Capillary blood flow compromised
- 25–30 mmHg: Onset of pain
- As compartment pressure nears the diastolic pressure, ischemia occurs.
- Diastolic pressure—compartment pressure < 30 mmHg: indication for fasciotomy.

Management:
- If high suspicion, proceed with fasciotomy.
- Prophylactic fasciotomy if concerned:
  - Vascular injury leading to ischemia time of 4–6 hours
  - In concert with some orthopedic procedures (comminuted fractures)
  - Also, remember the basics: Removal of tight dressings, placement of limb at level with heart, analgesics, supplemental oxygen.

Complications:
- Metabolic acidosis from ischemic muscle can lead to myocardial depression, hypotension.
- Hyperkalemia due to release of potassium from injured muscle.
- Dessication of open muscle. Avoid with wet-to-dry dressing.

**Clues to Vascular Injury in the Trauma Patient**
- Pulsatile or expanding hematoma.
- Pulsatile bleeding.
- Bruit/thrill.
- End-organ ischemia.
- Unexplained shock.
- Likely location.
- Injury to adjacent nerve.
- Extremity fractures with weak or absent pulses.

**A Few Facts**
- Carotid artery injury with hematoma can affect cranial nerves IX, X, XI, and XII.
- All zones in neck injury are likely to have vascular injury.
- Limb loss secondary to arterial injury associated with lower extremity fracture > 40%.
- Twenty percent of patients with penetrating abdominal trauma will have major vascular injury.

WARD TIP
Use direct pressure to control bleeding. Apply point pressure using a finger, not the entire hand. Tourniquets are really only a last resort.
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High-Yield Facts in Pediatric Surgery
Fluid Management

Maintenance fluid calculation:
- First 10 kg: 4 cc/kg
- Second 10 kg: 2 cc/kg
- Every additional kilogram past 20 kg: 1 cc/kg
- Minimal urine output: 1–2 mL/kg/hr

Neck Masses/Lesions

Types
- Congenital
- Inflammatory
- Neoplastic

Location

Location is key in diagnosis.
- **Midline:**
  - Thyroglossal duct remnants
  - Submental lymph node
  - Goiter
- **Lateral:**
  - Branchial cyst
  - Lymphadenitis
  - Lymphoma
  - Carotid body tumor (paraganglioma)
- **Midline or lateral:**
  - Cystic hygroma
  - Dermoid cyst
- **Bilateral:**
  - Cervical lymphadenitis

Thyroglossal Duct Cysts

See ENT chapter.

Branchial Cleft Cysts

definition

Remnants of the four branchial clefts in the anterior neck that can form cysts or sinus tracts leading through to the skin.

Signs and Symptoms
- Lateral neck mass that may present with drainage.
- Most common form is second branchial cleft cyst that present at the anterior border of sternocleidomastoid muscle.
- Usually painless.
- Fluctuant, mobile, and nontender.

diagnosis

Ultrasound (US).
**Treatment**
- Complete excision of cyst and entire tract.
- Antibiotics if infected.

**Cystic Hygroma**

**Definition**
Congenital lymphangioma.

**Signs and Symptoms**
- Most are identified at birth.
- May be recognized after injury or upper respiratory infection.
- Painless, soft, and mobile.
- Transilluminate brightly.
- May compress trachea or spread into the floor of mouth, causing upper airway obstruction.
- Do not regress spontaneously.

**Diagnosis**
Computed tomography (CT) scan (extent and involvement of surrounding structures).

**Treatment**
Surgical excision is necessary to prevent enlargement and possible airway compromise.

---

**Congenital Deformities of the Mediastinum and Thorax**

**pectus excavatum**

**Definition**
Abnormal asymmetric development of costal cartilages causing posterior distortion (caving in) of the sternum and xiphoid.

**Signs and Symptoms**
- Abnormal “funnel chest” appearance.
- May cause respiratory compromise and exertional dyspnea.
- Occasionally associated with mitral valve prolapse.

**Treatment**
- Surgery at 6 years of age or older.
- Osteotomy to remove abnormal cartilage with a temporary substernal strut to stabilize sternum until new cartilage forms.
- Alternatively: Nuss procedure—only strut placement with no osteotomy.

**pectus carinaTum**

**Definition**
Abnormal asymmetric growth of costal cartilages, causing anterior distortion of sternum and xiphoid; less common than pectus excavatum.

---

**WARD TIP**
Cystic hygroma is the most common lymphatic malformation in children and is most commonly found at the floor of the oropharynx.
**Signs and Symptoms**
- Abnormal “pigeon chest” appearance.
- Usually asymptomatic.

**Treatment**
- Brace to correct deformity, can be used up to 18 years of age.
- Surgery (osteotomy) after 6 years of age, usually for cosmetic purposes to avoid psychological distress in children not willing to wear braces.

**Tracheoesophageal (Te) Malformations**

**Definition**
Failure of complete separation of trachea from esophagus. Can occur with or without esophageal atresia and usually involves a TE fistula.

**Embryology**
Esophagus and trachea originate from a single diverticulum and divide at fifth week of gestation.

**Epidemiology**
- One in 3,500 births.
- Most commonly “type C” (esophageal atresia with a distal TE fistula).

**Signs and Symptoms**
- Respiratory distress or choking following first feeding.
- Excess drooling and salivation.

**Types (Figure 21-1)**
- Pure esophageal atresia.
- Esophageal atresia with proximal TE fistula.
- Esophageal atresia with distal TE fistula.
- Esophageal atresia with proximal and distal fistulas.
- “H-Type”—no esophageal atresia with TE fistula.

**Diagnosis**
- Esophageal atresia becomes evident within the first few hours of life because of severity of symptoms, inability to pass feeding tube.
- Chest x-ray (CXR): Tube to end or coil in the region of thoracic inlet.
- Abdominal x-ray (AXR).
- Contrast x-ray studies.

**WARD TIP**
Fifty percent of infants with esophageal atresia have associated anomalies. Most commonly part of **VACTERL** constellation of anomalies:
- Vertebral
- Anal
- Cardiac
- Tracheoesophageal fistula
- Renal
- Limb

TE malformations also associated with:
- Polyhydramnios
- Preterm birth
- Small for gestational age

**EXAM TIP**
Most common malformation with esophageal atresia is a TE fistula (95%).

**AXR Absence of air in stomach. Think: Esophageal atresia without fistula.**

**Figure 21-1. Five different forms of the fistula.** (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz’s Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015: Figure 39-8).
Treatment
- Decompress blind esophageal pouch and control oral secretions with sump tube on constant suction.
- Parenteral prophylactic antibiotics.
- Evaluate for other anomalies (primarily cardiac and renal)—cardiac echo to define aortic arch.
- If cardiac and respiratory stable, may repair surgically.
- Surgical repair:
  - Ligation of fistula and insertion of gastrostomy tube.
  - Anastomosis of two ends of esophagus.

Congenital Diaphragmatic Hernia (CDH)

Definition
Patent pleuroperitoneal canal through the diaphragm leading to pulmonary hyperplasia of the restricted ipsilateral lung and respiratory distress. May result in pulmonary hypertension.
Two types:
- Bochdalek: Posterolateral hernia.
- Morgagni: Anterior retrosternal hernia (in 50% of cases seen with other congenital anomalies).

Signs and Symptoms
- Significant respiratory distress within first few hours of life.
- Scaphoid abdomen.

Diagnosis
- US (prenatally)
- CXR:
  - Bowel gas pattern in hemithorax.
  - Mediastinal shift.

Treatment
- Respiratory and metabolic support (endotracheal [ET] and nasogastric [NG] tubes).
- Gastric decompression.
- Surgical correction by hernia reduction and repair of defect (primary repair or with a synthetic patch).
- If unstable, treated with extracorporeal membrane oxygenation (ECMO) before surgery.

Prognosis
- Survival rate is 50%.
- Predictors of mortality:
  - Pulmonary hypoplasia.
  - Pulmonary hypertension.

Pulmonary Sequestration

Definition
A nonfunctioning embryonic and cystic pulmonary tissue that receives its own blood supply from the systemic circulation (aorta) and does not communicate with functional airways.
Intralobar: Contained within normal lung.
- No pleural covering.
- Not commonly associated with other anomalies.
- Present with infection.
- CXR may reveal mass lesion and air-fluid level.
- Venous drainage via pulmonary veins.
- Extralobar: Separated from normal lung; can even be under diaphragm.
- Pleural covering present.
- Associated with diaphragmatic hernia.
- Incidental finding on CXR.
- Venous drainage via systemic veins.

Diagnosis
Magnetic resonance imaging (MRI) or aortography to demonstrate sequestration of lung tissue within systemic venous drainage.

Treatment
Surgical removal.

**Or Tip**
Prior to resection of lobe, it is necessary to examine blood supply to ascertain that it is not anomalously originating from the abdomen; cutting the vessel in this case can lead to its retraction into the abdomen and internal hemorrhaging.

**Ward Tip**
Congenital cystic adenomatoid malformation is the second most common congenital lung lesion (after lobar emphysema).

**Exam Tip**
Presentation of congenital cystic adenomatoid malformation similar to diaphragmatic hernia.

**Exam Tip**
Where is the NG tube tip?
- Tip in thorax: CDH
- Tip in abdomen: CCAM
This makes the difference between a thoracic and an abdominal surgery!

**Abdominal Wall Defects**

**Gastroschisis**
Centrally located, full-thickness abdominal wall defect, leading to exposed bowel.

**Epidemiology**
One in 10,000 live births.
**Diagnosis**
- Often detected during prenatal US, associated with high alpha-fetoprotein (AFP).
- Defect is usually to the right of umbilicus.
- Not usually associated with other anomalies.

**Treatment**
- Temperature regulation.
- Sterile covering with plastic wrap (Silo) to minimize evaporative loss.
- NG decompression.
- Broad-spectrum antibiotics.
- Total parenteral nutrition (TPN).
- Surgical correction and closure of abdomen.

**Prognosis**
- Greater than 90% survival rate, postsurgical.
- Twenty percent of cases are complicated by necrotizing enterocolitis (NEC).

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**omphalocele**

**Definition**
- Herniation of abdominal contents (often including the liver) into the base of the umbilical cord.
- Protective membrane present.
- Elements of the umbilical cord course individually over the sac and come together at its apex to form a normal-appearing umbilical cord.

**Epidemiology**
- One in 5,000 live births.
- More common in babies born to mothers < 20 years old and > 40 years old.

**Associated Anomalies**
- Beckwith–Wiedemann syndrome (gigantism, macroglossia, umbilical defect, hypoglycemia).
- Trisomy 13 and 18.
- Pentalogy of Cantrell (omphalocele, diaphragmatic hernia, cleft sternum, absent pericardium, intracardiac defects).
- Exstrophy of the bladder or cloaca.

**Types**
- Small: Contains only intestine.
- Large: Contains liver, spleen, and gastrointestinal (GI) tract.

**Treatment**
- Ruptured sac:
  - Similar to gastroschisis treatment.
  - Emergent surgical correction.
- Intact sac:
  - Less urgent.
  - Timing of surgery depends on size of defect, size of infant, and presence of other anomalies.

---

**WARD TIP**
No difference has been shown in outcomes for gastroschisis or omphalocele whether infant is delivered vaginally or via C-section.

**WARD TIP**
Seven percent have coexistent intestinal atresia—important to search for this at surgery.

**WARD TIP**
Ruptured omphalocele may be confused with gastroschisis, but the former does not have an intact umbilical cord at the level of abdominal wall.
**Prognosis**
Dependent on presence of other anomalies.

**Inguinal Hernia**

**Definition**
Protrusion of a viscus or part of a viscus through an abnormal weakening in the abdominal wall and through the inguinal canal. In children, most commonly an indirect inguinal hernia from a processus vaginalis that has remained patent.

**Epidemiology**
- One to 5% of newborns.
- Higher incidence in premature infants (30%).
- Indirect hernias are more common on the right side because of the right testicle descends later.

**Signs and Symptoms**
Bulge in the groin, scrotum, or labia, especially with increased intra-abdominal pressure (coughing, crying, straining, and blowing up a balloon).

**Diagnosis**
- Physical exam shows firm mass that can slip through the internal inguinal ring.
- Important to differentiate from a hydrocele, which does not extend to the internal ring.
- “Silk glove sign”: After hernia is reduced, sac and thickened spermatic cord can be palpated and rolled underneath the examiner’s finger.

**Treatment**
- Operative repair:
  - Should be repaired shortly after diagnosis except in premature infants.
  - Unlike adults, pediatric cases are usually not repaired with a mesh. In pediatrics, high ligation of the hernial sac (herniotomy) is performed without repair of the abdominal wall.
  - Reasons to operate:
    - Major risks include incarceration of a loop of bowel, an ovary, or a fallopian tube.
    - Fifteen to 30% of hernias are incarcerated.
    - Increased risk of incarceration in first few months of life.

**Gastrointestinal Defects**

**Pyloric Stenosis**

**Definition**
Narrowing of the pyloric canal due to hypertrophy of the musculature.

**Epidemiology**
- One in 250 births.
**causes**
Unknown.

**Signs and Symptoms**
- Usually evident between 2 weeks and 2 months old.
- Nonbilious, projectile vomiting.
- Hungry after vomiting.
- Dehydration.
- Midepigastric mass ("olive").
- Visible peristaltic wave (left to right).
- Hypochloremic metabolic alkalosis with paradoxical aciduria.

**diagnosis**
- Ultrasound (90% sensitivity):
  - Elongated pyloric channel (> 14 mm).
  - Thickened pyloric wall (> 4 mm).
- Radiographic contrast series:
  - *String sign*—from elongated pyloric channel.
  - Shoulder sign—bulge of pyloric muscle into the antrum.
  - Double track sign—parallel streaks of barium in the narrow channel.

**treatment**
- Correction of fluid and electrolyte and acid–base balance.
- IV fluid 5% dextrose in normal saline plus potassium chloride 3–5 mEq/kg.
- Surgical correction: Ramstedt pyloromyotomy—dividing the circular fibers of the pylorus without entering the gastric lumen.

**Biliary Atresia**

**definition**
Obliteration of the entire extrahepatic biliary tree at or above the porta hepatis.

**Signs and Symptoms**
- Neonatal jaundice (beyond first week).
- Hyperbilirubinemia.
- High \( \gamma \)-glutamyl transpeptidase (GGT) level.
- Signs of portal hypertension (hepatosplenomegaly, ascites).

**diagnosis**
- Radioisotope scanning.
- Ultrasound.
- Direct bilirubin:
  - Greater than 2 mg/dL.
  - Greater than 10% of total bilirubin.

**treatment**
- Laparotomy, liver biopsy, and operative cholangiography should be done in any suspicious case.
- Correctable type:
  - Blind-ending cystic dilation of the common hepatic duct.
  - Repaired by direct anastomosis with Roux-en-Y loop of jejunum.
Noncorrectable type: Kasai procedure—hepatoportoenterostomy (anastomosis between the porta hepatis and the small intestines to drain bile from liver).

Postoperative treatment:
- Prophylactic antibiotics.
- Phenobarbital.
- Liver transplantation.

**Malrotation and Midgut Volvulus**

**Definition**
- Incomplete rotation of the intestine during fetal development.
- May cause complete or partial duodenal obstruction.

**Embryology**
- Midgut = duodenum to mid-transverse colon.
- Develops extraperitoneally and migrates intraperitoneally at 12 weeks.
- During this migration, the midgut rotates 270° counterclockwise around the superior mesenteric artery (SMA).
- Problem results from abnormal fixation of the mesentery of the bowel.

**Signs and Symptoms**
- Acute onset of bilious vomiting.
- Abdominal distention.
- Lethargy.
- Skin mottling.
- Hypovolemia.
- Bloody stool (late sign).

**Diagnosis**
- AXR:
  - Presence of bowel loops overriding liver.
  - Air in stomach and in duodenum (double bubble sign).
  - No gas in GI tract distal to volvulus.
- Upper GI series: Duodenal C-loop does not extend to the left (stops at level of duodenum).
- Barium enema: Cecum located in right upper quadrant (RUQ).

**Treatment**
- Surgical emergency (can lead to intestinal infarction and death if surgery is delayed).
- Ladd procedure:
  - Reduced with counterclockwise rotation.
  - Ligation of Ladd’s bands (abnormal fibrous bands attached to cecum and causing obstruction).
  - Appendectomy—because cecum remains in RUQ, changing position of appendix and making appendicitis harder to diagnose.

**Prognosis**
- Ten percent chance of recurrent volvulus.

**Intestinal Atresia**

**Definition**
- Failure of the duodenum to recanalize during early fetal life.
associated conditions
- Down syndrome.
- Esophageal atresia.
- Imperforate anus.
- Small for gestational age.
- Polyhydramnios.

Signs and Symptoms
- Bilious vomiting.
- Abdominal distention.

diagnosis
Plain abdominal film:
- Dilated bowel proximal to obstruction.
- “Double bubble” sign (air in stomach and duodenum).

treatment
- Fluid resuscitation.
- Gastric decompression.
- Broad-spectrum antibiotics.
- Duodenal atresia: Side-to-side anastomosis (avoids injury to bile and pancreatic duct).
- Jejunoileal atresia: End-to-end anastomosis.

intussusception

definition
Invagination of one portion of the bowel into itself—proximal portion usually drawn into distal portion by peristalsis.

epidemiology
- Incidence 1–4 in 1,000 live births.
- Peak incidence 5–12 months.
- Age range: 2 months–5 years.

causes
- Idiopathic.
- “Lead point” (or focus) caused by:
  - Hypertrophied Peyer’s patches from viral infection (enterovirus in summer, rotavirus in winter).
  - In older children:
    - Meckel’s diverticulum.
    - Polyp.
    - Lymphoma.
    - Henoch–Schönlein purpura.
    - Cystic fibrosis.

Signs and Symptoms
- Classic triad:
  - Intermittent colicky abdominal pain.
  - Bilious vomiting.
  - Currant jelly stool.
  - Dance’s sign—absence of bowel in right lower quadrant (RLQ) with a diffuse sausage shaped RUQ mass.

WARD TIP
Passage of meconium does not rule out intestinal atresia.

WARD TIP
Differential diagnosis includes volvulus and annular pancreas.

WARD TIP
Since malrotation/volvulus has the same radiographic double bubble sign, get upper GI for confirmation.

EXAM TIP
Most common cause of acute intestinal obstruction under 2 years of age.
Most common site is ileocolic (90%).

EXAM TIP
Intussusception and link with rotavirus vaccine led to withdrawal of vaccine from the market.

intussusception
- Classic triad is present in only 20% of cases.
- Absence of currant jelly stool does not exclude the diagnosis.
- Neurologic signs may delay the diagnosis.
**Diagnosis**

- **AXR:**
  - Paucity of bowel gas.
  - Loss of visualization of the tip of liver.
  - “Target sign”—two concentric circles of fat density.
- **US:**
  - “Target” or “donut” sign—single hypoechoic ring with hyperechoic center.
  - “Pseudo-kidney” sign—superimposed hypoechoic (edematous walls of bowel) and hyperechoic (areas of compressed mucosa) layers.
  - Barium enema.

**Treatment**

- Correct dehydration.
- NG tube for decompression.
- Hydrostatic reduction.
- Barium enema:
  - Cervix-like mass.
  - Coiled spring appearance on the evacuation film.
- Contraindications:
  - Peritonitis.
  - Perforation.
  - Profound shock.
- Air enema:
  - Decreased radiation.
  - Fewer complications

**Recurrence**

- With radiologic reduction: 7–10%.
- With surgical reduction: 2–5%.

**Meckel’s Diverticulum**

**Definition**

Persistence of the omphalomesenteric (vitelline) duct (should disappear by seventh week of gestation):

- Arises from the antimesenteric border of ileum.
- Contains heterotopic epithelium (gastric, colonic, or pancreatic).
- A true diverticulum in that it contains all layers of bowel wall.

**Epidemiology**

- Two percent incidence.
- More common in males.
- Usually presents in children < 2 years old.

**Signs and Symptoms**

- Intermittent painless rectal bleeding.
- Intestinal obstruction.
- Diverticulitis.

**Diagnosis**

Meckel’s scan (scintigraphy) has 85% sensitivity and 95% specificity. Uptake can be enhanced with cimetidine, glucagon, or gastrin.
Treatment
Surgical: Diverticular resection. If base is thickened then segmental resection of small bowel adjacent to the diverticulum.

Imper for a te anus
Lack of an anal opening of proper location or size.

Causes
Results from a failure of the urinary and hindgut systems to separate.

Associated anomalies
VACTERL anomalies (Vertebral, Anorectal, Cardiac, TracheoEsophageal malformations, Renal, Limb).

Types
High and low: Classification depends on whether the rectum ends above (high) or below (low) the puborectalis sling.

Treatment
- Colostomy for high lesions.
- Perineal anoplasty or dilatation of fistula for low lesions.

Prognosis
The higher the lesion, the poorer the prognosis.

Hirschsprung’s Disease (congenital aganglionosis coli)
Definition
Congenital absence of ganglion cells in the Auerbach (myenteric) and Meissner (submucosal) plexi that results in intestinal obstruction.

Epidemiology
- Occurs in 1 in 5,000–8,000 live births.
- Family history.

Types
- Rectal.
- Rectosigmoid.
- Entire colon.

Signs and Symptoms
In neonatal period:
- Ninety-five percent present as delayed passage of meconium (> 24 hours).
- Rectal examination.
- An empty vault that is not dilated.
- Explosive release of feces.
- Most ominous presentation is enterocolitis (most common cause of mortality in Hirschsprung’s).

Meckel’s diverticulum may mimic acute appendicitis and also act as lead point for intussusception.

Hirschsprung’s disease is the most common cause of lower intestinal obstruction in the neonate.
Presentation later in childhood:
- Bilious vomiting.
- Chronic constipation.
- Abdominal distention.
- Failure to thrive.

diagnosis
- AXR to look for evidence of obstruction.
- Barium enema to look for transition zone (may not be present until 1–2 weeks of age).
- Rectal biopsy for definitive diagnosis (must include submucosa; samples are taken 1, 2, and 3 cm from dentate line, shows absence of ganglion cells).

treatment
Surgical repair:
- Temporary colostomy proximal to transition zone at diagnosis.
- Definitive repair when the infant is 6–12 months old.
- Closure of colostomy 1–3 months postoperatively.

ne o Ti zing enTer ocoLiTi s (nec)
definition
Serious intestinal injury and necrosis following a combination of vascular, mucosal, and toxic insults to a relatively immature gut.

epidemiology
- Occurs in 1–3 in 1,000 live births.
- Predominantly a disorder of preterm infants; incidence increases with decreasing gestational age.
- Increased incidence with congenital heart disease, severe intrauterine growth retardation (IUGR), sepsis, gastrochisis, and other neonatal disorders.

Signs and Symptoms
Presentation is similar to sepsis.
- Systemic:
  - Lethargy.
  - Feeding intolerance.
  - Fever.
  - Hypothermia.
  - Hypotension.
  - Apneic spells.
- GI:
  - Abdominal distention, tenderness.
  - Vomiting.
  - Bloody diarrhea.
  - Hematochezia.

diagnosis
- AXR: Pneumatosis intestinalis (gas within the bowel wall).
- Lateral decubitus or cross-table lateral:
  - Probable NEC:
    - Thickened bowel loop.
    - Fixed position loops on serial films.
    - Ascites.
Definite NEC:
- Intramural gas.
- Portal gas.
- Pneumoperitoneum/free air (denotes perforation).

**Treatment**
- Nothing by mouth.
- Bowel decompression.
- Antibiotics/sepsis evaluation.
- TPN.
- Monitor vital signs and abdominal girth.
- Monitor fluid intake and output.
- Definite indication for surgical resection:
  - Perforation.
  - Full-thickness necrosis of bowel.
- Possible indications:
  - Ascites.
  - Deterioration with medical management.
  - Intestinal obstruction.

**Prognosis**
Variable (depends on extent of injury). Overall > 95% survival rate.

### Neoplastic Disease

**Wilms’ Tumor**

**Definition**
Nephroblastoma—originates intrarenally.

**Epidemiology**
- Most common intra-abdominal malignancy in childhood.
- Usual presentation between 2 and 4 years; very rapidly growing.

**Associated Conditions**
- WAGR syndrome (Wilms’ tumor, Aniridia, Genitourinary anomalies, mental Retardation).
- Beckwith–Weidemann syndrome (hemihypertrophy, macroglossia, organomegaly).
- Denys–Drash syndrome (Wilms’ tumor, pseudohermaphroditism, and glomerulopathy).

**Signs and Symptoms**
Triad:
- Flank mass.
- Hematuria.
- Hypertension.

**Diagnosis**
- CT scan with contrast (metastasis—nodal enlargement and liver nodule).
- CXR or CT scan (lung metastasis).
- Intravenous urography (intrarenal solid mass).
- Ultrasound of abdomen (extension into renal vein and inferior vena cava [IVC]).
**Stages**

I: Involves only kidney.
II: Invades capsule and possibly perirenal fat.
III: No hematogenous metastasis, not completely resectable.
IV: Hematogenous metastasis to lung, brain, distal nodes.
V: Involves both kidneys.

**Treatment**

- Surgical resection of tumor, exploration of abdomen, evaluation of contralateral kidney.
- Chemotherapy + radiotherapy (for later stages).
- Ninety percent survival rate.

**Neuroblastoma**

**Definition**

- Arises from neural crest cells.
- May arise in adrenal medulla, sympathetic ganglia, and organ of Zuckerman (para-aortic ganglia).

**Epidemiology**

- Most common solid tumor in infants (>33% < 1 year old, 80% < 5 years old).
- Most common solid tumor of childhood outside of central nervous system.
- Abdominal tumors are most common presentation: 65% in adrenal gland.
- Thoracic tumors are next most common presentation.

**Signs and Symptoms**

- Abdominal pain and mass.
- Fever, failure to thrive, diarrhea, and lethargy.
- Neurological symptoms (ataxia, opsomyoclonus).
- Hypertension (25%).

**Diagnosis**

- Urine: Raised catecholamines (vanillylmandelic acid [VMA], homovanillic acid [HVA]).
- Intravenous pyelography (inferior displacement of opacified calyces—“drooping lily sign”).
- CT abdomen with contrast.

**Stages**

I: Tumor is limited to one organ, completely resectable.
II: Tumor does not cross midline.
III: Crosses midline.
IV: Distant metastases.

**Treatment**

- Stage I: Resection.
- Stages II and III: Resection and chemotherapy.
- Stage IV: Resection and aggressive chemo/radiotherapy.
AnATOMY

External Ear
See Figure 22-1.

- Auricle: Funnels sound waves into external auditory canal (EAC).
- External auditory canal: The outer one third is cartilaginous; the inner two thirds is osseous.
- Tympanic membrane (TM) is divided into two parts: Pars flaccida superiorly and pars tensa inferiorly. TM is attached to the malleus medially, and conducts sound waves from external ear to middle ear.

Middle Ear
Contains the eustachian tube orifice, ossicles, and entrance to the mastoid cavity:

- The eustachian tube connects the nasopharynx to the anterior wall of the middle ear cavity. It serves to equalize middle ear pressure to atmospheric pressure. The adenoids are in close proximity to nasopharyngeal opening of tube.
- Ossicles: Tympanic membrane → malleus → incus → stapes → oval window → perilymph of scala vestibuli of the inner ear. The ossicles serve as a lever arm to amplify sound. The malleus is fused to the TM internally, and with the incus medially. Tensor tympani muscle is attached to the manubrium of the malleus. The incus articulates with the other two ossicles. No muscular attachments. The neck of the stapes serves as the attachment for the stapedius muscle, innervated by the stapedial branch of cranial nerve (CN) VII. The footplate of the stapes is attached to the oval window.

Inner Ear
The inner ear labyrinth consists of a bony labyrinth encompassing the membranous labyrinth and organs of hearing and balance.

- Acoustic apparatus: Composed of the cochlea, which consists of the scala vestibuli and scala tympani, both of which are filled with perilymph. The
oval window transmits vibrations to the scala vestibuli at the base of the cochlea, which, via the helicotrema at the apex of the cochlea, transmits the pressure waves to the scala tympani. The scala tympani transmits the waves to the round window, where they are dissipated. The scala media, which contains the spiral organ of Corti, is the sensory portion of the inner ear and is filled with endolymph. The inner and outer hair cells in the organ of Corti transmit signals to the cochlear nerve (CN VIII). The neuroepithelium in the cochlea is arranged in a ribbon-like structure along the entire length of the cochlea, with tonotopic organization of sounds—higher frequencies are represented at the base of the cochlea, lower frequencies at the apex.

- **Vestibular apparatus:** Includes the vestibule (utricle and saccule) and semicircular canals, which provide input concerning gravitational pull/linear acceleration and angular acceleration, respectively. Cells in the neuroepithelial regions stimulate the vestibular nerve (CN VIII) and provide information about balance. The utricle and saccule as well as the semicircular ducts are filled with endolymph.

### Physiology

**Evaluation of hearing**

Sound waves can reach the cochlea in two ways:

- **Air conduction,** using the ear canal, TM, and middle ear structures. Pathology at any of these levels can decrease air conduction.
- **Bone conduction,** vibrating the temporal bone and therefore the cochlea, which lies within it, bypassing the external and middle ear structures; it depends on the integrity of cochlear structures, CN VIII, and central auditory pathways.

Causes of **conductive** hearing loss (CHL):

- Obstruction of external auditory meatus by cerumen, foreign body, or debris.
- Swelling of the EAC, neoplasms, or stenosis of the canal.
- Perforation of the TM.
- Ossicular discontinuity.
- Otosclerosis.
- Fluid, scarring, or neoplasms of the middle ear.

Causes of **sensorineural** hearing loss (SNHL):

- Damage to the hair cells of the organ of Corti by intense noise, baro-trauma, viral infections (e.g., human herpesvirus), or ototoxic drugs (e.g., aminoglycosides).
- Presbycusis (normal hearing deterioration with aging).
- Iatrogenic.
- Temporal bone fractures.
- Meningitis.
- Cochlear otosclerosis.
- Ménière’s disease (see later discussion).
- Acoustic neuromas (schwannomas).

**Functional Testing**

- **Weber test:** A 512-Hz fork is placed on the skull vertex; the patient is asked to which side the tone lateralizes. In CHL, the sound lateralizes to the affected ear. In SNHL, the sound lateralizes to the unaffected ear.
- **Rinne test:** Compares the ability to hear by air conduction to that of bone conduction. A vibrating tuning fork is placed on the patient’s mastoid process, and the patient is asked to indicate if they hear the sound and when it stops. The tuning fork is then placed at the EAC. Normally, sound is
heard louder by air conduction than by bone, called a positive Rinne test, but with CHL, bone conduction is louder, and thus the Rinne test is negative. With SNHL, this test is normal, although technically both bone and air sound perceptions are reduced.

**Pathology**

**otitis Media**
Acute otitis media (AOM), recurrent AOM, chronic otitis media.

**Definition**
- Inflammation of the lining of the middle ear cleft along with a bacterial infection.
- More common in children.
- Eustachian tube dysfunction and a preceding upper respiratory infection (URI) are the most documented etiologic actors precipitating AOM.
- Common bacterial pathogens include *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*.

**Signs and Symptoms**
Otalgia, ear pulling, and systemic symptoms including fever, anorexia, vomiting, and diarrhea.

**Diagnosis**
On otoscopic exam, the TM appears to be bulging, red, and immobile on pneumatic otoscopy.

**Treatment**
- Symptom control and analgesia, antibiotic therapy, and prevention of complications.
- For cases of recurrent AOM (more than three episodes of AOM in a 6- to 12-month period) antibiotic prophylaxis is recommended.
- A myringotomy with or without tympanostomy tubes may be recommended if the AOM is not responsive to antibiotics.

**Complications**
Complications of otitis media include TM perforation, hearing loss, and infection or inflammation of the temporal bone or intracranial cavity.

**otitis Media with Effusion (o ME)**

**Definition**
Fluid collection in the middle ear cleft without the pain and fever associated with AOM.

**Signs and Symptoms**
Children usually present with hearing loss and speech delay.

**Diagnosis**
Otoscopic examination reveals decreased TM mobility, opacity, and, occasionally, evidence of an air–liquid interface behind the TM (air bubbles).

**Treatment**
- There is a clear indication for treatment of children with chronic OME with *myringotomy and tympanostomy tubes* if:
  - There is evidence of change significantly decreased hearing to “conductive hearing loss.”
The child is at high risk for speech and language delay (craniofacial disorders, cleft palate).
- The child has a current delay.
- There are recurrent bouts of AOM superimposed on OME.
- There is evidence of structural changes to the TM.
- An adenoidectomy may be performed simultaneously in select patients to improve eustachian tube function.

**Otitis Externa**

**Definition**
- Generalized inflammation involving the EAC skin.
- Can be infectious or eczematous.
- Organisms include hemolytic Streptococcus, Candida, Staphylococcus, and Pseudomonas.
- **Malignant otitis externa** in immunocompromised patients (e.g., elderly diabetics) is usually caused by Pseudomonas spp. and can have more extensive complications (e.g., osteomyelitis).

**Signs and Symptoms**
- Ear pain, redness, tenderness, weeping, and otorrhea.
- It is often difficult to examine the ear due to pain.

**Treatment**
- Analgesia to control the pain.
- Topical antibiotics +/- topical steroid cream.
- Systemic antibiotics for severe cases only.

**Cholesteatoma**

**Definition**
- Benign but invasive tumor of the middle ear/mastoid caused by overgrowth of displaced keratinizing epithelium.
- Can be **acquired** or **congenital**.

**Signs and Symptoms**
- Foul otorrhea, aural fullness or pressure, hearing loss.
- May cause CHL, SNHL, or CN VII injury or have intracranial extension.

**Diagnosis**
- Diagnosis is made by noting an area of retraction filled with debris on otoscopic examination.
- Evaluation with computed tomography (CT) scan and audiologic testing.

**Treatment**
- Surgery is the definitive treatment, necessary to eradicate disease and prevent recurrence. It is sometimes performed in two stages to allow assessment for residual disease in the mastoid and middle ear (i.e., second-look procedure).

**Acoustic Neuroma (Vestibular schwannoma)**

**Definition**
- Benign tumor that arises in the cerebellopontine angle of the internal auditory canal (IAC).
- Associated with neurofibromatosis types I and II.
The most common symptom is progressive asymmetric SNHL, followed by tinnitus and balance disturbance.

Slow growing and nonmetastatic. They do, however, compress adjacent tissue as they grow, usually the vestibular and cochlear nerve within the IAC, but can compress the brain stem and facial nerve once they outgrow that space.

Audiogram shows unilateral SNHL.

Surgery or radiation therapy (RT): Surgical approaches include translabyrinthine, middle cranial fossa, and retrosigmoid/suboccipital.

The facial nerve innervates the muscles of facial expression; the digastric, stylohyoid, and stapedius muscles; as well as sensation to the anterior two thirds of the tongue and autonomic fibers for taste and to the salivary glands.

The circuitous path that the nerve takes has six segments: Intracranial, meatal, labyrinthine, tympanic, mastoid, and extratemporal.

It courses through the parotid gland on its way, and splits into five portions that innervate the face: Temporal, zygomatic, buccal, marginal mandibular, and cervical.

The posterior auricular branch comes off of the facial nerve before it splits into these five branches.

Trauma.
Iatrogenic injury.
Injury related to temporal bone fractures.
Viral infection (herpes zoster oticus [Ramsay Hunt syndrome]).
Lyme disease.
Bacterial infection (malignant otitis externa).
Systemic disease (Guillain–Barré syndrome, mononucleosis, sarcoidosis).

The most common form of paralysis.
Ten percent of patients experience recurrent paralysis.
Although the etiology is idiopathic, some suggest that the paralysis may result from a cycle of ischemic/inflammatory events of the nerve in the bony canal.
There is no degeneration of the nerve.

Occurs as a unilateral facial weakness of sudden onset that resolves spontaneously.
There should be no signs of concurrent central nervous system (CNS) or ear disease.
Sixty percent of patients experience a viral prodrome, and 60% experience facial, neck, or ear numbness or pain prior to the palsy. Symptoms usually improve within about 6 months, completely by 12 months.

**Diagnosis**

Evaluation includes a history and physical, an audiogram, and electrophysiologic testing of the nerve. If thought to be Bell’s palsy and not due to some other cause, radiologic imaging is not recommended.

**Treatment**

- A course of steroids early in the disease process help shorten recovery time and serve as an analgesic.
- Surgical intervention with decompression of the nerve remains controversial.

**Nose**

**Anatomy**

- **External structure**: Paired nasal bones, midline septum, paired upper and lower lateral cartilages, soft tissue.
- **Internal structure**: The nasal cavity opens to the face through the nares (nostrils). Its axis is at a right angle to the face. Posteriorly, the choana communicates with the nasopharynx.
  - The internal nose is divided into two sides, bound medially by the nasal septum, composed of multiple cartilages and bony structures.
  - The septum is covered by perichondrium and periosteum, which is then covered by nasal mucosa (respiratory epithelium).
  - The lateral border of the nasal cavities consists of three bony ridges called turbinates, which each cover an area called a meatus. Each meatus contains ostia, which drain the paranasal sinuses. The superior and middle turbinates are parts of the ethmoid bone; the inferior turbinate is an independent bone.
  - Four paranasal sinuses: Air spaces in the bony structure of the face that communicate with the nasal cavity via various ostia.
    - Paired maxillary sinuses laterally.
    - Ethmoids and sphenoids superoposteriorly.
    - Frontal sinuses superiorly.

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<tr>
<th>Structure</th>
<th>Opening</th>
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<td>Sphenoid recess</td>
<td>Sphenoid sinus</td>
</tr>
<tr>
<td>Superior meatus</td>
<td>Posterior ethmoidal air cells</td>
</tr>
<tr>
<td>Middle meatus</td>
<td>Frontal sinus, maxillary sinus, middle ethmoidal air cells, anterior ethmoidal air cells—osteomeatal complex</td>
</tr>
<tr>
<td>Inferior meatus</td>
<td>Nasolacrimal duct</td>
</tr>
</tbody>
</table>

**Blood supply**

Both the internal carotid system, via the anterior and posterior ethmoidal arteries, and the external carotid system, via the internal maxillary/sphenopalatine artery, supply the nasal cavity and paranasal sinuses. The facial artery also supplies the anterior nose.

**Nerve supply**

Sympathetic autonomic innervation has a vasoconstrictive effect; parasympathetic fibers function in vasomotor stimulation.
Physiology

- **Nasal respiration**: Humidification, warming, and filtration of inhaled air and substances.
- **Olfaction**.
- **Phonation**: Normal voice depends on contribution of nasal resonance; disorders can give rise to hypo- or hypernasal speech.

**Evaluation of the Nasal Cavity and Sinuses**

- **Anterior rhinoscopy**: Using a nasal speculum. Allows visualization of the anterior portion of the nose, septum, and turbinates.
- **Nasal endoscopy**: Using a flexible or rigid scope. Provides diagnostic information, with attention to color, edema, discharge of nasal mucosa; allows for visualization of masses or abnormalities. Also allows for visualization of the nasopharynx, oropharynx, and hypopharynx/larynx.
- **Imaging**: Plain radiographic imaging of sinuses is of limited value. CT imaging has better resolution of structures, can evaluate for mucosal disease, provides a good “road map” for surgery. Can evaluate for septal deviation, polyps, masses, the osteomeatal complex, ethmoid air cells.

### Sinus Disease

**Acute Bacterial rhinosinusitis**

**Definition**

- Infection of the paranasal sinuses/nasal mucosa, usually preceded by a URI, allergy, trauma, or dental infection.
- Impaired immune function predisposes to recurrent disease.
- Usually bilateral.
- Causative organisms include S. pneumoniae, H. influenzae, and M. catarrhalis.

**Signs and Symptoms**

Diagnosed after persistence of viral infection beyond 10 days, with severe or worsening symptoms, including maxillofacial pain, fever, dental pain, otalgia, malaise, and increased nasal drainage.

**Treatment**

- Medical management is with antibiotic therapy. Middle meatus culture or maxillary sinus aspiration can guide antibiotic treatment.
- Adjunctively, relief is provided with nasal saline irrigation, systemic decongestants, topical decongestants, and antihistamines if there is an allergic component.
- For treatment failure, a more comprehensive diagnostic workup is warranted, including imaging.

**Chronic sinusitis**

**Definition**

Defined by sinusitis symptoms for > 3 months.

**Management**

- Patient receives maximal antibiotic therapy for 4–6 weeks prior to imaging studies, which often demonstrates anatomic variants predisposing the patients to osteomeatal compromise. Medical therapy often fails, and surgical therapy is necessary.
- **Functional endoscopic sinus surgery (FESS)** is the procedure of choice to promote natural drainage and aeration of the sinuses by opening up the osteomeatal complex.
**Epistaxis**

**Etiology**

**Local Causes**
- Trauma: Nose picking, external trauma, dry mucosa (common in the winter months).
- Barometric pressure changes.
- Sinusitis, URI, allergy.
- Neoplasia.
- Septal perforations (with cocaine use).

**Systemic Causes**
- Systemic disease: Hypertension, renal disease, hepatic failure, heavy alcohol use, granulomatosis with polyangiitis (aka Wegener’s).
- Hematologic disease: Hemophilia, coagulopathy, thrombocytopenia.
- Medication related: Anticoagulants, NSAIDs.

**Assessment and Treatment**
- Identify anterior vs. posterior and left vs. right bleeds by routine nasal speculum examination.
- Manage bleed based on severity and location. Decongestant and topical anesthetics help decrease the bleeding and allow for better visualization.
  - Anterior bleeds can usually be controlled with local pressure, pinching the tip of the nose with or without silver nitrate or electrocautery. An anterior nasal pack can be used as well, absorbable vs. nonabsorbable packing material.
  - A posterior bleed may require more vigorous management—posterior packing. The bleeding source can be more easily identified by nasal endoscopy.
- Determine the etiology of the bleed based on history and physical exam (coagulopathy, trauma, neoplasia) and manage that as necessary.
- Failure of appropriately placed packing with continued bleeding may require surgical therapy or sphenopalatine, anterior ethmoidal, or posterior ethmoidal artery ligation.
- Selective arterial embolization by an interventional radiologist is extremely effective as well, however, does carry a low risk for cerebral emboli.

**Nasal Masses**

**Benign Masses**
- **Inflammatory polyps:**
  - Treatment of choice is topical corticosteroids.
  - Systemic steroids for severe cases.
  - Surgical options for disease refractory to medical management include nasal polypectomy and FESS.
- **Antrochoanal polyps:**
  - Treatment is surgical removal. (Most benign masses tend to recur despite surgical excision)

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**WARD TIP**

Ninety-five percent of bleeding occurs in the anterior nasal cavity, and ~90% of those are from Kesselbach’s plexus, a confluence of vessels at the anterior septum. These vessels are prominent and most subject to drying, mechanical trauma, and exposure to irritants.

**WARD TIP**

When dealing with posterior nasal bleeding, hemostasis can be achieved by inserting a Foley catheter into the patient’s nose, inflating the balloon, and applying traction.

**EXAM TIP**

Nasal polyps in an infant/young child should raise suspicion of cystic fibrosis—do sweat chloride test.
- **Juvenile nasopharyngeal angiofibroma (JNA):**
  - Treatment is primarily surgical removal.
  - Preoperative embolization can be performed within 72 hours prior to surgery to reduce blood loss.
- **Inverting papilloma** (~50% of nasal respiratory papillomas):
  - A benign nasal tumor that arises from the respiratory mucosa, associated with human papillomavirus (HPV), strains 6 and 11.
  - It is called inverting because the epithelium usually invades surrounding stromal tissue.
  - Treatment is with surgical resection, usually endoscopically.
  - There is a 5–10% chance of malignant transformation to squamous cell carcinoma (SCC).

**Malignancies**

- **Carcinoma:**
  - Nasal carcinoma accounts for between 27% and 35% of head and neck cancer.
  - Exposure to irritants and chemicals is associated with specific nasal carcinomas.
  - Treatment by en-bloc resection via a craniofacial approach is choice; however, due to extent and spread of the cancer, it is often limited.
  - RT is often used as an adjuvant, with chemotherapy for palliation.
- **Lymphoma:**
  - The most common nonepithelial malignancy of the nose.
  - Usually associated with Epstein–Barr virus (EBV); more common in Asia.
  - Radiologic evaluation demonstrates opacification of the sinuses, bony erosion, and occasionally a mass.
  - Fresh biopsy sections need to be sent during surgical removal in order to identify the cancer.
  - Combined RT and chemotherapy is the preferred method of treatment, as there is a risk of distant recurrence with RT alone.
- **Melanoma:**
  - Nasal melanoma accounts for ~1% of all melanomas.
  - Patients are usually over the age of 60.
  - On nasal endoscopy, the lesions may appear as a benign polyp, or a dark, fungating neoplasm.
  - Treatment is by surgical resection, with a 5-year survival of about 30%.

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### Oral Cavity and Oropharynx

**Anatomy**

- The oral cavity begins at the vermilion border of the lips, ends at the junction of the soft and hard palate superiorly and at the circumvallate papillae of the tongue.
- It includes the lips, buccal mucosa, superior and inferior alveolar ridges, part of the tongue, hard palate, and floor of the mouth.
- The salivary ducts terminate into the oral cavity, including the parotid Stensen’s ducts lateral to the second maxillary molars, the submandibular Wharton’s duct onto the floor of mouth, along with multiple sublingual duct orifices that drain into the floor of mouth as well.
- The tongue is included in the oral cavity; the frenulum is a fold of mucosa that is anteriorly attached to the tongue that attaches it to the floor of mouth mucosa.
The oropharynx begins at the junction of hard and soft palate superiorly and the circumvallate papillae on the tongue, and extends to the valeculae. The oropharynx contains a ring of lymphoid tissue called Waldeyer’s ring. It consists of the adenoids superoposteriorly, the palatine tonsils laterally, and the lingual tonsil at the base of the tongue.

**Blood supply**
- The palate is supplied by small branches of the descending palatine artery, a branch of the maxillary artery.
- Vascular supply to tongue is via the lingual artery (a branch of the external carotid) and respective vein.
- The tonsils are supplied by the tonsillar and ascending palatine branches of the facial artery, by a branch of the lingual artery, by the ascending pharyngeal artery, and the lesser descending palatine branch of the maxillary artery.

**Nerve supply**
- The anterior two thirds of the tongue has somatic sensory innervation from CN V₃, and taste is via the chorda tympani (CN VII). The posterior one third of the tongue is innervated by CN IX (gag reflex) for sensory and taste.
- The muscles of the pharynx are innervated by CN X, except for the tensor veli palatini, which is innervated by CN V₃, and the stylopharyngeus, which is innervated by CN IX. Sensory innervation to the pharynx is by CN V₂, IX, and X.
- Sensory innervation to the oral cavity is via V₃.

### Physiology
- The primary function of the oral cavity and oropharynx is mastication of food and food delivery to more distal structures.
- Saliva in the mouth lubricates and begins to digest the food.
- The tongue, lips, muscles, and palate move the bolus of food posteriorly into the oropharynx and then down to the esophagus.
- Once the bolus reaches the oropharynx, voluntary control of swallowing is switched to involuntary control to propel the bolus quickly past the closed glottis to the esophagus.
- During this time, the nasopharynx closes with palate elevation, and respiration must cease.

### Peritonsillar abscess
**Definition**
- Located deep to the tonsillar capsule, usually at the superior pole.
- May be preceded by a pharyngitis.

**Signs and Symptoms**
- Presents with fever, deviation of the tonsil and uvula, pain, soft palate swelling, trismus, and a hot potato voice.

**Diagnosis**
- Clinical examination.

**Complications**
- Parapharyngeal abscesses if the infection spreads beyond the superior constrictor muscle, bacteremia, endocarditis, mediastinitis, airway obstruction, and aspiration pneumonia.

**Treatment**
- Needle drainage, incision and drainage, or tonsillectomy.

**WARD TIP**
Indications for tonsillectomy: Recurrent tonsillitis (6/year × 1 year, 5/year × 2 years, 3/year × 3 years), upper airway obstruction due to tonsillar hypertrophy (obstructive sleep apnea), recurrent peritonsillar abscess, malignancy.
definition
- A mucocele of the sublingual gland that presents as a cystic mass on the floor of mouth.
- It is called a “plunging ranula” when it penetrates the mylohyoid muscle, and presents as a soft submental neck mass.

Treatment
Excision should include the entire sublingual gland to prevent recurrence, with care taken dissecting around the lingual nerve and Wharton’s duct.

oral Cavity Carcinoma
Definition
- Carcinoma of the lip, tongue, floor of mouth, gums, palate.
- Risk factors include tobacco smoke, pipes, betel nut chewing, alcohol, and sunlight exposure (cancer of the lip).
- Mostly SCC.

Signs and Symptoms
May present with leukoplakia or erythroplakia, mass or ulceration, or as symptoms due to the invasion of other structures (loose teeth, trismus).

Diagnosis
Assessment involves a biopsy, usually in the OR, along with panendoscopy to look for a second primary tumor. Patients also have a CXR and adjunctive imaging to assess the neck and evaluate extent of invasion. Staging with tumor-node-metastases (TNM) system.

Treatment
- Early stages are usually treated surgically.
- When advanced, combined modalities are used.
- The oral cavity has a rich lymphatic supply, so elective neck dissection is often recommended.
- Therapeutic neck dissection is performed when there is clinically apparent nodal disease.

oropharyngeal Carcinoma
Definition
- Ninety percent SCC, keratizing or nonkeratizing (often with submucosal spread).
- Lymphocytic malignancies include lymphoma of the palatine tonsil and base of tongue. May be the first symptom of a systemic lymphoma.

Signs and Symptoms
Jugulodigastric and submental lymph node groups are most commonly affected.

Diagnosis
- Physical examination includes careful assessment of the pharynx and larynx, as second primary tumors are common.
- Use bimanual palpation (especially for base of tongue), nasal endoscopy, and panendoscopy under sedation.
Treatment by RT is preferred, resulting in less morbidity and functional disturbance.
- Combined modalities, including surgery, chemotherapy, and RT are sometimes used as well, depending on where the lesion is (tonsil vs. base of tongue).
- Patients have an increased risk for distant metastasis; large bulky tumors and nodes should be followed closely and carefully for signs of local, regional, and distant recurrence.

**Larynx and Hypopharynx**

**Anatomy**
- Cartilaginous skeleton includes the cricoid, thyroid, arytenoids, corniculate, and cuneiform cartilages.
- The cricoid cartilage forms a complete ring.
- Superiorly, the hyoid bone forms an important framework for the larynx below it, as it is a major point of attachment for the extrinsic muscles of the larynx (see Figures 22-2 and 22-3).
- The laryngeal cavity is divided by the true vocal cords (TVCs):
  - Above them is the supraglottis, from the tip of the epiglottis to the apex of the ventricle, halfway between the true and false vocal cords. It includes the epiglottis, arytenoids, aryepiglottic folds, and false vocal cords.
  - The glottis is composed of the vocal folds—the middle of the laryngeal ventricle to 1 cm below the vocal folds.
  - The subglottis begins 1 cm below the TVC to the inferior edge of the cricoid cartilage.
- Muscle groups of the larynx are described as either:
  - **Extrinsic**: Depressors and elevators of the larynx, including the stenohyoid, thyrohyoid, omohyoid, geniohyoid, digastric, mylohyoid, and stylohyoid, innervated by cervical nerves, CN V, and CN VII.
  - **Accessory**: Pharyngeal constrictor muscles.
  - **Intrinsic**: Thyroarytenoid, thyroepiglottic, aryepiglottic; muscles of the arytenoids cartilage including the interarytenoid, posterior cricoarytenoid, and lateral cricoarytenoid muscles, which are responsible for abducting and adducting the vocal cords; and the cricothyroid muscle.

*Figure 22-2. Sagittal section of the larynx demonstrating anatomic divisions of the larynx. (Reproduced, with permission, from Lee KJ. Essential Otolaryngology: Head & Neck Surgery. 8th ed. New York: McGraw-Hill; 2003: 597.)*
Lymphatic drainage
- Drain mainly to the deep cervical group.
- The vocal cords themselves contain very few lymphatic channels, however the supra- and subglottic areas contain extensive lymphatic drainage (important in metastatic tumor spread).

Nerve supply
Two branches of the vagus nerve (CN X) supply the larynx:
- Superior laryngeal nerve: Divides into the external and internal branches.
  - External: Supplies motor fibers to the cricothyroid muscle only.
  - Internal: Sensory innervation to all areas of the larynx above the glottis.
- Recurrent laryngeal nerve: Due to embryonic development, the nerve descends into the thorax on the left side and passes under the aortic arch before it returns to the neck. Provides motor function to all of the intrinsic muscles of the larynx except for the cricothyroid muscle; sensory innervation to the laryngeal mucosa below the glottis.

Physiology
Functions of the larynx include:
- Phonation: The vocal cords act by vibrating. Intrinsic muscles determine the vibratory characteristics such as tension and contour of the vocal cords. The lateral cricoarytenoid and thyroarytenoid muscles adduct the cords, approximating the vocal processes and close the glottis during phonation; the posterior cricoarytenoid muscle primarily abducts the cords, thereby opening the glottis.
- Protection of the respiratory tract: The larynx acts as a sphincter—muscles elevate and close the larynx, determine epiglottis position, vocal cord adduction, cough reflex.
- Respiration: Muscular dilatation of the laryngeal aperture.
**Evaluation of the Larynx**

Indirect laryngoscopy:
- **Mirror examination**: Noninvasive but limited exam of the larynx, headlight illumination of a laryngeal mirror held up against the uvula, reflects an image of the larynx.
- **Flexible fiberoptic examination**: Through the nose.
- **Videostroboscopy**: A large-bore, rigid, 90° fiberoptic endoscope. A camera inside the endoscope continually flashes at a predetermined speed, allowing assessment of vocal fold symmetry, the mucosal wave, and subtle lesions.
- **Direct laryngoscopy**: Done in the OR under general anesthesia. A rigid laryngoscope is inserted; one can perform procedures through the scope (biopsy, laser).

**Benign lesions of the vocal cords**

**Polyp**

**Definition**
- A pedicled or sessile lesion that occurs on the vocal cords.
- They vary in size and appear as a smooth, glistening body.
- Can overhang the vocal cords, and may be evident on examination only when the patient is asked to cough and the polyp arises from the undersurface of the cords.

**Signs and Symptoms**
- Presents as long-standing hoarseness.
- Occasionally, they can be large and cause stridor and airway obstruction.

**Treatment**
- Definitive treatment is by lifestyle modifications (smoking, voice abuse), endoscopic removal of the lesion, and stripping of the cords if lesions are bilateral.
- If airway obstruction occurs, tracheotomy or laser debulking of the lesions may be necessary.

**Nodule (“singer’s Nodules”)**

**Definition**
- Localized traumatic laryngitis caused by vocal overuse (screaming, harsh talking, faulty singing technique), allergy, URI, sinusitis, smoking, and alcohol.
- Site of injury is usually the epithelium and basement membrane of the vocal cords.

**Signs and Symptoms**
- Usually presents as hoarseness.
- On exam, nodules are commonly found at the junction of the anterior and middle third of the vocal cords, the area of maximum vibration of the cords.
- Usually bilateral.

**Treatment**
- Behavioral modification, vocal reeducation, voice rest, and therapy.
- Microlaryngoscopic excision if nonresolving.
Papilloma

Definition
- The most common benign tumor of the larynx.
- Bimodal age distribution.
- Causative agent thought to be HPV. Certain subtypes of the virus can undergo malignant transformation.
- Can involve any region of the larynx and trachea, as well as the lungs.

Symptoms
- Aphonia or a weak cry in infants, dyspnea and stridor, hoarseness.

Treatment
Treatment includes CO₂ laser excision, microdebridement, therapy with interferon, intralresional cidofovir injection, topical application of mitomycin C (an antineoplastic).

Laryngeal Cancer

Definition
- Malignant tumors of the larynx and pharynx are usually SCC.
- The most common site for carcinoma in the upper aerodigestive tract.
- As there is an extensive lymphatic supply to the supra- and subglottic regions of the larynx, there is well-defined metastasis to the lymph nodes of the jugular chain or paratracheal regions. The exception to this rule is glottic carcinoma, which presents earlier and has a small likelihood of lymphatic metastasis (owing to the poor lymphatic drainage of the glottis).
- Direct spread and invasion by tumors can occur as well.
- The etiologic factors for most laryngeal SCC are tobacco smoke and alcohol exposure, which act synergistically.

Signs and Symptoms
- The presentation of laryngeal cancer is usually progressive hoarseness due to interference with the vocal cords.
- Other symptoms include dyspnea, dysphagia from pharyngeal involvement, and referred ear pain (an ominous sign).

Diagnosis
- Laryngoscopy must be done on a patient who has a persistent hoarse voice for > 2 weeks if over 40, and after 3–4 weeks if younger, assessing for asymmetry and the extent of any lesions.
- Tumors of the cords appear to be raised and warty in appearance; those of the supraglottis are usually exophytic.
- Vocal cord mobility must be noted.
- The neck is examined for metastatic lymphadenopathy.
- Diagnosis can occur after assessment of the larynx, pharynx, and oral cavity by direct microlaryngoscopy under general anesthesia.
- Depth of invasion and extent of the lesion need to be determined to stage the lesion (TNM classification) and guide treatment.
- Biopsies are taken.

Treatment
- Depends on stage and patient characteristics.
- It is either definitive removal or palliative.

WARD TIP

Tracheotomy vs. Tracheostomy
Tracheotomy: A temporary opening in the trachea; indicated for mechanical obstruction at or above the larynx, difficulty with clearing excessive secretions, airway protection during radical neck surgery, prolonged need for mechanical ventilation. These patients have intact oral airways and can be intubated through the orotracheal or tracheal approach (tracheostomy: more permanent).

Laryngectomy: A radical procedure to remove the larynx; indicated for advanced and recurrent laryngeal cancer. The trachea is rerouted to terminate at a stoma in the neck, and there is no longer patency between the oral cavity and the trachea. A patient like this cannot be intubated orally—must be intubated through their tracheal stoma.
Early tumors are treated by a single modality, usually radiotherapy, and more complicated cases often with a laryngectomy (partial or total depending on the disease state) and postoperative RT.

Options are available for voice reconstruction (a tracheoesophageal one-way valve) and return of swallowing that allow these patients to have a good quality of life.

Salivary Glands

An Atomy

Parotid
- Largest salivary gland; enclosed by deep cervical fascia. Composed of both deep and superficial lobes, separated by a plane of the facial nerve.
- Drains lateral to the second upper molar via Stensen’s duct.
- The facial nerve passes through the gland, and divides into its five branches.
- Parasympathetic supply originates in the inferior salivatory nucleus, travels with CN IX to Jacobsen's nerve to the otic ganglion; synapses and fibers are carried on the auriculotemporal nerve of CN V3.
- Sympathetic fibers come from the superior thoracic nerves and synapse with the superior cervical ganglion. They travel via arterial plexuses and sensory nerves to the gland.

Submandibular
- Drains into the mouth via Wharton’s duct, which courses between the sublingual gland and hyoglossus muscle; opens through a small opening lateral to the frenulum on the floor of mouth.
- Parasympathetic supply originates in the superior salivatory nucleus, travels via nervus intermedius of CN VII to the chorda tympani, which then joins the lingual nerve to the submandibular ganglion. Fibers synapse there and travel to the gland.
- Sympathetic supply is the same as for the parotid gland.

Sublingual
Smallest major salivary gland; lies in a submucosal location on the floor of mouth, and opens there through numerous small ducts called the ducts of Rivinius.

Minor salivary glands
Between 600 and 1,000 distributed all over the oral cavity and oropharynx, mostly on the hard and soft palate.

Physiology
- Parasympathetic stimulation increases saliva secretion; sympathetic slows it down.
- Saliva is high in potassium, low in sodium; it contains substances that begin the breakdown of food, to maintain and protect the oral cavity environment, and immunoglobulin A (IgA).
- Produce between 0.5 and 1.5 L of saliva/day.
**Radiographic Evaluation**
- CT/MRI is sometimes performed after injection of contrast medium directly into the ducts.
- Diagnostic ultrasound is also useful but has limitations.
- Fine-needle aspiration (FNA) for diagnosis of tumors vs. inflammatory lesions.

**Benign/systemic Disease**

**Sjögren’s syndrome**

**Definition**
- An autoimmune connective tissue disorder that is often associated with rheumatoid arthritis and systemic lupus erythematosus (SLE).
- Patients affected are usually middle-aged, menopausal women.

**Signs and Symptoms**
- Presents with keratoconjunctivitis sicca (dry, itchy eyes), xerostomia, taste changes, enlargement of salivary glands.

**Diagnosis**
- The diagnosis is made clinically: Measurement of salivary flow rates and salivary gland biopsy showing lymphocytic infiltrate and acinar atrophy confirming suspicions, in addition to relevant rheumatologic tests.

**Treatment**
- Treatment for xerostomia and dry eyes is symptomatic: Salivary and lacrimal substitutes.

**Sialolithiasis**

**Definition**
- Hydroxyapatite stones in the salivary glands/ducts.
- Most commonly affects the submandibular gland.
- More common in middle-aged men.

**Signs and Symptoms**
- Presents with pain and swelling in the affected area, with symptoms worsening prior to eating.

**Treatment**
- If the stone is in Wharton’s duct, it can be surgically removed transorally.
- If it is closer to the hilum of the gland, the gland may need to be removed.

**Sialoadenitis**

**Definition**
- Acute form is due to inflammation of the gland.
- Chronic form is a recurrent, painful enlargement of the gland, caused by salivary stasis.
- Occurs in debilitated and dehydrated patients, after major surgery, trauma, x-ray therapy (XRT).
- Infectious agent is typically Staphylococcus aureus.

**Signs and Symptoms**
- Erythema, pain, swelling, and purulent ductal discharge.
Treatment

- Acute form: Rehydration, sialogogues, warm compresses, and antibiotics.
- Chronic form: Medication to stimulate secretions, massages of the gland, and hydration.

**Mumps**

**Definition**
Infection with paramyxovirus in children, usually with a history of inadequate immunizations.

**Signs and Symptoms**
Bilateral painful parotid swelling, trismus, and malaise, along with other systemic manifestations (orchitis, pancreatitis, encephalitis).

**Diagnosis**
Measurement of antibody titers.

**Treatment**
This condition is usually self-limited, so supportive symptomatic treatment is recommended.

**Salivary gland Tumors**

- FNA may be a useful tool in diagnosis, especially for malignant lesions. CT/MRI imaging in conjunction, especially when planning for surgery.

Benign tumors

**Pleomorphic adenoma**
- Most common benign tumor; slight predilection for middle-aged women.
- Myoepithelial cells are thought to be the cell of origin.
- There is morphologic diversity: The tumor can be mucoid, chondroid, osseus, or myxoid.
- Slow growing, painless swelling of the gland, usually in the posterior region of the parotid gland.
- Rarely undergoes malignant transformation to carcinoma ex pleomorphic.
- Treatment: Parotidectomy.
- Care must be taken to expose the facial nerve proximal to where it enters the gland, and to follow it forward, dissecting tumor off of the nerve with care to preserve function.

**Monomorphic adenoma**
- A benign tumor with features similar to pleomorphic adenoma, but with only one morphologic cell type present.
- Slow growing and solitary, most often in the parotid gland.

**Warthin’s Tumor**
- AKA: Papillary cystadenoma lymphomatous.
- Men > women (5:1); usually in older individuals.
- Often presents as a benign, painless, compressible, slow-growing mass. Can be bilateral in 10%.
- Treatment: Excision of the involved gland.

**Oncocytoma**
- Benign tumor, mostly in the parotid; accounts for < 1% salivary gland tumors.
- Slow growing and circumscribed, not encapsulated.
Mucoepidermoid Carcinoma
- Most common malignant tumor of the salivary glands; derived from epithelial cells in the interlobar and intralobar epithelial cells of the gland.
- Can vary from low grade to highly malignant.
- Symptoms: Range from asymptomatic swelling (75%) to pain and facial nerve paralysis.
- Mostly affects the parotid.
- Most commonly induced by prior irradiation.
- Lymph node metastases are common in ~40% of patients.

Carcinoma Ex Pleomorphic
- Usually presents in patients who have undergone resection of a pleomorphic adenoma.
- Pathology shows remnants of benign mixed tumor.

adenocarcinoma
- Twenty percent of salivary tumors and only 4% of parotid tumors are adenocarcinomas.
- Eighty percent of patients are asymptomatic, although 20% have facial nerve paralysis and 15% facial pain due to fixation of the tumor to underlying/overlying structures.
- Arises from the terminal tubules and intercalated or strained duct cells in the gland.
- Many varieties have been described, and are graded as low, intermediate, or high.

adenoid Cystic Carcinoma
- Origin is the myoepithelial cell.
- Most common malignant tumor of the submandibular and minor salivary glands.
- Malignant but slow growing; most patients are asymptomatic on presentation even though a large percentage of those tumors are fixed to adjacent structures.
- Neurotropic tumor with early distant metastasis.

Acinic Cell Carcinoma
- Occurs exclusively in the parotid gland.
- Women > men.
- Pathologically defined by the presence of amyloid.
- Cell of origin in the serous acinar components and the intercalated duct cells.

Neck

Anatomy
- The neck is traditionally divided into anterior and posterior triangles (Figure 22-4).
- There are two main fascial planes (Figure 22-5):
  - Superficial cervical fascia: Encloses the platysma and muscles of facial expression. It begins at the zygoma of the face and extends inferiorly to the clavicle.
- Deep cervical fascia: Composed of four layers:
  1. **Superficial layer/investing fascia:** Encloses the trapezius, sternocleidomastoid (SCM), and strap muscles; submandibular and parotid glands; and the muscles of mastication. Stretches from the mandible to the clavicle.
  2. **Middle layer/visceral/pretracheal fascia:** Encloses the pharynx, larynx, trachea, and esophagus, thyroid/parathyroid glands, buccinator and constrictors, and the strap muscles. It goes from the skull base to the mediastinum. Posteriorly, this fascia forms a midline raphe that connects to the alar layer of the prevertebral fascia.
  3. **Deep layer/prevertebral fascia:** Encloses the paraspinal and cervical muscles; goes from skull base to the chest. Anteriorly, there are two layers to this fascia—the prevertebral layer lies anterior to the cervical vertebrae from the skull to the coccyx; anterior to that is the alar...
layer, which extends from the base of skull to the mediastinum. The danger space lies between the alar and prevertebral layers. Anterior to that is the visceral/buccopharyngeal fascial layer of the middle fascia.

4. Carotid sheath fascia: Envelopes the carotid artery, internal jugular vein, CN X. Runs from base of skull to the thorax.

- The spaces formed by the neck fascial layers are potential spaces for an infection to extend to, seed, and spread (Figure 22-6).

- **Above the hyoid bone:**
  - Parapharyngeal: Infections spread from tonsils, pharynx, teeth, parotid gland, and extension from other spaces. Parotid involvement, trismus, fever, and sepsis common with an infection here. Extraoral approach for drainage.
  - Submandibular.
  - Masticator: Infected when molar teeth infection spreads.
  - Parotid.
  - Peritonsillar: Loose connective tissue that lies between the tonsillar capsule medially and the superior constrictor laterally. (See section on peritonsillar abscess earlier in this chapter.)

- **Below the hyoid bone:** Visceral space encloses the pharynx, esophagus, larynx, trachea, and thyroid. The prevertebral and retropharyngeal spaces lie posterior to it.

- **Entire length of the neck:**
  - Retropharyngeal: Anterior to the alar layer and posterior to the buccopharyngeal fascia/esophagus and pharynx, an infection from here can spread to the danger space. Contains lymph nodes and connective tissue. The greatest number of LN here are found in children < 4 years old, and accounts for a large number of retropharyngeal abscesses.
  - Danger/alar space: Between alar layer and the prevertebral layer of deep cervical fascia. Infection spreads to superior mediastinum.
  - Prevertebral: Infection can spread to the coccyx.
  - Vascular: Infection of the carotid sheath.

**Vascular supply**
Carotid and subclavian systems.

**Nerve supply**
To the skin: C2–C4.
**Lymphatic drainage**
- The neck has a rich lymphatic network, and lymphogenous drainage from different sites in the head and neck is highly predictable.
- Lymph node areas are divided into different levels, which become relevant when performing a neck dissection and staging nodal disease.

**Congenital Diseases in the Neck**

**Branchial Apparatus Anomalies**
- The head, neck, and related structures form embryologically from five branchial arches, grooves, and pouches (Table 22-1).

**Table 22-1. The Five Branchial Arches**

<table>
<thead>
<tr>
<th>Arch</th>
<th>Nerve</th>
<th>Bones/Cartilage</th>
<th>Muscles/Vessels</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>First arch</td>
<td>Mandibular CN V3</td>
<td>Mandible, malleus, incus</td>
<td>Muscles of mastication, tensor tympani, anterior belly of digastic, tensor palatine</td>
<td>The pouch forms the middle ear cavity, and part of the tonsillar fossa and palatine fossa. The groove forms the EAC. A fistula would extend from the skin of the neck to the regions of the eustachian tube.</td>
</tr>
<tr>
<td>Second arch</td>
<td>Facial CN VII</td>
<td>Stapes, part of hyoid</td>
<td>Stapedius muscle, facial muscles, buccinators</td>
<td>The pouch forms the tonsillar fossa, palatine tonsil. A fistula would extend from the skin on the lower one third of the neck, anterior to the SCM, to the supratonsillar fossa. These are the most common fistulas.</td>
</tr>
<tr>
<td>Third arch</td>
<td>Glossopharyngeal CN IX</td>
<td>Part of hyoid bone</td>
<td>Stylopharyngeus, superior and middle constrictors, common and internal carotid artery</td>
<td>The pouch forms the thymus and inferior parathyroid gland</td>
</tr>
<tr>
<td>Fourth arch</td>
<td>Vagus CN X/superior laryngeal nerve</td>
<td>Thyroid and cuneiform cartilage</td>
<td>Inferior pharyngeal constrictor, cricopharyngeus, cricothyroid muscle, Laorta, and proximal R subclavian artery</td>
<td>The pouch forms the superior parathyroid glands, ultimobranchial body</td>
</tr>
<tr>
<td>Sixth arch</td>
<td>Vagus/CN X—recurrent laryngeal branch</td>
<td>Cricoid, arytenoids, corniculate, and tracheal cartilage</td>
<td>Intrinsic laryngeal muscles, inferior constrictor muscle, and ductus arteriosus</td>
<td>There is no associated pouch</td>
</tr>
</tbody>
</table>
Anomalies occur when pouches persist as a branchial sinus or a branchial fistula develops between the pouch and groove. A cyst can also develop if part of a groove or pouch becomes separated from the surface and does not resorb and become prone to repeated infections; need to be excised completely.

**Thyroglossal duct Cyst**

**Embryology/Definition**

- The thyroid develops from the foramen cecum at the base of the tongue and migrates down to the root of the neck along the thyroglossal duct.
- A remnant of the embryological migration becomes a thyroglossal duct cyst.

**Signs and Symptoms**

- Presents as a midline infrahyoid structure.
- The cyst can be at any level along the route of the duct, and usually moves with swallowing and protruding the tongue because of its attachment to the base of tongue.
- These cysts can become infected and drain cutaneously.

**Treatment**

- Surgical excision of the gland remnant (Sistrunk procedure), provided that ultrasound investigation reveals normal thyroid gland.
- The procedure involves removing the ectopic gland, duct, and central portion of the hyoid bone to minimize the chances of recurrence.

**Lymphatic Malformation**

**Embryology/Definition**

A malformation of the lymphatic system results in a multilocular neck mass filled with straw-colored fluid (formerly known as a cystic hygroma).

**Signs and Symptoms**

- Usually presents at birth with extensive neck and facial swelling; may complicate the airway.

**Treatment**

- Resection is indicated, both functionally and cosmetically.
- Instillation of a sclerosing agent has shown some promise as well.

**Infectious/inflammatory lesions of the neck**

**Ludwig's angina**

**Definition**

- Acute cellulitis of the submandibular triangle deep to the mylohyoid muscle. An emergency! Risk of sepsis and airway compromise.
- Usually arises from an oral cavity infection.

**Signs and Symptoms**

- Triangle is bound by attachment of the deep cervical fascia to the mandible and hyoid; suppuration that builds up creates a lot of pressure and pain.
- Infection can track posteriorly and potentially cause laryngeal edema.
Treatment
- Aggressive treatment with IV antibiotics is necessary.
- Intubation or tracheostomy may be needed to protect the airway.
- If nonresolving, may need I&D.

Retropharyngeal Abscess
Definition
- Usually results from a suppurating lymph node in the retropharyngeal space.
- Can be secondary to a penetrating pharyngeal injury.
- They can traverse to the danger space and track down to the mediastinum.

Signs and Symptoms
Patients are ill and febrile, dehydrated, complain of dysphagia and pain, and may be stridulous.

Diagnosis
Lateral soft tissue radiography is helpful in diagnosis when there is marked swelling of the prevertebral tissues, and CT can be used to find the exact location of the abscess.

Treatment
Drain abscess; maintain airway with an endotracheal tube or a tracheostomy.

WARD TIP
Intubate (consider nasotracheal intubation) before excision of Ludwig’s angina abscess if evidence of airway compromise.

Thyroid/Parathyroid Tumors
See Endocrine System chapter.

Lymphoma
Signs and Symptoms
- A disease of young and middle-aged adults.
- Usually presents with multiple, slow-growing, rubbery lymph nodes in the neck, which may be the only presenting symptom of the disease.
- Systemic symptoms, including fever and night sweats, imply a worse prognosis.

Diagnosis
Open biopsy of the node. Cellular architecture is important in both diagnosing Hodgkin’s vs. non-Hodgkin’s lymphoma and determining subtype of each.

Treatment
Depends on type and stage, and can include chemotherapy or RT.

Metastatic Lymphadenopathy
Signs and Symptoms
- Neck mass is the presenting sign of SCC in the head and neck or other anatomic location.
- Usually presents in older patients with a firm neck mass.

Diagnosis
- FNA helps differentiate between metastatic disease and other causes of chronic cervical lymphadenopathy (e.g., TB).
- Panendoscopy of the upper aerodigestive tract is useful for diagnosis.

WARD TIP
Neck dissection radical: Removal of all levels of lymph nodes, the SCM, internal jugular vein, and CN XI.
Modified radical: A more selective procedure—removal of all levels of lymph nodes, but preservation of either SCM, internal jugular vein, or CN XI.
Selective: All levels of lymph nodes are not removed.
**Treatment**

Dictated by the location of the primary tumor. A surgical dissection and removal of the neck nodes and associated structures is necessary to pathologically identify and treat the disease.

**References**


**WARD TIP**

The unknown primary head and neck cancer: Identify based on lymph node presentation pattern and characteristic locations draining to lymphatic groups.
General Neuroanatomy

Central Nervous System (CNS) Vasculature

Head Trauma

Skull Fractures

Diffuse Intracranial Lesions

Focal Intracranial Lesions

Management of Mild to Moderate Head Trauma

Management of Severe Head Injury (GCS < 9)

CNS Tumors

Primary Brain Tumors

Metastatic Tumors

Spine

Spinal Trauma

Cerebral Contusion

Diffuse Axial Injury (dai)

Cerebral Contusion

Intracerebral Hemorrhage

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Subdural Hematoma

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Glioma Tumor

Astrocytoma

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Ependymoma

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Spinal Cord Injuries

Spinal Cord Syndromes

Cervical Spine Fractures and Dislocations

Thoracic Spine Fractures

Thoracolumbar Junction and Lumbar Spine Fractures and Dislocations
**General Neuroanatomy**

**Cranial Foramina**

- **Optic canal**: Optic nerve and ophthalmic artery.
- **Superior orbital fissure**: Cranial nerves (CN) III, IV, VI, and V<sub>1</sub>.
- **Foramen rotundum**: CN V<sub>2</sub>.
- **Foramen ovale**: CN V<sub>3</sub>.
- **Carotid canal**: Internal carotid artery.
- **Internal acoustic meatus**: CN VII and VIII.
- **Stylomastoid foramen**: CN VII and stylomastoid artery.
- **Jugular foramen**: Internal jugular vein and CN IX–XI.
- **Hypoglossal canal**: CN XII.
- **Foramen spinosum**: Middle meningeal artery and vein.
- **Foramen magnum**: Spinal cord, CN XI (spinal accessory), and vertebral, posterior, and anterior spinal arteries.

**Central Nervous System (CNS) Vasculature**

Internal carotid artery (ICA) does not produce branches until it enters the petrous bone, where it gives off geographically occult feeders to the middle and inner ear.

**Major ICA Branches Visible with Angiography**

Meningohypophyseal, inferolateral trunk, ophthalmic, posterior communicating, anterior choroidal, middle cerebral, anterior cerebral.

**Anterior Cerebral artery (aCA) Branches**

Medial lenticulostriates, anterior communication, recurrent artery of Heubner, orbitofrontal, frontopolar, pericallosal, callosomarginal.

**Middle Cerebral artery (mCA) Branches**

Lateral lenticulostriates, anterior temporal, posterior cerebral, ascending frontal, lateral orbitofrontal, precentral, central, anterior parietal, posterior parietal, angular.

**Vertebral artery Branches**

Posterior meningeal, anterior spinal, posterior inferior cerebellar, vertebrais fuse to form the basilar artery.

**Basilar artery Branches**

Anterior inferior cerebellar, pontine perforators, superior cerebellar, posterior cerebral.

**Posterior Cerebral artery Branches**

Posterior thalamoperforators, medial posterior choroidal, lateral posterior choroidal, thalamogeniculoculater, inferior temporals, parieto-occipital, calcarine, posterior pericallosal.
**Figure 23-1.**  MR angiography demonstrating circle of Willis. (Reproduced, with permission, from Fleckenstein P, Tranum-Jensen J. Anatomy and Diagnostic Imaging. 2nd ed. Philadelphia, PA: WB Saunders; 2001: 244.)

Cerebral arteries, MR angiography, circle of Willis

1: Internal carotid artery, “siphon”  
2: Internal carotid artery in cavernous sinus  
3: Internal carotid artery in carotid canal  
4: Insular branches of middle cerebral artery  
5: Posterior communicating artery  
6: Anterior communicating artery  
7: Anterior cerebral artery  
8: Middle cerebral artery  
9: Basilar artery  
10: Superior cerebellar artery  
11: Posterior cerebral artery  
12: Anterior inferior cerebral artery (AICA)  
13: Vertebral artery

**Arterial**

Circle of Willis is complete in only approximately one fifth of persons (see Figure 23-1).

**Head Trauma**

**Anatomy and Physiology**

**Scalp**

- The scalp consists of five layers.
- Highly vascular structure, may be the source of major blood loss.

**Skull**

- Rigid and inflexible (fixed volume).
- Composed of the cranial vault and base.

**WARD TIP**

Layers of the scalp:

- Skin
- Connective tissue
- Aponeurosis (galea)
- Loose areolar tissue
- Pericranium
Brain
- Makes up 80% of intracranial volume.
- Partially compartmentalized by the reflections of dura (falx cerebri and tentorium cerebelli).

Cerebrospinal Fluid (CSF)
- Formed primarily by the choroid plexus at a rate of ~500 cc/day with 150 cc of CSF circulating at a given moment.
- Cushions the brain.

Cerebral Blood Flow
- Brain receives ~15% of cardiac output.
- Brain responsible for ~20% of total body O₂ consumption.

Cerebral Perfusion Pressure (CPP)
- CPP = MAP - ICP.
- MAP = mean arterial blood pressure (avoid systolic blood pressure [SBP] < 90 mmHg).
- ICP = intracranial pressure (ICP > 20 mmHg should be treated).
- Maintaining CPP between 50 and 70 mmHg in nonoperative brain injury is the fundamental treatment.

Monro–Kellie Hypothesis
The sum of the volume of the brain, blood, and CSF within the skull must remain constant. Therefore, an increase in one of the above must be offset by decreased volume of the others.

Assessment
- History.
- Identify mechanism and time of injury, loss of consciousness, concurrent use of drugs or alcohol, medications that may affect pupillary size (e.g., glaucoma medications), past medical history (especially previous head trauma and stroke with their residual effects, and previous eye surgery, which can affect pupillary size and response), and the presence of a “lucid interval.”

Vital Signs
Cushing reflex:
- Brain’s attempt to maintain the CPP.
- Hypertension and bradycardia in the setting of increased ICP.

Physical Exam
- Search for signs of external trauma such as lacerations, hemotympanum, ecchymoses, and avulsions, as these may be clues to underlying injuries such as depressed or open skull fractures.
- Anisocoria (unequal pupils) is found in a small percentage (10%) of normal people; however, anisocoria in the patient with head trauma is pathologic until proven otherwise.

Glasgow Coma Scale (GCS)
The GCS may be used as a tool for classifying head injury (see Figure 23-2):
Severe head injury GCS 8 or less
Moderate head injury GCS 9–13
Mild head injury GCS 14 or 15

Diagnostic Studies
- Assume C-spine injury in head injury patients and immobilize until cleared.
- Skull films have largely been replaced by computed tomography (CT) scan.
Indications for head/brain CT:
1. Neurologic deficit.
2. Persisting depression or worsening of mental status.
3. Depressed skull fracture or linear fracture overlying a dural venous sinus or meningeal artery groove (as demonstrated with skull x-rays).

Skull Fractures

**Linear (nondepressed)**

**Stellate**

- Depressed: Carries a much greater risk of underlying brain injury and complications, such as meningitis and posttraumatic seizures.
- Basilar: Signs include periorbital ecchymoses (raccoon’s eyes), retroauricular ecchymoses (Battle’s sign), otorrhea, rhinorrhea, hemotympanum, and cranial nerve palsies.

**Diffuse Intracranial Lesions**

**Cerebral Concussion**

- Transient loss of consciousness that occurs immediately following blunt, nonpenetrating head trauma, caused by impairment of the reticular activating system.
- Recovery is often complete; however, residual effects such as headache may last for some time.

**Diffuse axonal injury (DAI)**

- Caused by microscopic shearing of nerve fibers, scattered microscopic abnormalities.
- Frequently requires intubation, hyperventilation, CPP monitoring, and admission to a neurosurgical intensive care unit (ICU).
- Patients are often comatose for prolonged periods of time.
- Mortality is approximately 33%.

**Glasgow Coma Scale.**

<table>
<thead>
<tr>
<th>Eyes</th>
<th>Open spontaneously</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Open to verbal command</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Open to pain</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1</td>
</tr>
<tr>
<td>Best motor response</td>
<td>Obeys verbal command</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Localizes pain to painful stimulus</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Flexion–withdrawal</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Decorticate rigidity</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Decerebrate rigidity</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1</td>
</tr>
<tr>
<td>Best verbal response</td>
<td>Oriented and converses</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Disoriented and converses</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td></td>
<td>15</td>
</tr>
</tbody>
</table>

**Figure 23-2.**

- WARD TIP
  - Ring test for CSF rhinorrhea (in the presence of epistaxis): Sample of blood from nose placed on filter paper to test for presence of CSF. If present, a large transparent ring will be seen encircling a clot of blood.

- WARD TIP
  - Typical scenario: A 20-year-old female has brief loss of consciousness following head injury. She presents to the ED awake but is amnestic for the event and keeps asking the same questions again and again. Think Concussion.

- WARD TIP
  - No mass lesion is seen on CT in DAI.
Focal Intracranial Lesions

**Cerebral Contusion**
- Occurs when the brain impacts the skull. May occur directly under the site of impact (coup) or on the contralateral side (contrecoup).
- Patients may have focal deficits; mental status ranges from confusion to coma.

**Intracerebral Hemorrhage**
Caused by traumatic tearing of intracerebral blood vessels. Difficult to differentiate from a contusion.

**Epidural Hematoma**
- Collection of blood between the dura and the skull.
- Majority associated with tearing of the middle meningeal artery from an overlying temporal bone fracture.
- Typically biconvex or lenticular in shape (see Figure 23-3).
- Patients may have the classic “lucid interval,” where they “talk and die.” Requires early neurosurgical involvement and hematoma evacuation.
- Good outcome if promptly treated.

**Subdural Hematoma**
- Collection of blood below the dura and over the brain (see Figure 23-4). Results from tearing of the bridging veins, usually secondary to an acceleration–deceleration mechanism.


**Exam Tip**
Typical scenario: A 19-year-old male with a head injury has loss of consciousness followed by a brief lucid interval. He presents to the emergency department (ED) in a coma, with an ipsilateral fixed and dilated pupil and contralateral hemiparesis. Think: Epidural hematoma.
Classified as acute (<24 hours), subacute (24 hours–2 weeks), and chronic (>2 weeks old).

- Acute and subacute subdurals require early neurosurgical involvement.
- Alcoholics and the elderly (patients likely to have brain atrophy) have increased susceptibility.

Management of Mild to Moderate Head Trauma

- Safe disposition of the patient depends on multiple factors.
- Any patient with a persisting or worsening decrease in mental status, focal deficits, severe mechanism of injury, penetrating trauma, open or depressed skull fracture, or seizures, or who is unreliable or cannot be safely observed at home, should be admitted for observation.
- Patients with mild and sometimes moderate head trauma, brief or no loss of consciousness, no focal deficits, an intact mental status, a normal CT scan, and reliable family members who can adequately observe the patient at home can often be discharged with proper discharge instructions.
- Discharge instructions should include signs and symptoms for family members to watch for, such as persisting or worsening headache, dizziness, vomiting, inequality of pupils, confusion.
- If any of the above signs are found, the patient should be brought to the ED immediately.

WARD TIP

Acute subdural hematomas have a high mortality—approximately one third to two thirds—mostly due to underlying brain contusion and shear.

WARD TIP

When in doubt, admit the patient for observation.
Management of Severe Head Injury (GCS < 9)

- Check airway, breathing, circulation (ABCs).
  - Patient needs intubation by definition.
  - Hyperventilation is not recommended, $P_{co2}$ should be maintained at 35 mmHg and $P_{ao2} > 60$ mmHg.
- ICP measurement via ventriculostomy should be done in all patients with severe traumatic brain injury and CT scan abnormalities.
- Maintain an adequate blood pressure (BP) with isotonic fluids. Avoid SBP $<$ 90 mmHg.
- Treatment of increased ICP: $> 20$ mmHg with mannitol.
- Maintain CPP between 50 and 70 mmHg (preferably $> 60$ mmHg).
- Early feeding improves outcome.
- Use sequential compression device for deep venous thrombosis (DVT) prophylaxis.
- Corticosteroids are contraindicated in these patients.
- Consider prophylactic anticonvulsant therapy with phenytoin for 7 days.
- Hypothermia may be beneficial for neurological recovery.
- Treat the pathology whenever possible (e.g., surgical drainage of a hematoma).

Hydrocephalus

Enlargement of the ventricles with excess CSF.

General

- Prevalence, $-1\%$; congenital incidence, 1 in 1,000.

  Three general categories:
  - Communicating: Also known as normal-pressure hydrocephalus since flow remains between all the ventricles. Defective absorption by arachnoid granulations in the subarachnoid space.
  - Noncommunicating (obstructive): Increased pressure within the ventricular system due to disruption of CSF flow between the ventricles (e.g., aqueductal stenosis, tumor, cyst). May not affect all ventricles depending on the location of the block (e.g., aqueductal stenosis spares the fourth ventricle).
  - Ex vacuo: Atrophic parenchymal tissue loss resulting in dilated ventricles. Not pathologic hydrocephalus. (Example: Atrophy of caudate nuclei in Huntington’s disease causes expansion of the lateral ventricles.)

Imaging

See Figure 23-5.

Etiologies

- Acquired:
  - Hemorrhage: Subarachnoid hemorrhages cause meningeal adhesions.
  - Infectious/inflammatory: Meningitis will cause meningeal adhesions.
  - Obstructing masses.
  - Postoperative (particularly in pediatric posterior fossa procedures).

Clinical Presentation

- Communicating: Classic triad of gait apraxia, dementia, and incontinence. Gait apraxia is usually first and can have a slow onset.
Figure 23-5. **Brain CT demonstrating gross hydrocephalus.** Note massive dilatation of lateral and third ventricles. The fourth ventricle is decompressed. There is a VP shunt tip in the body of the right lateral ventricle. Note deformity of calvarium, which is consistent with long-standing hydrocephalus.

- **Noncommunicating:** Headache, nausea/vomiting, ataxia, abducens palsy, Parinaud’s syndrome.  
  - In children, check for bulging anterior fontanelle, increase in head circumference, irritability, poor feeding, and engorged scalp veins.

**Treatment**

- Acetazolamide to reduce CSF production and furosemide to promote diuresis (both only temporizing).
- Lumbar puncture: Used to quickly relieve CSF pressure. Considerable clinical improvement has high predictive value for success of shunt placement.
- Shunt placement:
  - Most commonly, a ventriculoperitoneal (VP) shunt is placed (see Figure 23-6). Alternatives include ventriculoatrial and ventriculopleural shunts.
  - Shunts are placed similar to an extraventricular drain except that the catheter is subcutaneously tunneled behind the ear, where a valve is attached and placed in the subgaleal space. The catheter is then tunneled over the clavicle and to the destination outsource: peritoneum (VP, most common), atrium (ventriculoatrial), or pleura (ventriculopleural). Ventriculoatrial shunts occasionally cause pulmonary hypertension as a complication.
Complications

- Obstruction (usually proximal).
- Infection: Staphylococcus.
- Subdural hematoma.
- Patient growth—possible need for replacement of the distal catheter as the infant/child grows (shunt tip will pull out of peritoneal cavity).

CNS Tumors

general

- Most brain tumors present with progressive neurologic deficit, motor weakness, headache, or seizure.
- Tumor headache is usually due to elevated ICP.
- Any new-onset seizure in adulthood should prompt an aggressive search for a brain tumor.

**Presentation**

- **Posterior fossa mass**: Headache, nausea/vomiting, ataxia, diplopia, Parinaud’s syndrome, cranial nerve paresis, rotatory/vertical nystagmus.
- **Supratentorial mass**: Headache, nausea/vomiting, diplopia, Parinaud’s syndrome, motor weakness, aphasia, tumor transient ischemic attack (TIA—hemorrhage/vascular compression).
- Dexamethasone may halt/reverse neurologic deterioration caused by vasogenic edema.

**Primary Brain Tumors**

**Glioma**

CNS tumor arising from glial cells (astrocytes or oligodendrocytes).

**Astrocytoma**

World Health Organization (WHO) grading system for astrocytomas most commonly used:

- Grade 1—Pilocytic astrocytoma: In children, consisting of cystic lesions, most often in the cerebellum, brain stem, or hypothalamus. Has good prognosis.
- Grade 2—Low-grade astrocytoma: Present at 20–40 years; median survival 5–10 years.
- Grade 3—Anaplastic astrocytoma.
- Grade 4—Glioblastoma multiforme: Present at 50–70 years; median survival 1–2 years; highly malignant with poor prognosis.

**Oligodendrogliaoma**

- Male > female (3:2).
- Mostly occur in middle-aged adults, slow growing.
- Predilection for frontal and temporal lobes (40–70%).
- Uniform cells with round nuclei (“fried egg” appearance).
- May have mixed astrocyte component, here called oligoastrocytomas.
- Most frequently presents with a seizure.
- Surgery for symptomatic lesions, lesions > 5 cm.
- Responds well to chemo.

**Ependymoma**

- Derived from cells lining the ventricles.
- Most occur in children.
- Fourth ventricle (most common).
- Spinal cord.
- Lateral ventricles.
- Most often there is hydrocephalus.
- Tends to disseminate through CSF (“seeding”).
- Surgical resection.
- Radiation if in fourth ventricle or spinal cord.
- Chemotherapy is of little benefit.

WARD TIP

Preop imaging of the spinal neuraxis should be performed to detect CSF seeding.
malignant glioma
- Originates from arachnoid granulations.
- Most common benign brain tumor.
- Highest incidence in sixth to seventh decades.
- Seen at superior convexities, sphenoid wing, orbital rim, cerebellar tentorium, ventricles.
- Rarely invasive or metastatic; many discovered incidentally.
- Observe with annual CT scans if asymptomatic and < 2 cm.
- Surgical excision if symptomatic.
- External beam radiotherapy/gamma knife if subtotal resection or unresectable.

pituitary adenoma
- Associated with multiple endocrine neoplasia (MEN) syndrome.
- Seen at the sella turcica with parasellar extension.
- May envelop carotid arteries.
- Cell of origin may be chromophobe (null cell or prolactinoma), acidophil (growth hormone [GH], causing acromegaly), or basophil (adrenocorticotropic hormone [ACTH]—Cushing’s syndrome).
- Generally slow, progressive enlargement.
- Headache, bitemporal hemianopsia (superior to inferior loss).
- Prolactinomas (prolactin secreting):
  - Medical treatment (dopamine agonists).
  - Surgical excision if no response to therapy, through transsphenoidal approach.
- Acromegaly (growth hormone secreting):
  - Surgical resection—50% cure.
  - Avoid surgery in asymptomatic elderly patients as there is no survival benefit.
  - Medical therapy with octreotide (somatostatin analogue).
- Cushing’s disease (cortisol secreting):
  - Surgery is the treatment of choice—85% cure.
  - Nonfunctional adenomas.
  - Observe if asymptomatic; otherwise, surgical resection.

neuroma
- CN VIII affected most frequently (acoustic neuroma).
- Typical extension of acoustic neuroma into cerebellopontine angle with possible compression of CN V, VII, IX, or X.
- Usually unilateral.
- Bilateral CN VIII neuromas pathognomonic for neurofibromatosis type 2 (NF2).
- Unilateral progressive hearing loss (sensorineural), tinnitus, disequilibrium, possible vertigo.
- Perform pretreatment audiometric and vestibular testing.
- Surgical excision or stereotactic radiosurgery.
- Conventional radiotherapy (RT).

Craniopharyngioma
- Most occur in childhood.
- Anterior superior pituitary margin, may extend upwards toward third ventricle; may compress optic chiasm or pituitary gland.
- Benign but difficult to cure.
- Arises from remnants of the craniopharyngeal duct or Rathke’s pouch.
- Headache and visual disturbance.
- Preop endocrinological evaluation.
- **Treatment:** May be observed; surgical resection if symptomatic.

### Hemangioblastoma

- Tend to appear clinically in middle adulthood.
- Associated with Von Hippel–Lindau disease (dominant inheritance).
- Most common adult posterior fossa tumor.
- Spinal cord (70% have associated syringomyelia).
- Benign.
- Ataxia, dizziness, concurrent hepatic, renal, or pancreatic cysts.
- Surgery curative in sporadic cases.
- RT to slow growth if surgically unresectable.

### Glomus Tumors

Tumors arising from paraganglion cells:

- **Glomus jugulare tumor:** Since located underneath floor of middle ear, typical presentation involves deafness, facial palsy, and possibly a palpable mass anterior to the mastoid eminence.
  - **Treatment:** Surgery with possible radical mastoidectomy followed with RT.
- **Carotid body tumor:** Painless mass below angle of jaw. May affect other cranial nerves in area. Small percentage of patients have experienced TIAs.
  - **Treatment:** Surgery; no RT. Possible embolization prior to surgery.

### Metastatic Tumors

- More than 50% of brain tumors are metastatic in origin.
- Most disseminate hematogenously.
- Incidence of cerebral metastases is increasing.
- Common sources: Bronchogenic lung cancer (40%), breast cancer (19%), melanoma (10%), colon adenocarcinoma (7%).
- Most present with progressive focal neurologic deficit or signs/symptoms of increased ICP.
- Certain metastases are more likely to hemorrhage: Melanoma, renal cell carcinoma, choriocarcinoma.
- Most metastases occur in the cerebral hemispheres at the gray–white junction or in the cerebellum.

### Imaging

- CT or magnetic resonance imaging (MRI—preferred).
- Typically, metastases are well circumscribed.
- Usually, significant surrounding edema greater than that seen with primary brain tumors.
- Metastases usually enhance (completely or ring enhancement).

### Evaluation

Search for a primary source:

- CT chest/abdomen/pelvis.
- Bone scan, mammogram in women, guaiac for occult blood.
management

- Biopsy for diagnosis if no other source identified.
- Resect most solitary symptomatic lesions and treat with RT postoperatively.
- If multiple metastases, proceed directly to RT.

Spine

definitions

- **Spondylosis**: Degenerative changes in spine; arthritis.
- **Spondylolisthesis**: Subluxation of one vertebral body on another.
- **Spondylolysis**:
  - Fracture or defect in pars interarticularis.
  - Mostly congenital at L5 level (spina bifida occulta).
  - If due to congenital/degenerative etiology generally does not require surgical intervention.
  - Traumatic spondylolysis requires spinal fusion.

low Back pain

See Orthopedics chapter.

Spinal Trauma

general

- Spinal trauma may involve injury to the spinal column, spinal cord, or both.
- Over 50% of spinal injuries occur in the cervical spine (see Figure 23-7), with the remainder being divided between the thoracic spine, the thoracolumbar junction, and the lumbosacral region.
- As long as the spine is appropriately immobilized, evaluation for spinal injury may be deferred until the patient is stabilized.

anatomy

- There are seven cervical, 12 thoracic, five lumbar, five sacral, and four coccygeal vertebrae.
- The cervical spine is the region most vulnerable to injury.
- The thoracic spine is relatively protected due to limited mobility from support of the rib cage (T1–T10); however, the spinal canal through which the spinal cord traverses is relatively narrow in this region. Therefore, when injuries to this region do occur, they usually have devastating results.
- The thoracolumbar junction (T11–L1) is a fairly vulnerable region as it is the area between the relatively inflexible thoracic region and the flexible lumbar region.
- The lumbosacral region (L2 and below) contains the region of the spinal canal below which the spinal cord proper ends and the cauda equina begins.

pathology and pathophysiology

Spinal injuries can generally be classified based on:

- Fracture/dislocation type (mechanism, stable vs. unstable).
- Level of neurological (sensory and motor) and bony involvement.
- Severity (complete vs. incomplete spinal cord disability).
neurogenic shock

- A state of vasomotor instability resulting from impairment of the descending sympathetic pathways in the spinal cord, or simply a loss of sympathetic tone.
- Signs and symptoms: Flaccid paralysis, hypotension, bradycardia, cutaneous vasodilation, and a normal to wide pulse pressure.

spinal shock

- State of flaccidity and loss of reflexes occurring immediately after spinal cord injury.
- Loss of visceral and peripheral autonomic control with uninhibited parasympathetic impulses.
- May last from seconds to weeks, and does not signify permanent spinal cord damage.
- Long-term prognosis cannot be postulated until spinal shock has resolved.

spinal cord injuries

Complete vs. incomplete:
- Complete spinal cord injuries demonstrate no preservation of neurologic function distal to the level of injury. Therefore, any sensorimotor function below the level of injury constitutes an incomplete injury.
- Sacral sparing refers to perianal sensation, voluntary anal sphincter contraction, or voluntary toe flexion, and is a sign of an incomplete spinal cord injury (i.e., better prognosis).
Physical exam

- Classification of spinal cord injuries as complete or incomplete requires a proper neurologic exam.
- The exam should include testing of the three readily assessable long spinal tracts (see Figure 23-8):
  - Corticospinal tract (CST):
    - Located in the posterolateral aspect of the spinal cord.
    - Responsible for ipsilateral motor function.
    - Tested via voluntary muscle contraction.
  - Spinothalamic tract (STT):
    - Located in the anterolateral aspect of the spinal cord.
    - Responsible for contralateral pain and temperature sensation and tested as such.
  - Posterior columns:
    - Located in the posterior aspect of the spinal cord.
    - Responsible for ipsilateral position and vibratory sense and some light touch sensation.
    - Tested using a tuning fork and position sense of the fingers and toes.

spinal Cord syndromes

- Anterior cord syndrome:
  - Pattern seen with injury to the anterior portion of the spinal cord or with compression of the anterior spinal arteries (artery of Adamkiewicz).
  - Involves full or partial loss of bilateral pain and temperature sensation (STT) and paraplegia (CST) with preservation of posterior column function.
  - Often seen with flexion injuries.
  - Carries a poor prognosis.
- Brown–Séquard syndrome:
  - Pattern seen with hemisection of the spinal cord usually secondary to a penetrating injury, but may also be seen with disk protrusion, hematoma, or tumor.
- Consists of ipsilateral loss of motor function (CST) and posterior column function, with contralateral loss of pain and temperature sensation.

**Central cord syndrome:**
- Pattern seen with injury to the central area of the spinal cord often in patients with a preexisting narrowing of the spinal canal.
- Usually seen with hyperextension injuries, its cause is usually attributed to buckling of the ligamentum fava into the cord and/or an ischemic etiology in the distribution of branches of the anterior spinal artery.
- Characterized by weakness greater in the upper extremities than the lower extremities, and distal worse than proximal.
- Has a better prognosis than the other partial cord syndromes with a characteristic pattern of recovery (lower extremity recovery progressing upward to upper extremity recovery, then the hands recover strength).

**Treatment of spinal Cord Injuries**
- Always start with the ABCs of trauma resuscitation.
- Maintain spinal immobilization throughout the resuscitation.
- Estimate level of neurologic dysfunction during the secondary survey.
- Obtain appropriate diagnostic studies.
- Establish early neurosurgical consultation.
- If blunt spinal cord injury is diagnosed, begin high-dose methylprednisolone (must be given within 8 hours of injury and not for penetrating injury).
- Loading dose of 30 mg/kg over 15 minutes during hour 1, followed by a continuous infusion of 5.4 mg/kg/hr; the infusion is continued for 23 hours if the bolus is given within 3 hours of injury, or for 47 hours if the bolus is given within 8 hours of injury.

**Cervical spine Fractures and dislocations**

**General**
As mentioned above, these injuries are usually classified on the basis of mechanism (flexion, extension, compression, rotation, or a combination of these), location, and/or stability.

**Imaging**
- Four views of the cervical spine are obtained (lateral, anteroposterior, oblique, and odontoid).
- A lateral view alone will miss 10% of cervical spine injuries.

**Jefferson Fracture**
- C1 (atlas) burst fracture.
- Most common C1 fracture.
- Consists of a fracture of both the anterior and posterior rings of C1.
- Results from axial loading such as when the patient falls directly on his or her head or something falls on the patient’s head.
- Consider all C1 fractures unstable even though most are not associated with spinal cord injury (see Figure 23-9).

**Odontoid Fractures**
See Figure 23-10.
- Type 1: Involves only the tip of the dens (stable).
- Type 2: Involves only the base of the dens (unstable).
- Type 3: Fracture through the base and body of C2 (generally unstable).
Hangman’s Fracture

See Figure 23-11.

- Fracture of both C2 pedicles (“posterior elements”).
- Usually due to a hyperextension mechanism.
- Unstable fracture; however, often not associated with spinal cord injury because the spinal canal is at its widest through C2.
Clay’s hovel’s Fracture
See Figure 23-12.

- Usually a flexion injury resulting in an avulsion of the tip of the spinous process (C7 > C6 > T1).
- May also result from a direct blow.

thoracic spine Fractures

- Most injuries occur at the junction between the relatively fixed upper thoracic spine and the mobile thoracolumbar region (T10–L5).
- The spinal canal in this region is narrow and the blood supply to this region of spinal cord is in a watershed area (the greater radicular artery of Adamkiewicz enters the spinal canal at L1, but provides blood flow as high as T4).
- Most thoracic spine fractures are caused by hyperflexion leading to a wedge or compression fracture of the vertebral body.
- Most fractures/dislocations in this area are considered stable because of the surrounding normal bony thorax; however, neurologic impairment resulting from injuries in this area is often complete.

thoracolumbar junction and lumbar spine Fractures and dislocations

- Results from axial loading and flexion.
- Potentially unstable.
- Neurologic injury is uncommon.
Distraction or seat-Belt Injury
- Frequently referred to as a Chance fracture.
- Horizontal fracture through the vertebral body, spinous processes, laminae, pedicles, and tearing of the posterior spinous ligament.
- Caused by an acceleration–deceleration injury of a mobile person moving forward into a fixed seat belt.

Thoracic outlet syndrome (To s)
- Subclavian artery/vein and brachial plexus pass through a space defined by the clavicle and first rib (thoracic outlet).
- Vascular compromise is more common than neurologic: Unilateral Raynaud’s phenomenon, Adson’s sign—loss of radial pulse on abduction and external rotation of the arm.
- Can present with T1 sensory loss, wasting of thenar muscles.

Etiologies
- Fibrous band compressing C8/T1 roots (inferior trunk).
- Elongated C7 transverse process—“cervical rib.”

Treatment
Surgical lysis of fibrous band or removal of C7 transverse process by either transaxillary or supraclavicular approach to thoracic outlet.
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Ischemic Heart Disease

**Definition**

Myocardial injury caused by chronic or acute episodes of ischemia.

**Causes**

Disorders that affect coronary blood flow:

- Atherosclerotic coronary artery disease (most common).
- Valvular heart disease.
- Aortic dissection with or without involvement of coronary ostia.
- Vasculitis.
- Congenital coronary anomalies.
- Emboli, mainly tumor, calcium, vegetation.

**Anatomy**

- **Right coronary dominance** (85–90% of patients): Right coronary artery (RCA) gives rise to posterior descending artery (PDA).
- **Left coronary dominance** (10–15%): Left circumflex artery gives rise to posterior descending artery. More common in males.
- **Codominance**: Occasionally, PDA arises from both the RCA and left coronary artery (LCA).
- Consider how a lesion from either side will affect the posterior and inferior walls.

**Pathophysiology**

- Ischemic areas arise from lack of blood flow relative to the metabolic demands of the myocardium.
- The oxygen extraction already being high under normal metabolic conditions (75%), the heart must rely on increased blood flow to meet heightened demand.
- Determinants of demand: Wall tension (vis-à-vis preload, afterload, wall thickness), heart rate, level of contractility.
- An atherosclerotic plaque impedes flow significantly when coronary cross-sectional area is reduced by 75% (a 50% reduction in diameter).
- Coronary atherosclerotic lesions are usually multifocal and multivessel.
- Plaque rupture is the main cause of escalation of symptoms. Intermittent closure of dynamic plaques underlies symptoms of unstable angina.

**Risk Factors**

- Hypertension (HTN)
- Smoking
- Hypercholesterolemia
- Abdominal obesity
- Diabetes
- Family history
- Psychosocial stress and depression

**Signs and Symptoms**

- Fatigue
- Angina pectoris

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**Exam Tip**

Coronary atherosclerosis is the most common cause of cardiovascular morbidity and mortality in the Western world. Before age 70, men are more commonly affected than women by a ratio of 4:1. After age 70, it is 1:1.
- Dyspnea
- Edema
- Palpitations
- Syncope
- Abnormal heart sounds
- Seventy-five percent present with classic angina, 25% present atypically, many have "silent" myocardial infarctions (MIs) (particularly diabetics and elderly).

**Diagnosis**

- Electrocardiogram (ECG) may reveal ST segment elevations or depressions, inverted T waves, or Q waves.
- Stress test (to look at myocardial response when myocardial demand is increased).
- Echocardiography (localize dyskinetic wall segments, valvular dysfunction, estimate ejection fraction [EF]).
- Cardiac catheterization with angiography and left ventriculography (specifies coronary anatomy and sites of lesions to quantify the severity of the disease and vulnerable areas of myocardium, as well as to provide a road map for surgical or percutaneous intervention).
- Emerging technologies include: Computed tomography angiography and Coronary intravascular ultrasound.

**Treatment**

- Medical—aspirin, β-blockers, calcium channel blockers, angiotensin-converting enzyme (ACE) inhibitors, diuretics, nitrates.
- Percutaneous transluminal coronary angioplasty (PTCA).
- Surgical—coronary artery bypass grafting.

**Coronary Artery Bypass Grafting (CABG)**

**Description**

- Bypass of discrete areas of obstruction in coronary vessels using the internal mammary artery (IMA), radial artery, a reversed segment of greater saphenous vein, inferior epigastric artery, or gastroepiploic artery.
- IMA used in 95% of CABGs, usually to left anterior descending (LAD).
- Three or four grafts are used on average.
- Minimally invasive direct CABG (MIDCAB): Fewer incisions, no cardiopulmonary bypass or cardioplegia is used (performed on a beating heart, limited to single-vessel disease). Minimizes pain, recovery time, and chances of wound infection.
- Port access technique: Endovascular aortic occlusion and cardiopulmonary bypass with cardioplegia allows for broader use of MIDCAB (multi-vessel disease, combined valve-coronary artery surgery).
- Vessel acronyms:
  - LAD—left anterior descending
  - RCA—right coronary artery
  - LMCA—left main coronary artery
  - LCX—left circumflex artery
  - PDA—posterior descending artery
  - OMn—oblique marginal artery number 1, 2, 3, etc.
  - (L)(R)IMA—(left)(right) internal mammary artery

**WARD TIP**

Severities of heart failure graded by **NYHA Classification**:
- Class I—no symptoms (fatigue, dyspnea, palpitations, angina)
- Class II—symptoms with severe exertion
- Class III—symptoms with mild exertion
- Class IV—symptoms at rest

**WARD TIP**

Diffuse patterns of coronary vessel obstruction, as can occur in diabetes, may not be amenable to CABG.
inDiCa tions

- Mild angina or asymptomatic:
  - LMCA stenosis > 50%.
  - LMCA equivalent: Proximal LAD and LCX stenoses > 70%.
  - Triple-vessel disease (survival benefit > with abnormal left ventricular [LV] function: EF ≤ 0.50).
- Chronic stable angina:
  - Same as mild angina or asymptomatic.
  - Disabling angina despite maximal medical therapy (acceptable-risk patient).
  - Double-vessel disease with significant proximal LAD stenosis: Either EF < 0.50 or ischemia on noninvasive testing.
  - Single- or double-vessel disease without significant proximal LAD stenosis, but with a large area of viable myocardium and high-risk criteria on noninvasive testing.
- Unstable angina (UA)/non–ST elevation MI (NSTEMI):
  - LMCA stenosis ≥ 60%.
  - LMCA equivalent: Proximal LAD and LCX stenoses > 70%.
  - Ongoing ischemia unresponsive to maximal nonsurgical therapy.
- ST elevation MI (STEMI)/acute MI (AMI) (emergency or urgent CABG):
  - Failed angioplasty with persistent pain or hemodynamic instability.
  - Persistent or recurrent ischemia refractory to medical therapy.
  - Postinfarction ventricular septal rupture, mitral valve insufficiency or left ventricular aneurysm.
  - Cardiogenic shock in patients < 75 years with ST segment elevation or left bundle branch block (LBBB) or posterior MI who develop shock within 36 hours of MI.
  - Life-threatening ventricular arrhythmias in the presence of ≥ 50% left main stenosis and/or triple-vessel disease.
  - Complications of PTCA stent replacement (rupture, dissection, thrombosis).

Co nt Ra i n Di Ca t i o n s

- Chronic congestive heart failure (CHF).
- Ischemic cardiomyopathy with no signs of angina or reversible ischemia.

PTCA versus CABG

PTCA is preferred over CABG when:

- Low-risk obstruction (single vessel, mild double vessel) is present, causing severe symptoms.
- Patient is at higher risk for complications from CABG.

PReoPeRat ive C onsiD eRat ions

- Evaluation for concurrent carotid disease (pre-CABG carotid endarterectomy may be required to reduce chances of perioperative stroke).
- Patencies of potential conduits are verified.
- Invasive monitoring (Swan–Ganz, central venous pressure [CVP], arterial line).
- Broad-spectrum antibiotics as needed.
- Aspirin discontinued 1–2 weeks preop.
- Warfarin discontinued 1 week preop.
- Antianginal meds continued until day of surgery.

WARD TIP

The IABP sits in the descending aorta (just distal to where the left subclavian takes off). It works by inflating during diastole and deflating during systole. Inflation increases coronary blood flow. Deflation creates a negative pressure gradient in the aorta, thereby reducing afterload.
Intra-aortic balloon pump (IABP) for those who need it (augments cardiac output [CO]).

Postoperative Considerations

Short-term:
- Continued monitoring of cardiac parameters.
- Chest tubes.
- Atrial fibrillation prophylaxis.
- Broad-spectrum antibiotics.

Long-term:
- Rehabilitation.
- Sternum heals in 3–6 months.
- Anticipate incisional pain.
- Risk factor modification (hyperlipidemia, smoking, sedentary lifestyle).
- Antiplatelet therapy (start the day after surgery).

Complications

- MI.
- Arrhythmias.
- Infection (particularly mediastinitis and sternal infection).
- Hemorrhage.
- Graft thrombosis.
- Sternal dehiscence.
- Tamponade.
- Postpericardiotomy syndrome.
- Stroke.

Pragnosis

- Average survival rate at 1, 5, 10, and 15 years are 97%, 92%, 81%, and 66%, respectively
- Risk factors for mortality: Severe LV dysfunction, advanced age, emergent CABG for AMI or UA, female gender.
- Multivessel disease and left main disease demonstrate greater survival with surgical intervention over medical therapy.
- IMA has 98% 1-year and 90% 10-year patency.
- Saphenous vein has 80–90% 1 year and 60–70% 10-year patency.

Valvular Heart Disease

Mitral Stenosis

tiology
- Rheumatic heart disease (most common).
- Congenital (rare).

epidemiology
More common in women.

Pathophysiology
- Mitral leaflets become thickened and calcified due to inflammation, resulting in commissural fusion in severe cases.
Leads to pulmonary congestion and pulmonary hypertension, left atrial dilation, atrial fibrillation, reduced CO, right ventricular (RV) hypertrophy, and secondary tricuspid regurgitation.

**Signs and Symptoms**
- Dyspnea, DOE (dyspnea on exertion).
- Rales.
- Cough.
- Hemoptysis.
- Systemic embolism (secondary to stagnation of blood in enlarged left atrium).
- Loud S1, opening snap.
- Accentuated right ventricle precordial thrust.
- Signs of RV failure.
- Hoarse voice and dysphagia (secondary to enlarged left atrium impinging on recurrent laryngeal nerve and esophagus).

**Diagnosis**
- Murmur is mid-diastolic with opening snap, low-pitched rumble.
- Best heard over left sternal border between second and fourth interspace.
- Chest x-ray (CXR) may show straight left heart border secondary to enlarged left atrium and Kerley B lines from pulmonary effusion.
- ECG may show left atrial enlargement, RV hypertrophy, atrial fibrillation.
- Echocardiography demonstrates diseased valve, fish-mouth opening, and decreased cross-sectional area; 1.5–3.5 cm², mild stenosis; 1.0–1.5 cm², moderate stenosis; less than 1.0 cm², severe stenosis (normal mitral valve cross-sectional area 4–6 cm²).
- Elevated transmitral pressure gradient > 10 mmHg (under normal conditions there is no transvalvular gradient).

**Treatment**
- **Medical:**
  - Asymptomatic patients need only endocarditis prophylaxis.
  - Symptomatic patients are treated with diuretics (to lower left atrial pressure), β-blocking agents, and/or calcium channel blocking agents (to maintain sinus rhythm).
  - Digoxin is helpful in controlling ventricular rate in patients who do go into atrial fibrillation.
  - Anticoagulation for atrial thrombus/fibrillation if present.
  - Percutaneous mitral valve balloon valvuloplasty if valve morphology allows.
- **Surgical:**
  - Indications for surgery:
    - NYHA Class III or IV symptoms.
    - Atrial fibrillation.
    - Worsening pulmonary hypertension.
    - Systemic embolization.
    - Infective endocarditis.
    - Class II patients > 40 of age.
  - Open commissurotomy:
    - Will suffice in 30–50% of cases.
    - Fused leaflets are incised, calcifications are debrided, problematic chordae are resected, papillary muscle heads may be split, and mitral ring added to prevent regurgitation (annuloplasty).
    - Valve replacement employed when excessive debridement would be required.
- Percutaneous balloon valvuloplasty not as effective in the long term, possibly because fused chordae cannot be corrected.
- Minimally invasive mitral valve surgery (port access technique) has recently been introduced.

**Prognosis**
- Ten years after commissurotomy, 7% require valve replacement.
- Yearly reoperation rates are 12% (balloon), 4% (commissurotomy), and 1.2% (valve replacement).
- Five-year survival after mitral valve replacement (MVR) 60–90%, 40–75% after 10 years (varies widely due to effect of risk factors, such as age, NYHA functional status, associated mitral insufficiency, additional need for CABG).

**Mitral insufficiency**

**Etiology**
- Papillary muscle dysfunction from either ischemia or infarction (post-MI papillary muscle rupture causes massive regurgitation).
- Rupture of chordae tendineae (can happen spontaneously in otherwise healthy individuals).
- Valve destruction—scarring from rheumatic heart disease or destruction from endocarditis.
- Prolapse (billowing of one or both leaflets) frequently progresses to valvular incompetence.

**Signs and Symptoms**
- Dyspnea.
- Fatigue.
- Weakness.
- Cough.
- Atrial fibrillation.
- Systemic emboli.
- Leads to pulmonary congestion, right-sided failure, left atrial dilation, atrial fibrillation, and volume overload of left ventricle. Cardiac output increases, then decreases.

**Diagnosis**
- Murmur is loud, holosystolic, high-pitched, apical radiating to the axilla.
- Wide splitting of S2 with inspiration (widening occurs in severe cases due to premature emptying of LV).
- S3 due to rapid filling of LV by blood regurgitated during systole.
- ECG shows enlarged left atrium.
- Echocardiography demonstrates diseased/prolapsed valve and can be used to quantify mitral regurgitation (MR). The severity of the regurgitation is gauged as a function of the distance from the mitral annulus that the regurgitant jet can be visualized (e.g., into the pulmonary veins) and by the width of the regurgitant jet. The regurgitation is scored on a scale from 1 (mild) to 4 (severe).
- Leaflet thickness of 5 mm or more increases the likelihood of prolapse.

**Treatment**
- Medical:
  - Not definitive but used until surgery or in poor surgical candidates.
  - Diuretics to reduce volume load, reduce LV diameter (and mitral annulus), and thus reduce regurgitant fraction.
Cornerstone of medical management is afterload reduction by vasodilators (mainly ACE inhibitors), thus favoring aortic forward flow.

- Anticoagulation for atrial fibrillation.
- Mitral insufficiency has a good prognosis if LV function is preserved, and patient needs closed follow up with serial echocardiograms.

**Surgical:**
- Valve replacement or repair.
- Indications for surgery: Symptoms despite medical management; severe MR with an identified structural abnormality, such as a ruptured chordae tendineae; development of pulmonary hypertension; or evidence of decline in left ventricular contractile function; atrial fibrillation; left atrium > 4.5–5 cm.

**Mitral valve reconstruction:**
- Involves resection of redundant areas of leaflets, chordal shortening, and ring annuloplasty (this corrects annular dilatation and stabilizes the repair).
- Preferable to MVR in degenerative disease; MVR better in advanced deformity not amenable to reconstruction (e.g., due to rheumatic disease).

**Prognosis**
- Better late survival in nonrheumatic patients undergoing reconstruction vs. replacement.
- Opposite in rheumatic patients.

**AoRtiC Stenosis**

**Etiology**
- Calcific aortic stenosis (secondary to heavy dystrophic calcification of a congenitally abnormal valve).
- Degenerative aortic disease (idiopathic, older population).
- Congenital stenosis.
- Bicuspid aortic valve.
- Rheumatic heart disease.

**Pathophysiology**
Obstruction of flow leads to left ventricular hypertrophy (LVH) (concentric type) and decreased LV compliance, LV dilation, and left ventricular failure (LVF).

**Signs and Symptoms**
Usually asymptomatic early in course. Then:

- Dyspnea
- Angina and syncope—particularly during exercise. Peripheral resistance falls; LV pressure remains the same due to stenotic valve; CO cannot maintain blood pressure (BP), causing syncope; low BP to coronary arteries causes angina.
- Heart failure.
- HTN (consider associated aortic coarctation).
- Symptoms are associated with reduction in the aortic valve area from the normal 3–4 cm$^2$ to < 1 cm$^2$.

**Diagnosis**
- Forceful apex beat with normally located point of maximal impulse (PMI).
- Loud systolic ejection murmur, crescendo-decrescendo, medium pitched, loudest at second right interspace, radiates to carotids.
- S4 (presystolic gallop) frequently present secondary to reduced LV compliance.
- Paradoxical splitting of S2.
- Narrow pulse pressure.
- ECG may show LV strain pattern, LV hypertrophy, with or without inverted T waves.
- Echocardiography demonstrates diseased valve and quantifies severity; > 1.5 cm² mild stenosis, 1.0–1.5 cm² moderate stenosis, and < 1.0 cm² severe stenosis.
- Calcification of aortic valve may be seen on CXR.
- Patients > 40 years of age, consider coronary angiogram to rule out concurrent coronary artery disease.
- Hematologic abnormalities associated with severe aortic stenosis: Low platelet function and decreased levels of von Willebrand factor.

**Treatment**

**Medical:**
- Avoid strenuous activity.
- Avoid afterload reduction.

**Surgical:**
- Indications for surgery:
  - Asymptomatic with high transvalvular gradient (> 50 mmHg) and LVH or declining EF.
  - Presence of symptoms: CHF is an indication of urgent intervention, while angina and syncope warrant elective surgical treatment.
  - Aortic valve area < 1.0 cm².
  - Aortic balloon valvuloplasty produces only temporary improvement as rate of restenosis is very high. Only potential role of valvuloplasty is in aged, frail, and possibly senile patients whose long-term survival is poor.
  - Valve replacement is definitive therapy. Nearly all patients attain symptomatic relief and improvement in EF while resolution of ventricular hypertrophy may require months.
  - Surgical mortality increases exponentially with decreasing LV function. Aortic valve replacement (AVR) in patients with CHF carries a mortality rate of up to 24%.
  - Intra-annular and supra-annular placement of prosthesis (latter for small annulus).
  - Ross procedure for AVR in patients with congenital aortic stenosis: Patient’s own pulmonary valve is substituted (autograft), while a cryopreserved homograft (cadaveric) is used to replace the pulmonary valve. No need for anticoagulation, plus good durability (20-year failure rate of 15%).

**Prognosis**

Ten-year survival after AVR > 80% except in high-risk patients (e.g., severely impaired LV function, NYHA Class IV, pulmonary hypertension).

**AoRtic Regurgitation**

**Etiology**
- Aortic root dilatation: Idiopathic (correlates with HTN and age), collagen vascular disease, Marfan’s syndrome.
- Valvular disease: Rheumatic heart disease, endocarditis.
Proximal aortic root dissection: Cystic medial necrosis (Marfan’s syndrome again), syphilis, HTN, Ehlers-Danlos, Turner syndrome, third trimester.
Aortitis: Rheumatoid arthritis, ankylosing spondylitis or Reiter disease.

Pathophysiology
Leads to LV dilation, eccentric hypertrophy, mitral insufficiency, cardiomegaly, CHF.

Signs and Symptoms
- Dyspnea, orthopnea, paroxysmal nocturnal dyspnea.
- Angina (secondary to reduced diastolic coronary blood flow due to elevated LV end-diastolic pressure).
- LVF.
- Wide pulse pressure.
- Bounding “Corrigan” pulse, “pistol shot” femorals, pulsus bisferiens (dicrotic pulse with two palpable waves in systole).
- Duroziez’s sign: Presence of diastolic femoral bruit when femoral artery is compressed enough to hear a systolic bruit.
- Hill’s sign: Systolic pressure in the legs > 20 mmHg higher than in the arms.
- Quincke’s sign: Alternating blushing and blanching of the fingernails when gentle pressure is applied.
- De Musset’s sign: Bobbing of head with heartbeat.

diagnosis
- High-pitched, blowing, decrescendo diastolic murmur best heard over second right interspace or third left interspace, accentuated by leaning forward.
- Austin Flint murmur: Observed in severe regurgitation, low-pitched diastolic rumble secondary to regurgitated blood striking the anterior mitral leaflet (similar sound to MR).
- A2 accentuated (due to high pulse pressure in the aorta at the beginning of ventricular diastole).
- Hyperdynamic down and laterally displaced PMI secondary to LV enlargement.
- ECG shows LV hypertroph, high peaked T waves, and prominent Q waves.
- Echocardiography demonstrates regurgitant valve.

treatment
- Medical:
  - Diuretics and afterload reduction: Afterload reduction achieved by ACE inhibitors. Nifedipine has been shown to delay the need for AVR.
  - Asymptomatic patients should have serial echocardiography to monitor for any systolic dysfunction or decreasing EF.
  - Endocarditis prophylaxis.
- Surgical:
  - Indications for surgery:
    - Asymptomatic with first sign of declining LV function or rapid increase in cardiac size. AVR to be performed before end-systolic dimension exceeds 55 mm.
    - Presence of symptoms.
  - Valve repair may be suitable for pure aortic insufficiency (no stenosis and no other valves involved) and in cases of aortic root aneurysm.
  - Valve replacement is necessary for severe cases and is the only definitive treatment.
Prognosis
- See Aortic Stenosis section.
- Valvular resection with annuloplasty: 10% reoperation rate at 2 years.

Tricuspid Stenosis

etiology
Rheumatic heart disease, congenital, carcinoid.

Signs and Symptoms
- Peripheral edema
- Jugular venous distention (JVD)
- Hepatomegaly, ascites, jaundice

diagnosis
- Murmur is diastolic, rumbling, low pitched.
- Murmur accentuated with inspiration.
- Accentuated precordial thrust of right ventricle.
- Diastolic thrill at lower left sternal border.
- Best heard over left sternal border between fourth and fifth interspace.
- Echocardiography demonstrates diseased valve and quantifies transvalvular gradient.

treatment
- Valve replacement for most cases.
- Commissurotomy with annuloplasty used for commissural fusion.

types of valve Prostheses

Mechanical Prostheses
- St. Jude.
- Offer greater durability (15-year failure rate 5%), but need for lifelong anticoagulation (with associated risk of hemorrhagic complications).
- A better choice for the young (fewer re-operations).

Bioprostheses
- Porcine or bovine xenografts.
- Fewer thromboembolic concerns (usually no need for anticoagulation) but less durable (15-year failure rate 50%).
- Better choice for the elderly.
- Calcification can complicate use in the young.

Lung Cancer

types

Small cell lung cancer:
- Represents 20% of all lung cancers and 80% of centrally located.
- Neuroendocrine in origin.
- Sensitive to chemotherapy (cisplatin and etoposide) and x-ray therapy (XRT).
Usually nonresectable at time of diagnosis (<5% candidates for surgery).
- Five-year survival: Very poor prognosis (2–4 months from diagnosis to death).
- Only T1, N0, M0 stage has 50% 5-year survival.

**Non-small cell lung cancer:**
- Includes squamous cell (SCCA), large cell, and adenocarcinoma (ACA).
- ACA most common lung cancer (45% of all lung cancers), and 75% are peripherally located.
- ACA metastasizes earlier than SCCA and more frequently to the central nervous system.
- SCCA represents 30% of all lung cancers and two thirds are centrally located.
- Large cell carcinoma accounts for 10% of all lung cancer, tends to occur peripherally, and metastasizes relatively early.
- Chemotherapy for non-small cell cancer (stage II or higher)—carboplatin, paclitaxel.
- Treated with surgery (debulking).
- Prognosis varies with stage.

**ePidiology**
- Leading cause of cancer death in both men and women in the United States.
- Cases have been decreasing in men but increasing in women.
- Smoking is by far the most important causative factor in the development of lung cancer.

**etiology**
- Smoking
- Passive smoke exposure
- Radon gas exposure
- Asbestos
- Arsenic
- Nickel

**signs and symptoms**
- Cough.
- Hemoptysis.
- Stridor.
- Dypneu.
- Hoarseness (recurrent laryngeal nerve paralysis).
- Postobstructive pneumonia.
- Dysphagia.
- Associated (paraneoplastic) syndromes (see Table 24-1).

**Diagnosis**

See section on diagnostic evaluation of a lung mass below.

**tReatment**

The two main types of lung cancer, small cell and non-small cell cancer, have different responses to radiotherapy, chemotherapy, and surgery (see Table 24-2).
**Syndromes associated with Lung Cancer**

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horner syndrome</td>
<td>Sympathetic nerve paralysis produces enophthalmos, ptosis, miosis, ipsilateral anhidrosis</td>
</tr>
<tr>
<td>Pancoast’s syndrome</td>
<td>Superior sulcus tumor injuring the eighth cervical nerve and the first and second thoracic nerves and ribs, causing shoulder pain radiating to arm</td>
</tr>
<tr>
<td>Superior vena cava syndrome</td>
<td>Tumor causing obstruction of the superior vena cava and subsequent venous return, producing facial swelling, dyspnea, cough, headaches, epistaxis, syncope. Symptoms worsened with bending forward, and on awakening in the morning</td>
</tr>
<tr>
<td>Syndrome of inappropriate antidiuretic hormone (SIADH)</td>
<td>Ectopic arginine vasopressin (AVP) release in the setting of plasma hyposmolality, producing hyponatremia without edema. Also caused by other lung diseases, CNS trauma or infection, and certain medications</td>
</tr>
<tr>
<td>Eaton–Lambert syndrome</td>
<td>Presynaptic nerve terminals attacked by antibodies, decreasing acetylcholine release, treated by plasmapheresis and immunosuppression; 40% associated with small cell lung cancer, 20% have other cancer, 40% have no cancer</td>
</tr>
<tr>
<td>Trousseau’s syndrome</td>
<td>Venous thrombosis associated with metastatic cancer</td>
</tr>
</tbody>
</table>

**Distinction between Small and Non-Small Cell Lung Cancer**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Small Cell Lung Cancer</th>
<th>Non-Small Cell Lung Cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Histology</td>
<td>Small dark nuclei, scant cytoplasm</td>
<td>Copious cytoplasm, pleomorphic nuclei</td>
</tr>
<tr>
<td>Ectopic peptide production</td>
<td>Gastrin, ACTH, AVP, calcitonin, ANF</td>
<td>PTH</td>
</tr>
<tr>
<td>Response to radiotherapy</td>
<td>80–90% will shrink</td>
<td>30–50% will shrink</td>
</tr>
<tr>
<td>Response to chemotherapy</td>
<td>Complete regression in 50%</td>
<td>Complete regression in 5%</td>
</tr>
<tr>
<td>Surgical resection</td>
<td>Not indicated</td>
<td>Stage I, II, IIIA</td>
</tr>
<tr>
<td>Included subtypes</td>
<td>Small cell only</td>
<td>Adenocarcinoma, squamous cell, large cell, bronchoalveolar</td>
</tr>
<tr>
<td>5-year survival rate—all stages</td>
<td>5%</td>
<td>11–83%</td>
</tr>
</tbody>
</table>

ACTH, adrenocorticotropic hormone; AVP, arginine vasopressin; ANF, atrial natriuretic factor; PTH, parathyroid hormone.

**Diagnostic Evaluation of a Lung Mass**

**Plain Film**
- Most malignant nodules seen by 0.8–1 cm in diameter (may be seen smaller) (see Figure 24-1).
- Comparison with previous films whenever possible.
- Nodules stable for 2 years need no further evaluation.
- Plain film may be the only imaging modality necessary if there is obvious bony metastasis or bulky, contralateral mediastinal adenopathy.
- If abnormal nodule present, need computed tomography (CT).

**Paraneoplastic Syndromes**
- Small cell carcinoma: ACTH, ADH.
- Squamous cell carcinoma: PTH-related peptide.
- ACTH secretion of small cell carcinoma is the most common paraneoplastic syndrome.
Ct
- Provides better characterization and location of mass as well as detecting mediastinal invasion.
- Mediastinal lymph nodes can also be assessed, especially if greater than 1 cm.
- Should be extended to include liver and adrenal glands as these are frequent sites of metastasis.

Bronchoscopy
- Method of choice of centrally located masses (squamous cell and small cell).
- Specimens can be obtained via direct biopsy of visualized lesions, brushings, washings, or transbronchial needle aspiration (TBNA).
- The most important application of TBNA is staging of mediastinal lymph nodes.
- Risks of this procedure are respiratory arrest, pneumothorax, and bleeding.
- Although this method works well for centrally located lesions, it is poor when it comes to peripheral lung nodules.

Trans-thoracic Needle Biopsy (t NB)
- Method of choice for peripherally located nodules. Most are CT guided.
- Sensitivity: 70–100%.

Thoracentesis
Test of choice for patients with pleural effusion and suspected malignancy.
**Solitary Pulmonary Nodule**

- A single small (<3 cm) intraparenchymal opacity that is reasonably well marginated.
- Most will be benign.
- Benign: Granulomas, hamartomas, or intrapulmonary lymph nodes.
- Malignant: Bronchogenic carcinoma.

**Small Cell Lung Cancer**

- Seventy percent metastatic at the time of diagnosis.
- Generally considered inoperable for cure.
- Two stages: Limited and extensive.
  - Limited: Confined to a single radiation portal.
  - Extensive: All others.

**Non-Small Cell Lung Cancer**

- Need to determine resectability: Chest CT and search for distant metastases.
- Malignant pleural effusion precludes curative resection.
- Tumor-node-metastasis (TNM) staging system.

---

**Thoracic Aortic Aneurysms**

**Definition**

- **Aneurysm**: Ballooning defect in the vessel wall.
- **Dissection**: Tear of the arterial intima.

**Types**

- **Degenerative**:
  - Due to abnormal collagen metabolism.
  - Seen with Marfan’s and Ehlers–Danlos syndromes (usually ascending aortic aneurysms).
- **Atherosclerotic** (usually descending aortic aneurysms): Due to remodeling and dilatation of the aortic wall.

**Anatomical Classification**

- DeBakey type I: Ascending and descending aorta.
- DeBakey type II: Ascending aorta only.
- DeBakey type III: Descending aorta only.
- Stanford A: Ascending aorta (same as DeBakey I/II).
- Stanford B: Descending aorta (same as DeBakey III).
- Ascending aorta and aortic arch aneurysms are worse than descending aortic aneurysms.
- Expansion rate is –0.56 cm/year for arch aneurysms and –0.42 cm/year for descending aorta.

**Epidemiology**

- Six per 100,000 a year.
- Male-to-female ratio is 2:1.
- Familial clustering.
- Patients tend to be younger than those with abdominal aortic aneurysm (AAA).
signs and symptoms of expansion or rupture

- “Tearing” or “ripping” chest pain radiating to the back.
- Acute neurologic symptoms (syncope, coma, convulsions, hemiplegia).
- Palpable thrust may be seen in right second or third intercostal space.
- Pulsating sternoclavicular joint may be seen (secondary to swelling at the base of the aorta).
- Hoarseness.
- Stridor.
- Dysphagia.
- New aortic regurgitation murmur.
- Hemoptysis or hematemesis.
- Absent or diminished pulses.

Diagnosis

- CXR (Figure 24-2):
  - Widened mediastinum.
  - Abnormal aortic contour.
  - “Calcium sign”: Reflects separation of intimal calcification from the adventitial surface.
- Contrast CT (Figure 24-3):
  - Sensitivity 85–100%.
  - Specificity 100%.
- Magnetic resonance imaging (MRI):
  - Excellent sensitivity and specificity.
  - Gives information about branch vessels that CT does not.
  - No need for contrast.
  - Limited to stable patients.
- Angiography (Figure 24-4):
  - Requires contrast dye like CT.
  - Invasive.

Figure 24-2. Thoracic aneurysm diagnosed on CXR.
Transesophageal echocardiography (TEE):
- Presence of intimal flap separating the true from the false lumen.
- Features of the false lumen: Larger in diameter, slower blood flow velocity.
- Can be used in relatively unstable patients as well.

Transthoracic echocardiography (TTE):
- Available at bedside.
- Noninvasive.
- Suitable for unstable patients.
- Requires operator expertise.
- Moderate ability to detect ascending and arch dissections; poor for detecting descending arch dissection.

Treatment and Prognosis

Medical:
- Control HTN with nitroprusside or labetalol.
- Parenteral analgesia.

Surgical:
- For ruptured aneurysms, it is the only definitive therapy. Carries very high risk of mortality. Most patients die before reaching the operating room. Of those that reach the OR, < 50% survive.

Exam Tip
Major complication of any thoracic aneurysm repair is paraplegia. Key is to avoid perioperative hypotension.
Surgical repair is considered for acutely symptomatic, ascending aortic and aortic arch aneurysms > 5.5 cm, rapid increase in size (growth rate > 0.5 cm/year). Mortality rate is 10–15%.

For degenerative aneurysms, the entire aortic root must be replaced.

Atherosclerotic aneurysms can be repaired either via open approach (median sternotomy approach for ascending arch and posterolateral thoracotomy for descending arch) or via endovascular technique.

Ascending and descending arch aneurysms are repaired with patient under cardiopulmonary bypass, anticoagulation, and in mild-moderate hypothermia.

Aortic arch aneurysms are repaired with patient in circulatory arrest and profound hypothermia.

## Complications

- Hemorrhage
- Paraplegia
- Stroke
- MI
- Visceral ischemia
- Death

## Thoracoabdominal Aneurysms

### Classification

Crawford classification:

- Type I: Most of descending thoracic aorta and abdominal aorta proximal to renal arteries.
- Type II: Most of descending thoracic aorta and abdominal aorta distal to renal arteries.
- Type III: Distal one half of descending thoracic aorta and abdominal aorta proximal to renal arteries.
- Type IV: Distal one half of descending thoracic aorta and abdominal aorta distal to renal arteries.

### Diagnosis

Made incidentally (routine physical exam or imaging for other reasons) or on postmortem exam (for ruptured ones).

### Treatment

- Elective repair undertaken after weighing risk vs. benefit.
- Open surgical approach decreasing in practice:
  - Type I: Thoracic incision.
  - Types II and III: Incision from sixth intercostal space into abdomen.
  - Type IV: Retroperitoneal incision from left flank to umbilicus.
- Endovascular repair of aortic aneurysm (EVAR):
  - Widespread use in the last decade except in urgent/emergent cases.
  - Need to have good preoperative imaging to evaluate anatomy of the aneurysm.
  - Suitable landing zones proximal and distal to the graft: 1.5–2.0 cm, no major arterial branches near the neck of aneurysm.
If a major artery (left subclavian, celiac axis, superior mesenteric artery, renal artery) is covered by the endovascular graft, then need a hybrid operation to bypass the covered artery.

Complications:
- Endoleak (leak through or around the graft) results in persistent enlargement of the aneurysm sac
- Aortic dissection
- Aneurysm rupture
- Stent collapse
- Stent migration
- Injury to femoral and external iliac arteries while graft insertion

**Prognosis**

Overall mortality < 10%.

**Complications**

- Risk of paraplegia 5–10%.
- Same as for thoracic aneurysm repair.
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Definitions

**Autograft:** Donor and recipient are same individual.

**Allograft:** Donor and recipient belong to same species but have different genetic makeup.

**Isograft:** Donor and recipient are genetically identical, for example, identical twin.

**Xenograft:** Donor and recipient are of different species.

**Orthotopic:** Transplant graft placed into its anatomic position.

**Heterotopic:** Transplant graft placed at different site.

Brain Death

Brain death is defined as irreversible loss of all functions of the brain including brainstem.

**Adult Criteria**

1. No cerebral function: Patient is in a deep coma, unresponsive to stimuli.
2. Absence of brainstem reflexes: No evidence of cranial nerve function; lack of reflexes (pupillary, corneal, cold water caloric, doll's eyes, pharyngeal and tracheal).

- **Requirements:** Normothermia, no central nervous system (CNS) depressants or neuromuscular blockers in effect. No severe electrolyte/acid base or endocrine disturbances.
- **Optional tests:** Electroencephalogram, angiography, nuclear scan transcranial Doppler.

No N-Heart-Beating donors

Applies to patients with cardiac arrest near hospital, and to patients requesting removal of life support or in whom death is expected.

Donor Management

**Goal:** To optimize organ function avoid hypoxia, hypotension, and fever.

Transplant Immunology

**Basics**

Major histocompatibility complexes (MHCs) present peptide antigens to T cells and are the major target of activated lymphocytes.

**MHCi**

- Found on all nucleated cells except cells in retina and brain. High level on antigen presenting cells.
- Consists of MHC coded α chain and non-MHC coded β chain, β2 microglobulin.
- Presents cytosolic proteins and viruses.
- Primary target for CD8 T cells in graft rejection.
- Gene loci: A, B, C.

**MHCii**

- Constitutively expressed on Antigen presenting cells like B-lymphocytes, dendritic cells, macrophages, and other phagocytic cells, also can be induced on other cells by cytokines.
- Composed of alpha and beta chains.
- Binds peptides from extracellular proteins.
- Primary target for T-helper cells.
- Gene loci: DR, DQ, DP.

**MHCiii**

Class 3 genes encode complement proteins.

**Tissue Typing**

- Determination of MHC alleles in an individual to minimize differences in histocompatibility.
- Of above alleles, only A, B, and DR are used in routine tissue typing.
- Of these three genes, DR is most important to match, followed by B and then A, as evidenced by United Network for Organ Sharing (UNOS) data regarding graft survival.

**Cross Matching**

- Test for preformed cytotoxic antibody in serum of potential recipient.
- Donor lymphocytes are cultured with recipient serum in the presence of complement and dye.
- Lymphocyte destruction is evidenced by uptake of dye, indicating a positive crossmatch.
- A positive crossmatch is generally a contraindication to transplant as hyperacute rejection is likely.
- Panel Reactive Antibodies (PRA): PRA refers to the percentage of an antibody-screening panel with which the patient’s serum reacts. High PRA means higher chances of rejection.

**Immunosuppression**

**Steroids**

- Most commonly prednisone (PO) or methylprednisolone (IV).
- Binds to intracellular receptor, is transported into nucleus—DNA-binding protein that works to limit, ultimately, the inflammatory response by blocking nuclear factor-κB (NF-κB), interleukin-1 (IL-1), tumor necrosis factor-α (TNF-α), interferon, and phospholipase A₂, as well as histamine and prostacyclin.

**Exam Tip**

Graft survival is improved with matching for kidney, pancreas, and heart transplants, but donor-specific crossmatching does not have relevance for liver transplantation, as graft survival is not affected by positive crossmatch.

**Exam Tip**

Immunosuppression is associated with increased risk of infection and malignancies.
- Side effects: Impaired glucose tolerance, impaired wound healing, fluid retention, insomnia, depression, nervousness, and psychosis.
- Chronic effects: Cushing’s syndrome, increased risk for peptic ulcer disease, osteoporosis.

### CyCloporsiNe (CSA)
- Calcineurin inhibitor: CsA binds to cyclophilin and, together, they bind to calcineurin-calmodulin complex, blocking calcium-dependent phosphorylation and activation of NF-AT, thereby preventing transcription of several genes needed for T-cell activation, including IL-2. Put simply, it inhibits T-cell activity.
- P450 metabolism: Levels are increased by P450 inhibitors such as ketoconazole, erythromycin, calcium channel blockers, and decreased by P450 inducers like rifampin, phenobarbital, and phenytoin.
- Side effects: Nephrotoxicity (dose related, based on vasoconstriction of proximal renal arterioles), hemolytic-uremic syndrome (uncommon), hypertension, tremors, headache, paresthesia, depression, confusion, seizures (rare), hypertrichosis, gingival hyperplasia, and hepatotoxicity.

### MyCoHeNo1AteMoFeil (MMF)
- Prodrug of mycophenolic acid (MPA): Noncompetitive reversible inhibitor of inosine monophosphate dehydrogenase—thereby halts purine metabolism, blocks proliferation of T and B cells, suppresses B-cell memory, and inhibits antibody formation.
- Side effects: Mild diarrhea or nausea.

### AzAtHiopriNe
- Inhibits DNA synthesis by alkylating DNA precursors and depleting cell of adenosine.
- Side effects: Leukopenia, hepatotoxicity.

### tAgOliliMus (FK-506)
- Drug of choice for liver transplant.
- Calcineurin inhibitor: Works in similar fashion to CsA, but binds initially to FK-binding protein.
- A hundred times more potent than CsA.
- Side effects as for CsA, though more neurotoxic and diabetogenic, and fewer cosmetic effects.

### ANiHyMoCytegleBoLiNi (Atg)
- Usually used for treatment of steroid-resistant rejection (SRR), but may also be used for induction therapy.
- Polyclonal antihuman γ-globulin extracted from horse sera of horses immunized with thymus lymphocytes.
- Side effects include fever and chills (20%), thrombocytopenia, leukocytopenia, rash (15%).

### oKt3
- Monoclonal antibody to CD3, a signal transducer on human T cells, thereby prevents transduction of antigen binding; also downregulates T-cell receptor (TCR).
Rejection

Hyper Acute

- **Cause:** Presensitization of recipient to donor antigen.
- **B cells** are responsible for antibody-mediated hyperacute rejection when the transplant contains an antigen that the recipient B cells have seen before.
- **Timing:** Immediately following graft reperfusion.
- **Mechanism:** Antibody binds to donor tissue, initiating complement-mediated lysis, which has a procoagulant effect. End result is thrombosis of graft.
- **Prevention:** ABO typing and negative crossmatch prevent hyperacute rejection in >99% of patients.
- **Treatment:** None (graft removal).
- **Outcome:** Graft failure/loss.

Acute

- **Cause:** Normal T-cell activity (would ultimately affect every allograft were it not for immunosuppression).
- **Timing:** Between postoperative day 5 and postoperative month 6.
- **Mechanism:** T cells bind antigens in one of two ways—directly through TCR or after phagocytosis and presentation of donor tissue, resulting in T-cell infiltration of graft with organ destruction.
- **Diagnosis:** Generally by decreased graft function and by biopsy.
- **Histology:** Lymphocytic infiltrate and/or graft necrosis. Liver rejection also characterized by eosinophilic infiltrate.
- **Prevention:** Minimize mismatch of MHC; usual immunosuppression; monitor for organ dysfunction as signs of rejection that may otherwise be asymptomatic.
- **Treatment:** High-dose steroids; OKT3 or ATG when SRR after 2 days.
- **Outcome:** Ninety to 95% of transplants are salvaged with treatment.

Chronic

- **Cause:** Cumulative effect of recognition of MHC by recipient immune system.
- **Timing:** Insidious onset over months and years.
- **Mechanism:** Recipient’s immune system recognizes donor MHC; other factors not yet understood.
- **Diagnosis:** Biopsy.
- **Histology:** Parenchymal replacement with fibrous tissue, some lymphocytic infiltrate, and endothelial destruction.
- **Prevention:** None known.
- **Treatment:** None.
- **Outcome:** Graft failure/loss.

Risk of Malignancy

- Overall incidence in kidney transplant recipients: 6%—lymphoma, skin cancer, genital neoplasms.
Posttransplant lymphoproliferative disease (PTLD): Caused by Epstein–Barr virus (EBV), leads ultimately to monoclonal B-cell lymphoma/treatment to lower or stop immunosuppression and restore immunity.

Organ Preservation

Maximum Times for Each Organ

- Heart: 4–6 hours.
- Lung: 4–6 hours.
- Liver: 8–12 hours.
- Pancreas: 12–18 hours.
- Kidney: 24–28 hours.

Kidney Transplantation

Background

Most common solid organ to be transplanted.

Causes of End-stage Renal Disease (ESRD)

- Diabetes
- Hypertension
- Glomerular nephritis
- Congenital anomalies
- Urologic abnormalities
- Dysplasia
- Focal segmental glomerular sclerosis

Types of Donors

- Cadaveric.
- Living:
  - May be related or unrelated.
  - Decreased warm ischemia time.
  - Associated with less delayed graft function and better outcome.
  - Donor mortality: 0.02–0.03%.
  - Living donor evaluation: Rule out potential donors with:
    - Diabetes, hypertension, malignancy, coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), renal disease, human immunodeficiency virus (HIV), hepatitis B/C, age < 18.
    - Genitourinary (GU) anomalies assessed by proteinuria > 300 mg/24 hr or creatinine clearance rate (CCr) < 80 mL/min, urologic abnormalities of donor kidney.

Appropriate Recipient

- General health assessment:
  - Identify comorbidities such as heart disease, COPD, and diabetes.
  - Assess ability to handle immunosuppression and compliance.

Exam Tip

Warm ischemia time should be minimized because it leads to rapid decline in adenosine triphosphate (ATP) and therefore decrease in biosynthetic reactions, a redistribution of electrolytes across cell membranes, and continuation of biodegradation reactions leading to acidosis and ultimately loss of organ viability.

Exam Tip

Morbidity and mortality associated with dialysis: A 49-year-old with end-stage renal disease (ESRD) on dialysis has an expected duration of life of 7 years, compared to 30 years for a healthy 49-year-old.

Exam Tip

Decreased quality of life in ESRD is associated with time commitment for dialysis and increased number of hospital days.

Exam Tip

Cardiovascular disease is responsible for 50% of dialysis patients’ deaths, and infection accounts for 15–30%.
Laboratory evaluation consisting of chemistries, complete blood count (CBC), urinalysis, serologies for hepatitis B and C, cytomegalovirus (CMV), and HIV.

- Conduct ECG and chest x-ray (CXR).
- Further cardiac workup based on need.
- Psychosocial evaluation regarding compliance.
- May need bilateral nephrectomy for recurrent urinary tract infections (UTIs) or polycystic kidney disease (PCKD).
- See Table 25-1 for contraindications to kidney transplantation.
- The donor kidney is usually placed in the iliac fossa with vascular anastomosis to the iliacs and the native kidney of the recipient need not be removed excepting for PCKD and recurrent UTIs.

**Early Complications**

- Delayed graft function.
  - Evidenced by oliguria or anuria (assess volume status, ensure that Foley is working; once checked, but still anuric/oliguric, Doppler ultrasound [US] indicated to assess blood flow). If blood flow is adequate, look for urine leak or obstruction at ureterovesicular junction (UVJ) with US or renal scan. Once all workup negative, diagnosis is delayed graft function.
  - Management may include dialysis in postoperative period.

  **Graft thrombosis:**
  - Requires immediate reoperation to save transplant.
  - Diagnosis indicated by abrupt cessation of urine output.
  - May assess with Doppler US.

  **Urine leak:**
  - Usually at UVJ.
  - **Diagnosis:** Decreased urine output, lower abdominal pain, scrotal or labial edema, rising creatinine.
  - **Tests:** US with fluid aspiration and analysis, renal scan with extravasation of radioisotope.
  - **Treatment:** Reexploration and repair.

**Late Complications**

- **Ureteral stricture:**
  - Rising creatinine and hydronephrosis on US.
  - Distal stricture result of rejection or ischemia; antegrade pyelogram best diagnostic tool.
  - **Treatment:** Balloon dilatation; longer ones require surgical repair.

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**Compliance Specific to Kidney Transplantation**

- **Contraindications to Kidney Transplantation**

<table>
<thead>
<tr>
<th>Absolute Contraindications</th>
<th>Relative Contraindications</th>
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<tr>
<td>Cancer (other than squamous cell or basal cell carcinoma of skin)</td>
<td>Sickle cell disease</td>
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<tr>
<td>Infection (HIV, tuberculosis)</td>
<td>Likely to be noncompliant</td>
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<tr>
<td>Cirrhosis (chronic active)</td>
<td>Ischemic heart disease severe, without possibility of coronary artery bypass graft or angioplasty</td>
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<tr>
<td>Ongoing drug use</td>
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</table>
Renal artery stenosis:
- Ten percent of renal transplants within first 6 months.
- **Presentation:** Hypertension, fluid retention.
- **Diagnosis:** Angiogram, US, magnetic resonance angiography (MRA).
- If distal to anastomosis, may be secondary to rejection, atherosclerosis, clamp or other iatrogenic injury. Occurs more frequently with end-to-end anastomoses.
- **Treatment:** > 80% correctible with angioplasty; others require surgical repair.
- **Donor complications:** UTI, wound infection, pneumothorax.

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**Pancreas Transplantation**

**Indications**
- Type 1 (insulin-dependent) diabetes mellitus.
- Intensive insulin therapy may slow the rate of secondary complications of diabetes, but at increased risk of hypoglycemic episodes.

**Exclusions for recipients**
- Significant CAD.
- Severe peripheral vascular disease (PVD) resulting in amputations.
- Severe visual impairment.
- Untreated malignancy.
- Active infection.
- HIV.

**Exclusion criteria for donors:**
- Pancreatic disease and type 1 DM.

**Timing of Operation**
- **Pancreas transplant (5%):** For the nonuremic diabetic patient with minimal or no evident nephropathy.
- **Pancreas transplant after kidney transplant (PAK) (15%):** Previously underwent kidney transplant, but now have poor glucose control or progression of diabetes complications.
- **Simultaneous pancreas and kidney transplants (SPK) (80%):** In diabetic and uremic patients.

**Drainage Options**
- **Bladder drainage:**
  - Advantage: Urinary amylase may be used as a sign of rejection; rejection of exocrine pancreas precedes the endocrine pancreas.
  - Up to 25% may require conversion to enteric drainage.
  - Avoids bacterial contamination.
- **Enteric drainage:**
  - Avoidance of postop GU complications that affect 30% of bladder-drained patients.
  - Avoidance of chronic dehydration.
  - No need for bicarbonate replacement.
  - Equal efficacy, graft survival, morbidity.
In SPK, rejection usually involves pancreas and kidney. May diagnose by creatinine and renal biopsy.

Enteric drainage more common in US.

**Complications (specific to Pancreatic Transplant)**
- Thrombosis, venous more common than arterial
- Bleeding
- Intra-abdominal abscess
- Pancreas specific complications like pancreatitis/pseudocysts/fistulas
- If bladder drainage has cystitis/hematuria/metabolic acidosis/diarrhea/dehydration

**Liver Transplantation**

**Indications**
- Irreversible liver failure.
  - **Chronic (more common):**
    - Cirrhosis (posthepatitic/alcoholic).
    - Primary and secondary biliary cirrhosis.
    - Primary sclerosing cholangitis.
    - Metabolic defects (α1-antitrypsin deficiency, amyloidosis, hemochromatosis, sarcoidosis, tyrosinemia, ornithine transcarabinase deficiency, Crigler–Najjar).
    - Malignancy (hepatocellular carcinoma [HCC] or cholangiocarcinoma).
    - Others: Biliary atresia, cystic fibrosis.
- **Acute or fulminant:**
  - Viral or alcoholic hepatitis.
  - Wilson’s disease.
  - Hepatotoxic drugs (e.g., acetaminophen overdose).
  - Contraindications.
  - Multisystem organ failure/life-threatening systemic disease.
  - Severe cardiopulmonary disease.
  - Sepsis secondary to nonhepatic source.
  - Metastatic malignancy.
  - Noncompliance with medical therapy/active drug/alcohol abuse.
  - Severely impaired neurologic status.

**Types of Liver Transplantation**
- Cadaveric: Can also be split between two recipients
- Living donor transplantation

**Evaluation**
- The Model for End-Stage Liver Disease (MELD) score is used for adult liver transplant assessment (uses patient’s values for bilirubin, international normalized ratio [INR] for prothrombin time, and creatinine). Also takes into consideration if patients are being dialysed. It is a numerical score from 6 to 40 and gives a score based on how urgently patient needs liver transplant in next 3 months.
- Preoperative control of:
  - Variceal bleeding: Transjugular intrahepatic portosystemic shunt (TIPS) when needed.
  - Ascites: Diuretics and/or paracentesis.
**Complications**

- Graft failure: Usually secondary to primary nonfunction, recurrence of disease, biliary or vascular complications (not generally due to rejection).
- Systemic complications like renal failure, cardiopulmonary complications.
- Rejection occurs in first 3 months posttransplant with 50% incidence, but is well treated with steroids or antilymphocyte therapy (indicated by elevated liver function tests [LFTs], particularly γ-glutamyl transpeptidase [GGTP]).

**Small Bowel Transplantation**

**Indications**

Life-threatening complications due to intestinal failure and complications related to parenteral nutrition.

- Adults: Short bowel syndrome, due to Crohn’s disease, mesenteric thrombosis, trauma.
- Children: Short bowel syndrome, due to necrotizing enterocolitis, intestinal pseudo-obstruction, gastroschisis, volvulus, intestinal atresia.

**Operation**

- Isolated intestinal failure: Isolated intestinal transplant.
- With liver failure: Liver-intestine combined transplant.
- Sometimes, multivisceral transplant: Liver, pancreas, duodenum, small intestine.
- Stoma usually placed for monitoring and biopsies.

**Complications**

- Graft-versus-host disease (GVHD): Prevent with immunosuppression and/or pretreatment of donor.
- Rejection: More difficult to treat than in other organs; newer agents may prove to be more useful than older ones (tacrolimus based).
  - CMV infection
  - Diagnosed by fever, abdominal pain, elevated white count, ileus, gastrointestinal (GI) bleed, positive blood cultures; also by biopsy showing cryptitis, villi shortening, mononuclear infiltrate.

**Cardiac Transplantation**

**Indications**

- Severe cardiac disability on maximal medical therapy (multiple hospitalizations for congestive heart failure [CHF], New York Hospital Association class III [NYHAIII] or IV, or peak oxygen consumption < 12–14 mL/kg/min).
- Symptomatic ischemia or recurrent ventricular arrhythmias refractory to usual therapy, with left ventricular ejection fraction (LVEF) < 30%, or with unstable angina and not a candidate for CABG or percutaneous transluminal coronary angioplasty (PTCA).
- Severely symptomatic hypertrophic or restrictive cardiomyopathy
- All surgical alternatives already excluded.
Co Nd i Ca t i o ns

- Irreversible, severe, pulmonary hypertension, renal, or hepatic dysfunction.
- Unstaged or incompletely staged cancer.
- Active infection.
- Psychiatric illness.
- Severe systemic disease.

e v A l u A t i o N

Matching/compatibility based on:
1. ABO compatibility.
2. Body size.
3. HLA crossmatching for presensitized heart transplant recipients.

Co M p l i C A t i o N s

- The most common cause of perioperative death is infection (50%). Other common causes are pulmonary hypertension and nonspecific graft failure.
- Infection:
  - Peak incidence: Early postoperative period.
  - Most patients will have an infection at some point in the first five postoperative years.
- Rejection:
  - At least 75% incidence in first three posttransplant months.
  - Diagnosis confirmed by endomyocardial biopsy (via right internal jugular).
- Histology: Lymphocyte infiltration and/or myocytic necrosis.
- Treatment: Pulse steroids if early on (later in course, mild rejection treated by 3-day increased dose of oral prednisone); refractory rejection treated with ATG or OKT3.
- Chronic rejection: Concentric intimal proliferation with smooth muscle hyperplasia yielding atherosclerosis more diffuse than in nontransplanted hearts.
- Prevalence of CAD: 25% at 1 year and up to 50% at 5 years. Because of denervation, patients do not develop angina.
- Yearly angiogram recommended. Alternative for surveillance is intravascular US.
- Cancer: Increased risk, with immunosuppression, of skin, vulvar, or anal cancer; B-cell lymphoproliferative disorder (BLPD) and cervical intraepithelial neoplasia (CIN).

Lung Transplantation

t y p e s o f o p e r A t i o N s A N d i Nd i C a t i o N s

- Single lung for fibrotic lung disease: Pulmonary fibrosis, emphysema, bronchopulmonary dysplasia, primary pulmonary hypertension (without cardiac dysfunction), posttransplant obliterator bronchiolitis.
- Bilateral for cystic fibrosis, bronchiectasis, COPD.
- Heart-lung for pulmonary vascular disease, end-stage lung disease with cardiac dysfunction.
- Lobar lung, to increase donor pool from living related and cadaveric donors.

WARD TIP

Typical scenario: A 53-year-old woman who is status post liver transplant calls your office asking what she can take for some musculoskeletal pain. Think: Patient is on tacrolimus, which ultimately causes renal insufficiency in most patients. Do not give anything that could potentiate its nephrotoxicity. First-line drug for pain would be acetaminophen, standard doses of which a transplanted liver should be able to tolerate.

CARDIOVASCULAR GRAFT VASCULOPATHY

Cardiac transplant: Cold ischemic time up to 6 hours may be tolerated, though 3–4 is ideal (2 hours maximum for patients with pulmonary hypertension).

Cardiac graft vasculopathy is a major cause of late graft failure. It is an aggressive form of atherosclerosis.

Transplanted heart has increased sensitivity to catecholamines, with increased density of adrenergic receptors with loss of norepinephrine uptake; cardiac output and index remain at low normal, with adequate but abnormal exercise response (increase in heart rate is usually delayed).

Patients have increased risk of cholelithiasis and strong consideration should be given to pre and posttransplant surveillance.
**Contraindications**

- Significant systemic disease, including hepatic or renal disease.
- Active infection.
- Malignancy.
- Noncompliance.
- Current smoking.
- Chronic hepatitis B/C and HIV.

**Evaluation**

- ABO compatible.
- Infection-free donor and recipient.
- Donor: Good pulmonary gas exchange, no smoking history, similar lung volume, where possible.

**Complications**

**Acute rejection:**
- Sixty to 70% incidence in first postoperative month.
- Diagnosed by clinical parameters: Fever, dyspnea, decreased Pao₂, decreased forced expiratory volume in 1 second (FEV₁), CXR with interstitial infiltrate.
- Confirm with biopsy via bronchoscopy: Lymphocytic infiltrate to varying degrees.

**Infection:** Early, predominance of gram-negative bacillus (GNB) infection.
- **Viral:** CMV, treated with ganciclovir, also EBV.
- **Fungal:** Candida, Aspergillus.
- **Protozoan:** Pneumocystis jiroveci pneumonia.

**Outcomes:**
- Kidney transplants have best outcomes, 5-year graft survival for living donor is 81% (90% patient survival) in the United States.
- Small bowel and lung have the lowest rate of organ survival.

**Exam Tip**

CMV ocurs at 75–100% incidence in cardiac transplant patients and has been identified as trigger for graft-related atherosclerosis (treated with ganciclovir and hyperimmune globulin).

Chronic rejection manifests as bronchiolitis obliterans, seen in upto 50% after 5 years. Dense eosinophilic scarring of bronchioles. No proven beneficial treatment.
# The Genitourinary System

Anatomy of the Genitourinary (GU) System
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- Scrotum
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Transitional Cell Carcinoma of the Collecting System and Ureters

Bladder Cancer
- Signs and Symptoms
- Diagnosis and Staging
- Treatment
- Prognosis
Anatomy of the Genitourinary (GU) System

Penis
- Made up of three cylindrical bodies (see Figure 26-1):
  - Two corpora cavernosa covered by tunica albuginea, comprising the erectile tissue of the penile shaft.
  - One corpus spongiosum, which surrounds the urethra and expands to form the glans.
  - Buck’s fascia surrounds the corpora cavernosa and corpus spongiosum structures.
- Blood supply: Internal pudendal artery. Dorsal veins drain into the pudendal plexus, which in turn drains into pudendal vein.
- Lymphatic drainage: Deep and superficial inguinal nodes.

Scrotum
- Made up of smooth muscle, elastic tissue layers of Darto’s fascia, which is a continuation of Scarpa’s fascia from the abdominal wall.
- Blood supply: Femoral and internal pudendal artery.
- Lymphatic drainage: Inguinal and femoral nodes.

Testes
- Lie in the scrotum, usually vertically.
- Encased in tunica albuginea except posteriolaterally, where it is attached to the epididymis. This entire unit is then wrapped in the tunica vaginalis, which attaches to the scrotum posterior-inferiorly via the gubernaculum.
- Vestigial structures: The appendix testis is located in the superior aspect of the testicle, and is of Müllerian duct origin. The appendix epididymis is located on the epididymis, and is of Wolffian duct origin.

EXAM TIP
Cryptorchidism increases the risk for cancer. Approximately 10% of testicular tumors (usually seminoma) arise from undescended testes.

EXAM TIP
Bell-clapper deformity: Insufficient attachment of the testicles to the tunica vaginalis, allowing for a pathologically large degree of rotational freedom. This increases risk of torsion.

Figure 26-1. Cross-section of the penis. (Reproduced, with permission, from Le T, Krause K. First Aid for the Basic Sciences: Organ Systems. New York: McGraw-Hill; 2009: 753.)
Blood supply to testis: Gonadal artery, deferential artery, and cremasteric vessels. All three travel in the spermatic cord!

Venous return: Pampiniform plexus, which in turn drains into the gonadal vein and, to a lesser extent, the external pudendal, vasal, and cremasteric veins.

Lymphatic drainage: External, common iliac, and periaortic nodes.

**Epididymis**
- Tubular structure, originating from the Wolffian duct.
- Four to 5 m in length compressed to an area of about 5 cm.
- Site of sperm maturation and motility.

**Vas deferens**
- Originates from the Wolffian duct.
- A muscular tube that originates at the epididymis, exiting the scrotum laterally. It then courses into the inguinal canal via the external ring and exits the canal at the internal ring. Here, the vas deferens diverges from the spermatic cord, crossing medially behind the bladder and over the ureters to form the ampulla of vas deferens. It then joins the seminal vesicles to form the paired ejaculatory ducts in the prostatic urethra.
- Spermatic cord contains vas deferens, testicular vessels, cremasteric muscle fibers, and spermatic fasciae.

**Prostate**
- Originates from the urogenital sinus and matures via dihydrotestosterone stimulation.
- Secretory gland that contributes to seminal fluid, along with seminal vesicles, Cowper’s glands, and epididymis.
- Posterior surface easily palpable on digital rectal exam (DRE).

**Kidneys**
- Kidneys, like all urologic organs, are retroperitoneal structures.
- Lie obliquely on psoas muscle and quadratus lumborum.
- Renal hilum consists of (from anterior to posterior) vein, artery, and ureter/renal pelvis. Occasionally, there is an accessory artery elsewhere. On the left side, the gonadal vein inserts perpendicularly into the renal vein. However, on the right side, the gonadal vein inserts obliquely directly into the inferior vena cava (IVC) (Figure 26-2).
- Surrounded by perirenal fat, both the kidneys and adrenal glands lie within Gerota’s fascia.
- The adrenal gland is located superior-medially to the kidney. However, in cases of renal ectopia and agenesis, the adrenal gland remains orthotopic.

**Ureters**
Ureters course along the psoas muscle, crossing the iliacs anteriorly at the bifurcation of the external/internal iliac arteries. The ureters then course medially toward the bladder, inserting posteriorly.

**Bladder/urethra**
- Bladder consists of detrusor muscle, covered by a urothelial layer. Histologically, this is transitional epithelium.
Both the detrusor and the bladder neck (internal sphincter) are smooth muscle with autonomic innervation. The external (striated) urinary sphincter is under voluntary control.

In males, the prostatic urethra is located between the internal and external sphincters (this is a common site of adenomatous growth and urinary obstruction).

### Hematuria

**Definition**

- Presence of blood in the urine.
- Can be gross or microscopic.
- ≥3 red blood cells (RBCs) per high power field of spun urine.

**Causes**

- **Pediatric**: Acute urinary tract infection (UTI), glomerular origin, congenital urinary tract abnormality, stones, trauma.
- **Adult/elderly**: Acute UTI, bladder cancer, urolithiasis, benign prostatic hyperplasia.

**Other causes**

- **Upper tract**: Sickle cell disease, collagen vascular disease, renal disease (glomerulonephritis, vascular abnormalities, pyelonephritis, polycystic kidney, granulomatous disease, interstitial nephritis, neoplasm/renal cell carcinoma [RCC]).
- **Lower tract**: Urethritis, stones, cystitis, prostatitis, epididymitis. Mild hematuria is a normal finding in a patient with recent urologic instrumentation (including catheterization).
Coagulopathy and anticoagulation may unmask (but not cause) hematuria and a proper workup should be considered regardless of the etiology or level of coagulopathy.

Priapism

**Definition:** An erection lasting more than 4 hours, generally not maintained by sexual stimulation.

**Two types:** Diagnosed by history and physical and intracorporeal arterial blood gas (ABG)—see below.

- **High flow:** Not ischemic (pH ~7.4), not painful. May result from arteriovenous (AV) fistula secondary to trauma (usually prior injection/irrigation as treatment for low flow).

- May be erect for days to weeks. Erection not fully turgid and glans generally not engorged. Treated with angioembolization of AV fistula site. Less common than low flow.

- **Low flow:** More common. Painful and entirely erect. Ischemic (pH <~7.2), so risk of long-term erectile dysfunction, corporal fibrosis, or organ loss in severe cases. Common causes: Intracorporeal Caverject (alprostadil) injections, cocaine, trazadone, PDE5 inhibitors (e.g., sildenafil), sickle cell.

- Initial treatment involves local ice application, IM verapamil and Sudafed. If these fail (and they will!), patients require intracorporeal phenylephrine injections with or without saline irrigation of the corporal. (Remember to monitor hemodynamics closely when giving intra-corporeal phenylephrine injections!) All of the above can be used when treating sickle cell priapism, but systemic treatments should be first attempted (i.e., exchange blood transfusion, oxygen administration, aggressive hydration hydroxyurea).

Acute Scrotum

**Differential diagnosis:** Testicular torsion, epididymo-orchitis, testicular tumor, peritesticular tumor (rhabdomyosarcoma, leiomyosarcoma, liposarcoma), torsion of testicular appendage, orchitis, trauma to scrotum, hernia, hydrocele, varicocele.

- Testicular torsion is a surgical emergency and must always be considered in the case of acute scrotal pain or mass.

- The diagnosis of an acute scrotal mass will depend largely on the history and physical examination.

- A hydrocele is a collection of fluid between the testicle and the tunica vaginalis. It is typically painless; however, it may occur in response to an inflammatory testicular process.

**Signs and symptoms**

Important findings to look for in acute scrotal mass:

- **Lateralization of swelling.**
- Erythema of scrotal skin.
- Position of the testicle.
- Localization of testicular mass and pain.
- Presence of hydrocele by transillumination.
- Presence/absence of cremasteric reflex.

**EXAM TIP**

What can cause gross hematuria with a dipstick negative for blood? Think: Anthocyanin dye in beets and berries, pyridium, rifampin, porphyria, some food colorings.

**WARD TIP**

 Whereas adult hydroceles gradually enlarge, pediatric hydroceles grow throughout the day and typically disappear overnight as the fluid flows back through the processus vaginalis into the peritoneal cavity.

**WARD TIP**

Transillumination can easily distinguish a hydrocele from other testicular masses.
- Prehn’s sign—relief of pain by elevation of the testicle; indicative of epididymitis.
- Urethral discharge, as well as inguinal lymphadenopathy.
- Fever.
- Recent history of trauma.
- History of similar pain.
- Acute vs. subacute onset.
- Previous history of urethral discharge, sexually transmitted infections, or unprotected sex.

Testicular Torsion

- Peak incidences during the first year of life and at puberty (when growth rates are the fastest).
- Undescended testis confers a higher risk of ipsilateral torsion.
- Salvage rate is 80–100% if surgical intervention is < 6 hours from the onset of pain.
- Frequently preceded by strenuous activity or athletic event.
- Acute onset.

Pathophysiology

- The testicle twists on its spermatic cord, causing obstruction of venous return leading to swelling. If obstruction persists, venous thrombosis occurs, followed by arterial occlusion. Infarction ensues quickly in the absence of blood supply.
- “Bell-clapper deformity” (see above).

Signs and Symptoms

- Acute onset of severe pain in testicle, lower abdomen, or the inguinal canal.
- Scrotum is elevated, swollen, and tender.
- May be accompanied by nausea or vomiting.
- Classic sign: High-riding testis with a horizontal lie. However, this is often difficult to distinguish because of the degree of swelling in the scrotum.
- Presence of a reactive hydrocele.
- Loss of cremasteric reflex.

Diagnosis

- Since epididymitis is most commonly confused with testicular torsion, several elements may aid in distinguishing the two entities. In torsion:
  - Urinalysis (UA) and complete blood count (CBC) will be unremarkable.
  - Onset is acute and often recurrent.
  - Most will be afebrile.
  - Since the ability to salvage the affected testis is dependent on how quickly the testis becomes detorsed, time is tests! If the diagnosis is thought to be torsion, surgical exploration is indicated.
  - Color Doppler ultrasound (US) or nuclear scintigraphy have become the tests of choice, showing the presence or absence of blood flow to the suspected testis.

WARD TIP

The cremasteric reflex is elicited by stroking the medial aspect of the patient’s thigh, resulting in a contraction of the cremasteric muscle elevating the scrotum and ipsilateral testis.

WARD TIP

Always suspect torsion in a patient with inguinal/abdominal pain and an empty scrotum.

EXAM TIP

Typical scenario: An adolescent presents with acute testicular pain and swelling immediately after a sporting event. He is writhing in pain. On further questioning, he has had similar episodes of this in the past. Think: Intermittent testicular torsion/detorsion.

EXAM TIP

The higher the undescended testicle, the higher the lifelong risk of cancer (irrespective of whether or when the testicle is brought into the scrotum surgically).
Surgical repair is the definitive treatment. If the testis is viable, bilateral orchiopexy is performed. If the tissue is necrotic, the testicle is removed to prevent gangrene. The contralateral testis undergoes orchiopexy as a prophylactic measure.

**Testicular Tumor**

**Definition and Epidemiology**
- The most common malignancy to affect young men.
- There is a peak frequency in early childhood, and a larger peak incidence between 20 and 35 years of age. Uncommon after age 40.
- Occurs in whites more than African-Americans.

**Risk Factors**
- Men with cryptorchid (undescended) testes (intra-abdominal testes with the highest risk). It is important to note that both testicles are at risk.
- Surgical placement of the testis into the scrotum does not decrease malignant risk, but facilitates surveillance.
- Testicular cancer in the contralateral testis.

**Signs and Symptoms**
- Painless enlargement of the testicle.
- Firmness of the testicle.
- Back or abdominal pain secondary to retroperitoneal (intra-aortocaval) lymphadenopathy.
- Weight loss.
- Dyspnea secondary to pulmonary metastasis.
- Gynecomastia secondary to hormonal secretions of the tumor.

**Diagnosis**
- Physical examination.
- Testicular US.
- CT scan/magnetic resonance imaging (MRI) of abdomen and pelvis to assess for metastasis and lymphadenopathy.
- Tumor markers—α-fetoprotein (AFP), human chorionic gonadotropin (hCG), and lactic dehydrogenase (LDH).
- Roughly 10% of seminomas secrete hCG; 90% of seminomas do not have elevated tumor markers.

**Classification and Pathology**
- Classification is based on the cell type from which the tumor is derived—germinal or stromal.
- Germinal cell tumors comprise 95% of all testicular tumors:
  - Seminomas (pure single-cell tumors) comprise 35% of testicular tumors.
  - Nonseminomas (embryonal cell carcinoma 20%, teratoma 5%, choriocarcinoma < 1%).
  - Combination tumors 40%.

**EXAM TIP**

The prognosis of seminomas is excellent due to its exquisite sensitivity to radiation!
- Tumors of gonadal stroma (1–2%):
  - Leydig cell.
  - Sertoli cell.
  - Primitive gonadal structures.
  - Gonadoblastomas (germinal cell + stromal cell).

**Staging Evaluation**

- To determine whether the cancer is localized to the testis, regional lymphatics or widely metastasized.
- Abdominal and pelvic CT scan to determine the presence of adenopathy or visceral involvement, chest x-ray (CXR) ± chest CT.
- There are many staging systems; however, most include three stages:
  - **Stage I**: No clinical or radiographic evidence of tumor presence beyond the confines of the testis. Markers normalize after orchiectomy.
  - **Stage II**: Retroperitoneal adenopathy on CT scan, subdiaphragmatic disease, and failure of markers to normalize after orchiectomy.
  - **Stage III**: Distant metastases or visceral involvement.

**Treatment**

- **Surgical approach**: High radical inguinal orchiectomy.
- Trans-scrotal biopsy of the testis or a trans-scrotal orchiectomy should not be performed if the diagnosis of testicular cancer is suspected because the lymphatic drainage of the testes is different from that of the scrotum; a scrotal incision in the presence of testicular cancer may cause local recurrence, metastasis, and unpredictable lymphatic spread.
- **Early seminoma**: Orchiectomy + retroperitoneal x-ray therapy (XRT).
- **Advanced seminoma**: Orchiectomy and combination chemotherapy followed by restaging.
- **Stage I nonseminoma**: Orchiectomy + retroperitoneal lymph node dissection (RPLND) or surveillance.
- **Stage II nonseminoma**: The optimal management of this group of patients is controversial. RPLND can be curative but have a high relapse rate. If relapse occurs, chemotherapy can be given as adjunctive therapy. Alternatively, chemotherapy can be given prior to RPLND.
- Advanced stage nonseminoma: Orchiectomy + chemotherapy ± tumor reductive surgery.
- The most commonly used chemotherapeutic regimen: BEP (etoposide, bleomycin, cisplatin).

**Urolithiasis**

**Definition and Epidemiology**

- One of the most common diseases of the urinary tract.
- Familial tendency in stone formation.
- Average risk is 1%/year.
- Tendency for recurrence: Thirty-six percent of patients with a first stone will have another stone within one year, and 50% will recur within years.
Etiology

- Most common are calcium oxalate or a mixture of calcium (75%).
- Magnesium-ammonium-phosphate stones (struvite) 15%—seen in UTI with urea-splitting bacteria (Proteus). May cause staghorn calculi.
- Uric acid stones (15%), which are radiolucent (will not show up on x-ray, but are visible on noncontrast CT).
- Less than 1% are cystine stones (secondary to an inborn error of metabolism).
- Indinavir stones are not visible on CT scan or plain films.

Risk Factors

- Poor fluid intake/residence in a hot climate.
- Disease processes that increase free calcium, including bony destruction, multiple myeloma, hyperparathyroidism, osteolytic lesions, and sarcoidosis.
- Prolonged immobilization.
- History of calculus in the past and in family members.
- Drug ingestion (analgesics, alkalis, uricosuric agents, protease inhibitors).
- Prior history of gout/Lesch–Nyhan.
- Underlying gastrointestinal (GI) disease/malabsorption (Crohn’s, ulcerative colitis, peptic ulcer disease [PUD], gastric bypass surgery).
- In a normal individual, dietary calcium intake is not a risk factor.

Signs and Symptoms

- Severe, abrupt onset of colicky pain, which begins in the flank and may radiate toward the groin. In males, the pain may radiate toward the testicle. In females, it may radiate toward the labium majoris.
- Nausea and vomiting. GI upset may be the only symptom in patients with chronic stones and infection, as seen in xanthogranulomatous pyelonephritis (XGP).
- Fever.
- Abdominal distention from ileus.
- Gross or microhematuria.

Diagnostic Tests

- Urinalysis and culture:
  - Some patients will have RBCs in the urine (absence does not rule out stones).
  - White blood cells (WBCs) or bacteria may suggest underlying UTI and should be aggressively treated.
- Radiographic studies:
  - Noncontrast abdominal/pelvic CT (Figure 26-3):
    - Fast, requires no IV contrast.
    - Most useful to diagnose small stones (95% sensitivity), hydronephrosis, hydrourereter, and perinephric stranding.
    - Useful for revealing other abdominal/pelvic pathology.
    - Study of choice!
  - Plain abdominal film (KUB): Only radiopaque stones ~5 mm will be seen (60–70% specific in the diagnosis of a calculus). However, it is a cheap and quick test to do and is useful in combination with other studies.
  - Renal US: Fast, easy, and relatively inexpensive. Primarily useful in patients who should avoid radiation, such as pregnant women.

Exam Tip

Typical scenario: A 40-year-old man presents with sudden onset left-sided flank pain that he rates a 10/10. He is writhing, unable to stay still or find a comfortable position. Think: Renal colic. Check a urine dip for blood and order a noncontrast CT scan.
Analgesia with nonsteroidal anti-inflammatory drugs (parenteral ketorolac) and/or opiates.

IV or PO hydration.

During passage of a stone, there are five sites where the passage is likely to become arrested. These are narrowest points of the urinary system:

- Ureteropelvic junction
- Pelvic brim
- Iliac crossing
- Ureterovesical junction
- Vesicle orifice

If stone impaction occurs and hydronephrosis develops, decompression of the affected kidney may be necessary to preserve kidney function.

For stones unlikely to pass spontaneously:

- Extracorporeal shockwave lithotripsy (ESWL) is effective for stones visible on plain abdominal films (< 2 cm).
- Ureteroscopy with stone extraction for ureteral calculi and small renal calculi.
- Percutaneous nephrolithotomy, which establishes a tract from the skin to the collecting system, is used when stones are too large or too hard for lithotripsy.
- Surgical emergency if accompanied by fever, UTI, and/or acute kidney injury, necessitating stent placement or nephrostomy tube.

**Benign Prostatic Hyperplasia**

**Definition**

- Benign prostatic hyperplasia is the most common benign tumor in men.
- Incidence is directly proportional to age, affecting approximately 90% of men > 80.
The prostate enlarges (within the confines of a tight prostatic capsule) during puberty when it undergoes androgen-mediated growth. It remains stable in size until about the fifth or sixth decade, when its size increases again.

**Pathophysiology**
- Hyperplasia begins in the periurethral area, then progresses to the remainder of the gland; hence, the most common initial symptoms are of urinary outflow obstruction.
- Histologically, the hyperplastic tissue is comprised of glandular epithelium, stroma, and smooth muscle.
- As hyperplasia increases with increasing obstruction, frank urinary retention can occur or may be precipitated by extrinsic etiologies, such as infection, anticholinergic drugs, $\alpha$-agonists, or alcohol.

**Signs and Symptoms**
- Early symptoms include hesitancy in initiating voiding, weak stream, postvoid dribbling, and the sensation of incomplete emptying.
- As the amount of residual urine increases symptoms may include nocturia, overflow incontinence, and urinary frequency and urgency.

**Diagnosis**
- Clinical history.
- DRE—in hyperplasia, the prostate will be smooth, firm, but enlarged.
- Measurement of postvoid residual urine volume and uroflow.

**Management**
- Medical:
  - 5-$\alpha$ reductase inhibitor (finasteride, dutasteride): Blocks conversion of testosterone to dihydrotestosterone, may shrink prostatic hyperplasia up to 20%.
  - $\alpha$-adrenergic antagonists (prazosin, terazosin, tamsulosin) decrease urethral resistance.
- Surgical (it is important to note that these procedures are not cancer surgeries):
  - Transurethral prostatectomy (TURP) for smaller prostates.
  - Open simple prostatectomy for larger prostates.
  - Retrograde ejaculation is a consequence of these surgeries.

**Prostatic Carcinoma**

**Definition**
- The most common malignancy in men in the United States.
- Rare before age 50.

**Risk Factors**
- Increasing age
- African-American
### Classification
- Ninety-five percent are adenocarcinoma. The remaining 5% are squamous, transitional cell, sarcoma, and occasional metastatic tumors.
- Predilection to originate in the peripheral zone and is often multifocal.

### Signs and Symptoms
- Most patients are asymptomatic at the time of diagnosis, secondary to widespread use of PSA screening tests. It is important to note, that patients on 5-a reductase inhibitors have artificially reduced PSA levels (up to 50%).
- In symptomatic patients, common symptoms include obstructive or irritative voiding complaints.
- Metastatic disease to the bone may cause bone pain.
- Symptoms in advanced disease may include ureteral obstruction, spinal cord compression, deep venous thrombosis (DVT), and pulmonary emboli.

### Diagnosis
- On DRE, the prostate is usually hard, nodular, fixed, and irregular.
- Prostate-specific antigen (PSA) is the most sensitive test for early detection of prostatic cancer. Following diagnosis, PSA is used to follow progression of disease and response to treatment. However, the PSA is not a specific test. PSA can be elevated in prostatic hyperplasia or prostatitis. Although debate is ongoing, a PSA of 4 in a male over the age of 50 is generally agreed upon as indication for transrectal ultrasonography (TRUS) biopsy.
- **Imaging studies:**
  - TRUS, used for image-guided biopsies: Carcinomas appear as hypoechoic densities in the peripheral zone, has very low sensitivity and specificity.
  - MRI or CT may also be helpful in identifying metastasis and lymph node involvement.
- **Biopsy:** Essential in establishing the diagnosis of prostate cancer.

### Staging
- Metastatic spread occurs via:
  - Direct extension (into the seminal vesicles and/or bladder floor).
  - Lymphatic spread to obturator, internal iliac, common iliac, presacral, and periaortic nodes.
  - Hematogenous spread occurs to bone more frequently than viscera.
- Standard staging scheme used is the **tumor-node-metastases (TNM) system.**
- Gleason grading system is based on a histologic evaluation of prostate tissue samples. The Gleason score is the sum of the two most common cell patterns seen in the tissue sample. The patterns can range from 1 (well differentiated) to 5 (poorly differentiated, highly malignant). Overall scores range from 2 to 10. A grade of 2 has the best prognosis, while a grade of 10 represents poorly differentiated tissue and confers the worst prognosis.
- Bony metastasis can contain both osteoblastic components. Axial skeleton is most commonly affected. Skeletal survey has a low sensitivity for detecting bony metastasis. Radionuclide bone scan has a much higher sensitivity and is also useful in monitoring progression and response to therapy.
- CT of abdomen and pelvis is used to assess lymph node involvement.
Treatments vary in aggressiveness depending on patient age, health status, and stage/grade of disease. Treatments may include any/all of the following:

- Watchful waiting/active surveillance.
- Androgen deprivation therapy: It has been established that prostatic carcinomas are hormonally dependent. Some degree of control can be obtained by hormonal therapy. Androgen deprivation can be achieved via:
  - Surgical castration (bilateral orchiectomy results in 90% reduction in testosterone).
  - Gonadotropin-releasing hormone (GnRH) agonist (leuprolide) therapy.
  - Androgen receptor antagonists (flutamide, bicalutamide) and/or androgen synthesis inhibitors (ketoconazole).
  - Estrogen administration.
- Radiation: Can be via XRT or brachytherapy (radioactive seed placement).
- Radical prostatectomy: May be retropubic (transabdominal), transperineal, laparoscopic, or robotic.
- Chemotherapy is not very effective, but may be used as a last resort in cases of very advanced, hormone-refractory disease.
- Combinations of all of the above are commonly used.

Renal Cell Carcinoma (RCC)

etiology

- Eighty-five percent of all primary malignant renal neoplasms.
- Peak incidence between 50 and 60 years.
- Environmental factors: Cigarette smoking; exposure to cadmium, asbestos, and solvents.
- Hereditary link: Genetic defect linked to translocations between chromosomes 3 and 8.

signs and symptoms

- Classic triad of gross hematuria, flank pain, and palpable abdominal mass (seen in 10–15% of cases).
- Most commonly an incidental finding.
- Most often diagnosed via its systemic symptoms: Fatigability, weight loss and cachexia, intermittent fever, and anemia.
- Other symptoms may relate to the production of hormones and hormone-like substances (paraneoplastic syndromes):
  - Hypercalcemia (parathyroid hormone)
  - Galactorrhea (prolactin)
  - Cushing’s syndrome (glucocorticoid)

diagnosis

- It is most important to differentiate cystic from solid lesions.
- Ultrasound has improved the ability to differentiate a solid from a cystic lesion.
- CT is the method of choice for diagnosis and staging of RCC.

WARD TIP

Complications of radiation therapy for prostate cancer:
- Cystitis
- Acute proctitis (diarrhea)
- Urethritis (can lead to strictures)
- Rectal strictures and fistula
- Impotence
- Secondary malignancy
**Stage I:** Tumor confined within the kidney parenchyma.

**Stage II:** Invasion through the kidney capsule, involves perinephric fat but confined within Gerota’s fascia.

**Stage III:** Involvement of regional lymph nodes, ipsilateral renal vein, or vena cava.

**Stage IV:** Distant metastasis, adjacent organ involvement.

**Treatment**

- Radical nephrectomy is the treatment of choice if there is no evidence of metastasis. Partial nephrectomy (complete resection of mass) is oncologically equal to radical nephrectomy.
- Radiation therapy can be used in the palliation of patients with metastatic RCC.
- There are no standard chemotherapeutic regimens or hormonal therapy for metastatic disease, and these treatments have been employed with limited success.
- Chemotherapeutic agents (sorafenib).

**Prognosis**

Five-year survival rates:
- Stage I: 91–100%
- Stage II: 74–96%
- Stage III: 59–70%
- Stage IV: 16–32%

**Urothelial Cancer**

**Definition and Epidemiology**

The lining of the urinary system from the renal pelvis to the urethra is made up of transitional cells. This entire lining is subject to carcinomatous changes. However, the bladder is involved most frequently and will therefore be discussed most extensively.

**Transitional Cell Carcinoma of the Collecting System and Ureters**

- Collecting system and ureteral lesions have a high (~60%) likelihood of spreading to the bladder, as this disease is a field defect. Rate of contralateral spread is ~5%, so patients require lifelong surveillance.
- Nephroureterectomy is treatment of choice.
- As with bladder cancer (see below), advanced disease has poor prognosis.
- Presentation and risk factors are similar to bladder (see below).

**Bladder Cancer**

- Second most common cancer of the GU tract.
- Men are affected three times more than women.
Peak incidence occurs between the ages of 60 and 70.

Ninety-eight percent of bladder cancers are epithelial; in the United States, most are transitional cell carcinomas.

Nontransitional cell carcinomas, such as squamous cell and adenocarcinomas have a worse prognosis compared to transitional cell carcinoma.

Squamous cell carcinoma (SCC) tends to arise secondary to chronic irritation and inflammation (indwelling catheter, recurrent UTI, schistosomiasis).

Adenocarcinoma tends to be metastatic or direct spread from adjacent organ (large bowel, uterus, uracus).

Transitional cell carcinoma (by far the most common!) may result from cigarette smoking (twofold increased risk), dye, or chemical exposures.

**Signs and Symptoms**

- Gross and microscopic hematuria are the most common presenting symptoms (80–95%).
- Other symptoms include dysuria, urinary frequency, urgency, and ureteral obstruction.

**Diagnosis and Staging**

- Urine cytology for exfoliated malignant cells (poor sensitivity, high specificity).
- Intravenous pyelography (IVP)—ureteral obstruction with hydronephrosis or filling defect.
- Cystoscopy with tumor biopsy.
- Additional staging may be obtained via CT of abdomen and pelvis and endoscopic resection of a bladder neoplasm.

**Superficial:**
- Tis: Carcinoma in situ, mucosal involvement.
- Ta: No invasion.

**Invasive:**
- T1: Submucosal involvement.
- T2: Involvement of bladder muscularis.
- T3: Involvement of perivesical fat.
- T4: Involvement of adjacent viscera.

**Metastatic:** Uses TNM system.

**Treatment**

- Transurethral resection of bladder tumor (TURBT): Endoscopic resection is necessary for initial evaluation of the lesion to assess for depth of invasion. Superficial localized tumors can be treated with TURBT and surveillance (Tis–T1).
- Intravesical chemotherapy using BCG (bacillus Calmette–Guérin) or mitomycin has been shown to have preventative effects after TURBT for lesions up to and including T1.
- Radical cystectomy is the treatment of choice in patients with T2 and greater disease who can tolerate a surgery of this magnitude. Resection of the iliac lymph nodes during this surgery has shown to be both diagnostic and therapeutic.
- The oncologic indications for partial cystectomy are the same as for radical cystectomy. However, the former is generally performed when the tumor(s) are in a location in the bladder, and of a sufficiently small size, amenable to a bladder-sparing procedure. Lymph node dissection is also performed with this surgery.
- Neoadjuvant chemotherapy before (partial) cystectomy improves survival.
- XRT is a reasonable option for muscle invasive (T2–T3) lesions for poor surgical candidates. There is less long-term data on this modality.
- The prognosis of metastatic urothelial cancer is poor. However, chemotherapeutic agents have been shown to prolong survival. Commonly used agents are cisplatin, methotrexate, gemcitabine, doxorubicin, cyclophosphamide, and vinblastine.

Prognosis

The survival rate of patients with metastatic disease is generally < 2 years.
## Fractures

- **Open Fracture**
- **Pathologic Fracture**
- **Stress Or Fatigue Fracture**
- **Comminuted Fracture**
- **Green Stick Fracture**

## Fat Embolism Syndrome
- **Definition**
- **Onset**
- **Pathophysiology**
- **Signs and Symptoms**
- **Diagnosis**
- **Treatment**

## Shoulder Dislocations
- **Complications**
- **Complications Specific to Type Of Dislocation**
- **Radiograph**
- **Treatment**

## Compartment Syndrome
- **Definition**
- **Cases**
- **Signs and Symptoms**

## Osteomyelitis
- **Acute**
- **Chronic**

## Brodie's Abscess

## Septic Bursitis

## Low Back Pain
- **Epidemiology**
- **History**
- **Physical Examination**
- **Diagnosis**
- **Treatment**

## Bone Tumors
- **Radiography**
- **Benign Bone Tumors**
- **Malignant Bone Tumors**
- **Metastatic Bone Tumors**

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**High-Yield Facts in Orthopedics**

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Fractures

- Categorized by anatomical location (proximal, middle, or distal third of shaft), direction of fracture line (transverse, oblique, spiral), and whether it's simple or comminuted.
- Patient presents with loss of function, severe pain, tenderness, swelling, abnormal motion, and often deformity.

Open Fracture

- Fracture communicates with the external environment due to a breach of the overlying soft tissue.
- **True orthopedic emergency**: Almost always results in bacterial contamination of soft tissues and bone.
- Prognosis dependent on extent of soft tissue injury and by type/level of bacterial contamination.
- **Treatment plan**: Prevent infection with early antibiotics and tetanus, debride affected soft tissues and bone, restore available soft tissue, achieve bony union, and institute early joint motion and muscle rehabilitation.

Pathologic Fracture

- Occurs due to minimal trauma on a bone weakened by preexisting disease.
- **Predisposing conditions**: Primary or metastatic carcinoma, osteoporosis, bone cysts, enchondroma, giant cell tumors, osteomalacia, osteogenesis imperfecta, scurvy, rickets, and Paget’s disease.
- Orthopedic surgeon must not only treat the broken bone but should also diagnose and treat the underlying condition.

Stress or Fatigue Fracture

- An incomplete or complete fracture resulting from repetitive application of minor trauma.
- Most stress fractures occur in the lower extremities and commonly affect individuals involved in sports and military recruits (“march fracture”).
- Pathophysiology of stress fractures is unclear but possibly due to inability of the fatigued muscle to protect bone from strain.
- If the patient is seen within first 2 weeks of onset of symptoms, the plain radiograph is likely to be normal.
- Patients usually complain of pain only with activity.
- **Treatment**: Decrease physical activity or period of limited weight bearing.

Comminuted Fracture

Fracture in which the bone is divided into more than two fragments by fracture lines.

Salter–Harris Fracture

- Fracture involving the physis (growth plate). Occurs irregularly through the weak zone of hypertrophic cartilage (Figure 27-1).
- **Type I**: Fracture is transverse and does not travel vertically across the germinal cell layer. Prognosis for normal healing is good.
- **Type II**: Fracture extends from physis into metaphysis of bone.
- **Type III:** Fracture extends from physis to epiphysis.
- **Type IV:** Fracture traverses the growth plate in a vertical fashion often causing angular deformity from continued growth. Surgical intervention may be necessary.
- **Type V:** Crush injury to the physis such that metaphysis and epiphysis are impacted on one another. No visible fracture line. Poor prognosis with high risk of growth arrest.

**Greenstick Fracture**

- An incomplete and angulated fracture of the long bones. A transverse crack that hangs on to its connection.
- Very common in children, rarely seen in adults.
- Since kids have “softer,” less brittle bone and thicker (leathery) periosteal membrane, they get incomplete fractures with unique patterns.

**Fat Embolism Syndrome**

**Definition**

An acute respiratory distress syndrome caused by release of fat droplets from the marrow as may occur following a long bone fracture.
Common Causes

- Long bone fracture.
- Use of intramedullary nails for treatment of fractures.
- Burns.
- Severe infection.
- Inhalation anesthesia.
- Metabolic disorders.
- Cardiopulmonary bypass.
- Decompression sickness.
- Others: Hemoglobinopathy, collagen disease, diabetes, renal homotransplantations.

Pathophysiology

- Microdroplets of fat are released into the circulation at the site of fracture, occluding pulmonary circulation causing ischemic and hemorrhagic changes.
- Another theory: Release of free fatty acids from the marrow has toxic effects in all tissues, especially the lung.

Symptoms and Signs

- Symptoms may occur immediately or 2–3 days after trauma.
- Shortness of breath with respiratory rate above 30.
- Confusion, restlessness, disorientation, stupor, or coma.
- Fleeting petechial rash on chest, axilla, neck, and conjunctiva.
- Fever, tachycardia.

Diagnosis

- **Hallmark finding:** Arterial hypoxemia. Arterial \( \text{PO}_2 \) < 60 mmHg is suggestive.
- Cryostat—frozen section of clotted blood reveals presence of fat.
- Absence of fat globules in urine makes diagnosis unlikely; however, their presence is not specific for fat embolism.

Treatment

- Administer oxygen to decrease hypoxemia and monitor \( \text{PO}_2 \) to maintain it over 90 mmHg.
- In severe hypoxemia: Mechanical ventilatory support.
- Use of ethanol, heparin, hypertonic glucose, or steroids has been suggested but their effectiveness is questionable.
- Prevent fat embolism syndrome by careful, early appropriate stabilization of fractures and effective treatment of shock.

Prognosis

Mortality from fat embolism thought to be as high as 50% following multiple fractures.

Shoulder Dislocations

See Table 27-1.
### Table 27-1: Shoulder Dislocations

<table>
<thead>
<tr>
<th></th>
<th><strong>Anterior Dislocation</strong></th>
<th><strong>Posterior Dislocation</strong></th>
<th><strong>Inferior Dislocation</strong> (Luxatio Erecta)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Features</strong></td>
<td>High risk of recurrence 70% Occur in patients younger than 30 years of age</td>
<td>Diagnosis missed in 60% of cases Often precipitated by a convolution, seizure, electrical shock, and falls</td>
<td>&lt; 1% of all shoulder dislocations</td>
</tr>
<tr>
<td><strong>Types</strong></td>
<td>Subcoracoid (most common), subclavicular, subglenoid</td>
<td>Subacromial (most common), subglenoid, subspinous</td>
<td></td>
</tr>
<tr>
<td><strong>Mechanism of injury</strong></td>
<td>Abduction and external rotation of the arm causes strain on anterior capsule and glenohumeral ligaments</td>
<td>Internal rotation and adduction (when one falls on an arm that is forwardly flexed and internally rotated)</td>
<td>Hyperabduction always results in detachment of rotator cuff</td>
</tr>
</tbody>
</table>
| **Signs and symptoms**  | - Arms held to the side  
- Patient resists medial rotation and adduction  
- Prominent acromion  
- Loss of normal rounded shoulder contour | - Patient holds arm medially rotated and to the side  
- Abduction limited  
- External rotation limited  
- Prominence of the coracoid process and posterior part of shoulder  
- Flattening of anterior aspect of shoulder | - Patient in severe pain  
- Arm held in 180° elevation  
- Arm appears shorter compared to opposite side  
- Humeral head often felt along the lateral chest wall |

### Complications Common to All Dislocations

Axillary artery injury (more common with luxatio erecta), venous injury, injury to nerves of brachial plexus (most common being axillary nerve):

- Palpate radial pulse to check axillary artery.
- Check motor component of axillary nerve by assessing strength of the deltoid muscle.
- Check sensory component of axillary nerve by assessing sensation over the lateral part of upper arm.
- Do a neurologic exam to evaluate all brachial nerve lesions.

### Complications Specific to Type of Dislocation

<table>
<thead>
<tr>
<th></th>
<th><strong>Anterior Dislocation</strong></th>
<th><strong>Posterior Dislocation</strong></th>
<th><strong>Inferior Dislocation</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>- Rotator cuff tear</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Glenoid labral lesions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Coracoid fractures</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| - Greater tuberosity fractures  
(seems to decrease recurrence) |                          |                           |                           |
| - Hill–Sachs deformity  
(compression fracture of the posterolateral humeral head) |                          |                           |                           |
| - Fractures of the lesser tuberosity |                          |                           |                           |
| - Fractures of posterior glenoid rim and proximal humerus humeral head |                          |                           |                           |
| - Rotator cuff tear     |                          |                           |                           |
| - Fractures of greater tuberosity |                          |                           |                           |
### Compartment Syndrome

**Definition**

A condition in which increased pressure within a limited space compromises the circulation and function of tissues within that closed space.

**Causes**

- Fractures.
- Soft tissue crush injuries.
- Vascular injuries.
- Drug overdose with prolonged limb compression.
- Burn injuries.
- Trauma.
- Muscle hypertrophy and nephrotic syndrome.

**Signs and Symptoms**

- Clinical presentation often indefinite and confusing.
- Hallmark finding: PAIN! Pain in a conscious and fully oriented person that is out of proportion to injury or findings. Must always consider...
compartment syndrome if pain not controlled with appropriate treatment. Can also see paresthesia at the first web space (i.e., between great and second toes) but this is often later and concerning for permanent injury.

- **Pain**: Deep, unremitting, and poorly localized. Pain increases with passive stretching of involved muscle.
- **Pallor**: Not necessary for diagnosis, may not be present.
- **Paresthesias**: Of cutaneous distribution supplied by the compressed nerve is an early sign.
- **Paralysis**: Occurs after ischemia is well established.
- **Pulselessness**: Shown to occur late at times. Pulse may be present.
- **Compartment tense on palpation.**

### Diagnosis

This is a clinical diagnosis. Diagnosis made with recognition of high-risk patients and uncontrollable pain out of proportion with injury. Be aware of distracting injuries. Measure pressure within compartment with commercially available monitors.

- Pressure < 30 mmHg usually will not produce a compartment syndrome.
- Pressure > 30 mmHg is an indication for fasciotomy.

### Treatment

Complete fasciotomy: Goal is to decompress all tight compartments and salvage a viable extremity.

---

**Osteomyelitis**

### Acute

**Pathophysiology**

- Bacteria lodge in end artery of metaphysis and multiply.
- Local increase in serum and white blood cells (WBCs).
- Decrease in blood flow and pressure necrosis.
- Pus moves to Haversian and medullary canals.
- Goes beneath the periosteum.

**Causes**

- Route of infection is mainly hematogenous, rarely trauma.
- Most cases of acute hematogenous osteomyelitis caused by Staphylococcus aureus.

**Signs and Symptoms**

- History of infection (e.g., skin or throat) or trauma.
- Significant pain in the affected area, anorexia, fever, irritability, nausea, malaise, rapid pulse.
- Limited joint motion, tenderness, swelling of soft tissue, inability to bear weight and guarding apparent on physical exam.

---

**WARD TIP**

Most common site for acute osteomyelitis is the metaphyseal end of a single long bone (especially around the knee).
**Diagnosis**
- Elevated WBCs, erythrocyte sedimentation rate (ESR), and C-reactive protein; ± anemia.
- Deep circumferential soft-tissue swelling with obliteration of muscular planes.

**Differential Diagnosis**
- Septic arthritis: Swelling and tenderness directly on the joint with intense pain on joint movement, high WBC, and positive culture.
- Rheumatic fever: More insidious onset, less local and constitutional symptoms.
- Ewing's sarcoma: Early symptoms are more insidious and less intense and can present with bone destruction.

**Treatment**
- **Medical:** Infection must be diagnosed early. Intravenous antibiotics (usually oxacillin or cloxacillin 8–16 g adult) started soon after obtaining specimen for culture. Monitor temperature, swelling, pain, WBC, and joint mobility.
- **Surgical:** Open drainage of abscess if antibiotic fail or signs of abscess appear. After surgical drainage, wound is left open to heal by secondary intention.

**Chronic**

**Epidemiology**
Often seen in lower extremities of a diabetic patient.

**Pathophysiology**
- Untreated acute osteomyelitis results in a cavity walled off by an involucrum containing granulation tissue, sequestrum, and bacteria.
- Drainage of pus into surrounding soft tissue and skin via sinus tracts.
- Persistent drainage can lead to carcinoma.
- Bone fragments and exudates unreachable by antibiotics.
- Result is severely deformed bone and possibility of pathologic fracture.

**Causes**
- Usually an end result of untreated or treatment failed acute osteomyelitis. Occasionally due to trauma or surgery.
- Cause is usually polymicrobial—difficult to eradicate.

**Signs and Symptoms**
- Characterized by persistent drainage following an episode of acute osteomyelitis or onset of inflammation and cellulitis following an open fracture.
- Fever (not always present), pain, mild systemic symptoms, tenderness.
- Easy to diagnose when drainage is present and x-ray shows bone destruction and deformity. In cases with absence of drainage, radionuclide imaging studies can be helpful.

**Diagnosis**
Radiographic findings:
- Areas of radiolucency within an irregular sclerotic bone.
- Irregular areas of destruction present. Often, periosteal thickening can be seen.
**Diagnosis**
- Acute suppurative arthritis.
- Rheumatic fever: Examine synovial fluid.
- Cellulitis: Absence of soft tissue swelling on radiographs.

**Treatment**
- Varies from open drainage of abscess or sequestrectomy to amputation.
- Most effective: Extensive debridement of all necrotic and granulation tissue along with reconstruction of bone and soft tissue defects with concomitant antibiotics.
- Excellent adjunct: Temporary placement of absorbable or nonabsorbable antibiotic containing beads in the wound for local administration of antibiotics.

**Complications**
- Soft tissue abscess.
- Septic arthritis due to extension to adjacent joint.
- Metastatic infections to other areas.
- Pathologic fractures.
- If significant spinal involvement, paraplegia.

**Brodie’s Abscess**
- Subacute pyogenic osteomyelitis in the metaphysis.
- X-ray finding: Lucent lesions surrounded by sclerotic bone.
- Usually caused by *S. aureus* and Staphylococcus albus.

**Septic Bursitis**
- Infection of the superficial bursa commonly affecting the bunion, olecranon, and prepatellar bursa.
- Most common offending organism is *S. aureus*.
- Clinically presents with painful bursal swelling, often along with intense cellulitis. Systemic signs of sepsis can be present along with regional lymphadenopathy.
- Treatment: Aspirate bursa for culture and sensitivity. Give broad-spectrum antibiotic. Take care not to aspirate the joint since passing the needle through the area of cellulitis might spread it to the joint! Can require surgical debridement or bursectomy.

**Low Back Pain**
- Four out of five people suffer from low back pain sometime in life.
- Incidence 15–20%, males > females.
- Most patients with low back pain have no systemic disorder.
- Back pain can be symptom of a systemic illness such as primary or metastatic neoplasm, infectious disease, aortic aneurysm, renal disease or stones, or an inflammatory disorder, especially if very intense or acute onset without antecedent trauma.

**Low Back Pain**
is the leading cause of an orthopedic visit.
History

- Very important, although often the only presenting complaint is pain that is poorly localized.
- Character of pain needs to be described: What is the pain like? Does it radiate? When does it occur? How does it interfere with sitting, standing, walking? What factors make the pain better or worse? How many episodes have you had? Any other symptoms along with back pain?
- Give patient a diagram and ask patient to mark areas of pain.
- History of pain development and how it affects everyday life.
- History of weight loss, malaise, fever, gastrointestinal (GI) or genitourinary (GU) illnesses.
- Psychological assessment in patients with chronic pain.

Physical Examination

- Straight leg-raising test: Positive (reproduces the complaining symptom) in nerve root irritation.
- Check for reflexes and motor and sensory deficits.
- Check presence of nonorganic signs (Waddell's signs) when patient responds to axial loading, local touch, and simulated rotation—signifies faking or exaggerating symptoms for secondary gain.
- Check spine for range of motion.
- Bowel and bladder symptoms are suggestive of cauda equina syndrome (surgical emergency).
- Leg and buttock pain are suggestive of herniated disk.

Diagnosis

- X-rays of lumbar spine especially if patient is > 50 years of age and has history of other medical illnesses or trauma.
- Magnetic resonance imaging (MRI) of the lumbar spine if x-rays are negative: Great for assessing neural tissue.
- Computed tomography (CT) scan if MRI not helpful.
- Technetium bone scan and gallium scan can be done if an infection of the spine is suspected.

Treatment

- Rule out a serious pathologic condition.
- Goal is early return to normal activities.
- Patients with acute low back pain should avoid sitting or lifting and use mild analgesics and anti-inflammatory drugs.
- Physical and occupational therapy programs prove to be helpful.
- Antidepressants often help those with pain persistent for 3 months.
- Other treatment options: Transcutaneous electrical nerve stimulation (TENS), traction, manipulation with radicular signs, biofeedback, acupuncture, trigger point injections, and muscle relaxants.

Bone Tumors

- Occur due to uncontrolled cellular proliferation of a single clone of cells whose regulatory mechanisms are defective.
- Benign tumors are 200 times more likely to occur than malignant ones.
A careful history and physical is very crucial and will reveal the duration of the mass, onset of pain, other associated symptoms, and the chronological sequence of these symptoms.

A thorough physical exam consists of evaluation of patient’s general health status. The mass should be noted for size, location, consistency, mobility, tenderness, local temperature, and change with position. Note any muscular atrophy.

**Radiography**

- Never diagnose a bone tumor without an x-ray.
- Bone reacts to a benign or malignant tumor by bone production or destruction.
- An x-ray appearance can show bone production, destruction, or both.
- Three patterns of x-ray appearance:
  - **Permeative**: Implies a rapidly spreading intramedullary tumor; tumor replaces marrow and fat.
  - **Moth eaten**: Implies a poorly circumscribed, slow-growing malignant tumor.
  - **Geographic**: Implies a well-circumscribed slow-growing tumor, therefore bone has time to react and results in sclerotic margins.

**Benign Bone Tumors**

Patient is usually asymptomatic, and the x-ray shows a well-defined lesion with sclerotic margins.

**Osteoid Osteoma**

**Signs and Symptoms**

- Most common osteoid-forming benign tumor (10%).
- Male-to-female ratio is 2:1 to 3:1, three fourths of cases between ages 5 and 25.
- Most common sites: Diaphysis of long tubular bones, especially the proximal femur.
- Local tenderness and dull aching pain that is localizable, tends to be more severe at night and relieved by nonsteroidal anti-inflammatory drugs (NSAIDs).
- Pain can radiate and mimic other diseases (sciatica if present in vertebra).

**Radiography**

Localized area of bone sclerosis with a central radiolucent nidus. Little sclerosis seen if it is present in cancellous bone.

**Histology**

Usually measures < 1 cm. Circumscribed, highly vascular nidus made of fibroconnective tissue and woven bone.

**Treatment and Prognosis**

Mostly treated symptomatically with aspirin or NSAIDs. If this fails, surgical intervention to remove the nidus or radio frequency ablation (RFA). Prognosis is excellent.

**Osteoblastoma**

**Epidemiology**

- One percent of benign bone tumors. Larger than osteoid osteomas.
- Male > female, most patients between ages 10 and 35 years.
- Most common sites: Mostly axial skeleton, less common in jaw, hands, and feet. One third to one half of cases are in the vertebral column.
Pain is the major complaint. Tenderness and swelling may be present over the lesion.

**Radiography**
Nonspecific x-ray findings, which may be interpreted as osteoid osteomas, aneurysmal bone cysts, or malignant tumors.

**Histology**
Histologically similar to osteoid osteoma.

**Treatment and Prognosis**
- Vigorous curettage of the lesion. Prognosis is generally good except occasionally can become locally aggressive or recur locally if they are not adequately excised.
- Have potential to undergo malignant transformation and metastasize.

**Osteochondroma**
- An outgrowth of bone capped by cartilage.
- Most common benign tumor of the bone (45%) with most patients in their first two decades of life.
- Usually solitary. Originates in childhood from growing epiphyseal cartilage plate. Mostly stage I lesions.
- Most common sites: Metaphysis of long bones of extremities; rarely in fat bones, vertebrae, or clavicle.

**Signs and Symptoms**
May be asymptomatic; patient may complain of pain, mass, or impingement syndromes.

**Radiography**
Shows a mushroom-like bony prominence.

**Histology**
Trabecular, cancellous bone continuous with the marrow cavity and covered by hyaline cartilage cap.

**Treatment and Prognosis**
- Surgical excision if the patient complains of pain or if it enlarges after puberty.
- Rarely undergo malignant transformation to chondrosarcoma (<1%).

**Enchondroma**
- Neoplasm consisting of mature hyaline cartilage (chondroma).
- A centrally located chondroma. Can be single or multiple.
- Ten percent of benign tumors. Peak incidence in ages 20–50 years.
- Most common sites: Tubular bones of hands and feet.
- Chondromas can arise close to cortex or periosteum (ecchondroma) or in relation with synovium, tendons, or joints (synovial chondroma).
- Stage I or stage II lesions.

**Signs and Symptoms**
Asymptomatic until a pathologic fracture brings attention to it.

WARD TIP
Multiple hereditary osteochondroma is an autosomal dominant disorder in which multiple bones have osteochondromas (1% risk of malignant transformation).

WARD TIP

WARD TIP
Mu lt iple  h e re d it a ry o st e o ch o n d ro m a is an autosomal dominant disorder in which multiple bones have osteochondromas (1% risk of malignant transformation).
Geographic lysis in a well-circumscribed area with spotty calcifications.

**Histology**
Consists of hyaline cartilage often with active nuclei. Interpretation depends on size, location, and growth.

**Treatment and Prognosis**
No treatment if patient is asymptomatic. If pathologic fracture occurs, then allow fracture to heal. Perform a simple excision and bone grafting procedure.

**Giant cell tumor**
- Thought to arise from mesenchymal stromal cells supporting the bone marrow.
- Five to 10% of benign bone tumors. Peak incidence in 30s.
- Female-to-male ratio is 3:2.
- Most common sites: Around the knee (distal femur, proximal tibia), distal radius, and sacrum.
- Mostly stage II or III lesions.

**Signs and Symptoms**
- Pain, swelling, and local tenderness; often presents with arthritis or joint effusions due to proximity to the joint.
- May also present with a pathologic fracture.

**Radiography**
- A radiolucent lesion occupying the epiphysis and extending into the metaphysis; asymmetrical with bone destruction.
- Occasional “soap bubble” appearance due to a thin subperiosteal bone shell.

**Histology**
Abundant mononuclear stromal cells interspersed with a lot of giant cells with numerous nuclei.

**Treatment and Prognosis**
- Curettage and bone grafting (recurrence rate > 50%).
- Aggressive curettage with adjuvant phenol, hydrogen peroxide, or liquid nitrogen (recurrence rate 10–25%).
- Important to obtain CXR or CT every 6 months for 2–3 years for monitoring.
- Often recurs after incomplete removal.

**Malignant Tumors**
Metastatic tumors are much more common than primary tumors.

**Osteosarcoma**
- Tumor made of a malignant spindle cell stroma producing osteoid.
- Many subtypes of osteoid forming sarcomas.
- Peak incidence in ages between 10 and 30 years, male > female.
- Most common sites: Around the knee (distal femur, proximal tibia), proximal humerus, rarely mandible.

**WARD TIP**
Factors that predispose to malignancy:
- Size (> 4.5 cm)
- Location (long and axial bones)
- Growth (active and painful)

**WARD TIP**
Osteosarcoma can occur secondary to Paget’s disease.
Signs and Symptoms
- Pain associated with a tender mass.
- Dilated veins may be visible on the skin over the mass.
- Constitutional symptoms may be present.

Radiography
- X-ray shows a poorly defined lesion in the metaphysis with areas of bone destruction and formation.
- Codman’s triangle: Due to new bone formation under the corners of the raised periosteum.
- Sun-burst appearance: Occurs when the bone spicules are formed perpendicular to the surface of the bone.

Histology
- Spindle-shaped tumors cells with odd, hyperchromatic nuclei showing a high mitotic rate. Giant cells may be present.
- High-dose methotrexate, doxorubicin, cisplatin, and ifosfamide along with surgical intervention.
- Tumors hematogenously metastasize to the lung.
- Surgery plus chemotherapy: Five-year survival is about 60%.
- Better prognosis if the tumor is in a small bone.

Chondrosarcoma
- Low-grade malignant tumor that derives from cartilage cells.
- Seven to 12% of primary bone tumors; male > female.
- Peak incidence between ages 30 and 60 years.
- Most common sites: Pelvis, femur, flat bones, proximal humerus, scapula, upper tibia, and fibula.

Signs and Symptoms
Pain and swelling over months or years.

Radiography
- Central chondrosarcomas show well-defined radiolucent areas with small, irregular calcifications to ill-defined areas breaking through the cortex.
- Peripheral chondrosarcomas look like large, lobulated masses hanging from the surface of a long bone with calcification.

Histology
Varies from well-differentiated hyaline cartilage with little nuclear atypia to highly anaplastic spindle cell tumor with little cartilaginous differentiation.

Treatment and Prognosis
- Surgical resection of the tumor.
- Do not respond to radiation or chemotherapy.
- Prognosis better than osteosarcoma since chondrosarcoma grows slowly and metastasizes late.

Ewing’s sarcoma
- Tumor of small round blue cells arising in the medullary cavity.
- Seven percent of primary bone tumors.
- Ninety percent of cases between ages 5 and 25 years; male > female.
- Most common sites: Diaphysis or metaphysis of long bones, pelvis, and scapula; potential to occur anywhere in the body.

EXAM TIP
Ewing’s sarcoma is the most lethal of all bone tumors.
Signs and Symptoms
- Pain that increases with time and is more severe at night.
- Local swelling and a tender mass.
- Malaise, fever, leukocytosis, mild anemia, increased ESR.
- Often mimic subacute osteomyelitis, syphilitic osteoperiostitis, or other tumors.

Radiography
- Shows lytic bone lesions with a permeative pattern.
- Elevations and permeations of the periosteum give rise to lamellated “onion skin” appearance.

Histology
Area contains densely packed small round blue cells containing glycogen with little intercellular stroma arranged in sheets, cords, or nests.

Treatment and Prognosis
- Vincristine, cyclophosphamide, actinomycin D, and adriamycin along with surgery or radiation.
- Advanced metastatic disease: Five-year survival is 30%.
- Surgically resectable lesion treated with drugs and surgery has a 70% chance of 5-year survival.
- Males have worse prognosis.

Multiple Myeloma
- A malignant plasma cell tumor with multiple site involvement.
- Most common primary malignant tumor of the bone (45%).
- Ninety percent of cases in patients over the age of 40 years; male > female.
- Most common sites: Vertebral column, ribs, skull, pelvis, femur, clavicle, and scalpula; can occur anywhere in the body.

Signs and Symptoms
- Bone pain.
- Weight loss, weakness, neurologic impairment if pathologic fractures in the vertebrae present.
- Pathologic fractures or deformities.
- Susceptibility to infections.
- Amyloidosis.
- Kidney damage due to protein plugging of renal tubules.

Labs
- Increased serum calcium due to bone reabsorption.
- Elevated uric acid due to increased cell turnover.
- Monoclonal gammapathy, Bence-Jones proteinuria, increased ESR, and rouleaux formation.
- Anemia due to marrow suppression.

Radiography
Classically shows sharply punched out lesions giving a soap bubble appearance or often shows diffuse demineralization.

Histology
Marrow aspirate shows larger-than-normal plasma cells with many nuclei, a nucleoli, and showing mitotic activity.

WARD TIP
Ewing’s sarcoma usually occurs after age 5. If patient < 5 years of age, think metastatic neuroblastoma instead.

EXAM TIP
Classic triad for multiple myeloma: pAM
- punched out”lytic lesions
- Atypical plasma cells
- Monoclonal gammopathy

EXAM TIP
Two percent of myeloma cases will present with pOeMs:
- Polyneuropathy
- Organomegaly
- Endocrinopathy
- M-component spike
- Skin changes/sclerosis of bone
Chemotherapy with melphalan, often with prednisone.
Bisphosphonates and other bone anti-resorptive agents help in reducing pathologic fractures.
Untreated cases rarely survive more than 6–12 months.
Chemotherapy induces remission in about 50–70% of cases.
Poor prognosis with 90% of patients dying within 2–3 years.

Metastatic tumors comprise 95% of all malignant bone tumors, primary bone tumors 5%.
Spread via direct extension, lymphatics, vascular system, or intraspinal seeding.
In children: Bone metastasis most likely from a neuroblastoma.
Most common sites: Vertebral column, ribs, pelvis, upper ends of femur and humerus.

Pain (most common symptom).
Spine involvement: Neurologic symptoms due to pressure on nerve roots or spinal cord.
Hypercalcemia and anemia.
Pathologic fractures are common.
Increased acid phosphatase in prostatic metastasis.

Order complete blood count (CBC), ESR, liver and renal panels, alkaline phosphatase, and serum protein electrophoresis.
CT of chest-abdomen-pelvis, x-ray of the entire effected bone.
Staging bone scan (more sensitive than x-ray).

Lesions may be multiple or solitary (kidney), well or poorly circumscribed, osteoblastic or osteolytic (majority).
Osteoblastic: Breast and prostate.

Generally, primary tumors produce matrix for the tumor stroma, whereas epithelial tumors form clusters in fibrous tissue.

Radiation as palliative treatment along with chemotherapy.
Surgical intervention aims at relieving pain and prevents pathologic fractures.
Radioactive iodine for thyroid carcinoma metastasis.
Tamoxifen for metastatic carcinoma of the breast.
Bilateral orchietomy, estrogen, or antiandrogens for metastatic prostate tumors.
Poor prognosis with average survival time being 19 months after suffering a pathologic fracture.
## The hand

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<td><strong>Other Common Hand and Wrist Injuries</strong></td>
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</table>
Anatomy

Muscles

- Intrinsic muscles of the hand have their origin and insertion in the hand. See Table 28-1.
- Extrinsic muscles of the hand have their muscle bellies in the forearm and their tendon insertions in the hand. See Table 28-2.

Bones

- There are 27 bones in the hand: 5 metacarpals, 14 phalanges, and 8 carpals.
- Each finger has one metacarpal.
- Each finger or digit (except the thumb) has three phalanges—proximal, middle, and distal.
- The thumb has only two phalanges—a proximal and a distal phalanx.
- The joints between the metacarpal and the proximal phalanx is the metacarpophalangeal (MCP).

Table 28-1. Intrinsic Muscles of the hand

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Innervation</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Thenar Group</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abductor pollicis brevis</td>
<td>Median</td>
<td>Abduction of thumb</td>
</tr>
<tr>
<td>Adductor pollicis brevis</td>
<td>Median</td>
<td>Adduction of thumb</td>
</tr>
<tr>
<td>Flexor pollicis brevis</td>
<td>Median</td>
<td>Flexes thumb MCP joint</td>
</tr>
<tr>
<td>Opponens pollicis</td>
<td>Ulnar (deep branch)</td>
<td>Opposes—pulls thumb medially and forward across palm</td>
</tr>
<tr>
<td><strong>Remainder of hand</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Palmar interossei</td>
<td>Ulnar</td>
<td>Adduct finger toward center of third digit</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flex MCP, extend PIP and DIP</td>
</tr>
<tr>
<td>Dorsal interossei</td>
<td>Ulnar</td>
<td>Adduct finger from center of third digit</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flex MCP, extend PIP and DIP</td>
</tr>
<tr>
<td>Lumbricals</td>
<td>First and second: Median</td>
<td>Flex MCP, extend PIP and DIP</td>
</tr>
<tr>
<td></td>
<td>Third and fourth: Ulnar (deep branch)</td>
<td></td>
</tr>
<tr>
<td>Palmaris brevis</td>
<td>Ulnar (superficial branch)</td>
<td>Aids with hand grip</td>
</tr>
<tr>
<td><strong>Hypothenar Group</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abductor digiti minimi</td>
<td>Ulnar (deep branch)</td>
<td>Abducts little finger</td>
</tr>
<tr>
<td>Flexor digiti minimi</td>
<td>Ulnar (deep branch)</td>
<td>Flexes little finger</td>
</tr>
<tr>
<td>Opponens digiti minimi</td>
<td>Ulnar (deep branch)</td>
<td>Aids little finger with cupping motion of hand</td>
</tr>
</tbody>
</table>
Table 28-2. extrinsic Muscles of the hand

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Compartment</th>
<th>Innervation</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexor carpi radialis</td>
<td>Anterior</td>
<td>Median</td>
<td>Flexes and abducts hand at wrist</td>
</tr>
<tr>
<td>Palmaris longus</td>
<td>Anterior</td>
<td>Median</td>
<td>Flexes hand</td>
</tr>
<tr>
<td>Flexor carpi ulnaris (humeral</td>
<td>Anterior</td>
<td>Ulnar</td>
<td>Flexes and abducts hand at wrist</td>
</tr>
<tr>
<td>and ulnar heads)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexor digitorum superficialis</td>
<td>Anterior</td>
<td>Median</td>
<td>Flexes middle phalanx</td>
</tr>
<tr>
<td>(humeroulnar and radial heads)</td>
<td></td>
<td></td>
<td>Assists with flexion of proximal phalanx and hand</td>
</tr>
<tr>
<td>Flexor digitorum profundis</td>
<td>Anterior</td>
<td>Median and ulnar nerves</td>
<td>Flexes distal phalanx</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Assists in flexion of middle and proximal phalanx and wrist</td>
</tr>
<tr>
<td>Extensor carpi radialis longus</td>
<td>Lateral</td>
<td>Radial</td>
<td>Extends and abducts hand at wrist</td>
</tr>
<tr>
<td>Extensor carpi radialis brevis</td>
<td>Posterior</td>
<td>Radial</td>
<td>Extends and abducts hand at wrist</td>
</tr>
<tr>
<td>Extensor digitorum</td>
<td>Posterior</td>
<td>Radial</td>
<td>Extends fingers and hand</td>
</tr>
<tr>
<td>Extensor digitii minimi</td>
<td>Posterior</td>
<td>Radial</td>
<td>Extends little finger MCP joint</td>
</tr>
<tr>
<td>Extensor carpi ulnaris</td>
<td>Posterior</td>
<td>Radial</td>
<td>Extends and adducts hand at wrist</td>
</tr>
<tr>
<td>Abductor pollicis longus</td>
<td>Posterior</td>
<td>Radial</td>
<td>Abducts and extends thumb</td>
</tr>
<tr>
<td>Extensor pollicis longus</td>
<td>Posterior</td>
<td>Radial</td>
<td>Extends distal phalanx of thumb</td>
</tr>
<tr>
<td>Extensor pollicis brevis</td>
<td>Posterior</td>
<td>Radial</td>
<td>Extends thumb MCP joint</td>
</tr>
<tr>
<td>Extensor indici</td>
<td>Posterior</td>
<td>Radial</td>
<td>Extends index finger MCP joint</td>
</tr>
</tbody>
</table>

- The joints between the phalanges are the proximal interphalangeal (PIP) and distal interphalangeal (DIP).
- The thumb has only MCP and DIP joints.
- Carpal bones (see Figure 28-1):
  - Scaphoid
  - Lunate
  - Triquetrum
  - Pisiform
  - Trapezium
  - Trapezoid
  - Capitate
  - Hamate

Nerves

- Sensory:
  - Radial: Sensory to lateral aspect of dorsum of hand and radial 3.5 digits.
  - Median: Sensory to skin on lateral half of palm and palmar aspect of the radial 3.5 digits.
  - Ulnar: Sensory to skin on ulnar aspect of dorsum of hand, hypothenar eminence, and ulnar 1.5 digits.
- Motor: See Tables 28-1 and 28-2 for muscles innervated by the radial, median, and ulnar nerves.

EXAM TIP

The wrist bones are easily remembered by the saying: Some Lovers Try Positions That They Can’t handle.

WARD TIP

The radial nerve does not innervate any of the intrinsic muscles of the hand. See Table 28-3 for clinical maneuvers to test function of the hand.
**Table 28-3. Clinical Maneuvers for Testing Muscles of the hand**

<table>
<thead>
<tr>
<th>Patient Maneuver</th>
<th>Muscle Tested</th>
</tr>
</thead>
<tbody>
<tr>
<td>Making a fist</td>
<td></td>
</tr>
<tr>
<td>Bending the tip of the thumb</td>
<td>Flexor pollicis longus</td>
</tr>
<tr>
<td>Bending each individual fingertip against resistance while PIPs are stabilized by examiner</td>
<td>Flexor digitorum profundus</td>
</tr>
<tr>
<td>Bending each individual fingertip against resistance while DIPs are stabilized by examiner</td>
<td>Flexor digitorum superficialis</td>
</tr>
<tr>
<td>Bring thumb out to side and back</td>
<td>Extensor pollicis brevis and abductor pollicis longus</td>
</tr>
<tr>
<td>Flexing and extending a fist at the wrist</td>
<td>Extensor carpi radialis longus and brevis</td>
</tr>
<tr>
<td>Raising thumb only while rest of hand is laid flat</td>
<td>Extensor pollicis longus</td>
</tr>
<tr>
<td>Making a fist with little finger extended alone</td>
<td>Extensor digiti minimi</td>
</tr>
</tbody>
</table>
Tendons

Zones of the hand

- Two main groups: Flexors and extensors.
- Extensor tendon lacerations can usually be repaired in the emergency department (ED).
- Flexor tendons are more difficult and usually require operative repair and careful rehabilitation.
- The flexor and extensor tendons are grouped into zones (see Figures 28-2 and 28-3).
- Flexor tendon injury repair timetable by zone:
  - Zones I and II: 1–3 weeks.
  - Zones III–V: Immediate.
  - Zone IV injuries are technically difficult because tendons lie within the carpal tunnel.
  - Zone V tendon injuries are relatively easy to fix, but functional outcome is often poor due to associated nerve injury.

![Diagram of tendon zones](Artwork by Elizabeth N. Jacobson, Mayo Medical School)
History

Focused hand history:
- Hand dominance.
- Time of injury.
- Status of tetanus immunization.
- Occupation.
- Cause and mechanism of injury.

Physical Examination

See Figure 28-4.

1. Sensibility:
   - Pinprick (two-point discrimination): Normal is $< 6$ mm when the points are static and $< 3$ mm when the points are moving. Abnormal values seen with underlying nerve injury.
   - Immersion test: Skin on palm of hand should wrinkle within 10 minutes when immersed in water. Failure to do so suggests underlying nerve injury.
2. Strength:
   - Test grip
   - Fromment’s sign

3. Vascular:
   - Capillary refill: Normal is < 2 seconds.
   - Allen test:
     - Patient makes a tight fist for 20 seconds.
     - Examiner occludes both ulnar and radial arteries by holding direct pressure.
     - Examiner releases ulnar artery—a normal (patent) ulnar artery will perfuse the hand within 5–7 seconds (color returns).
   - Test is repeated with the radial artery released to check ulnar flow.

4. Motor and sensory function: See section under Nerves for which nerves supply which muscles and sensory areas.

**Nerve Blocks**
- Used to anesthetize a portion of the hand innervated by certain nerve(s) (see Figure 28-4).
- Advantages over local anesthesia:
  - Does not distort area you want to examine/suture.
  - Eliminates need for multiple injections.

**Infections of the Hand**

**Felon**

**Definition**
Infection of the pulp space of any of the distal phalanges (see Figure 28-5).
etiology
Caused by minor trauma to the dermis over the finger pad.

Complications
Results in increased pressure within the septal compartments and may lead to cellulitis, flexor tendon sheath infection, or osteomyelitis if not effectively treated.

Treatment
- Using a digital block, perform incision and drainage (I&D) with longitudinal incision over the area of greatest induration but not over the flexor crease of the DIP.
- Drain may be placed and wound checked in 2 days.
- Antibiotics: Usually first-generation cephalosporin or anti-Staphylococcus penicillin.

Paronychia
definition
Infection of the lateral nail fold (see Figure 28-6).

etiology
Caused by minor trauma or activities such as nail biting or manicures.

Treatment
- Without fluctuance, this may be treated with a 7-day course of antibiotics, warm soaks, and retraction of the skin edges from the nail margin.
- For more extensive infections, unroll the skin at the base of the nail and at the lateral nail or I&D at area of most fluctuance using a digital block. Pus below the nail bed may require partial or total removal of the nail. Warm soaks and wound check in 2 days. Antibiotics are usually not necessary unless area is cellulitic.

sePTic Flexor TenosynovitiTis

etiology
This is a surgical emergency requiring prompt identification. Infection of the flexor tendon and sheath is caused by penetrating trauma and dirty wounds (e.g., dog bite). Infection spreads along the tendon sheath, allowing involvement of other digits and even the entire hand, causing significant disability.

Organisms
- Polymicrobial.
- Staphylococcus most common.
- Neisseria gonorrhoeae with history of sexually transmitted disease (STD).
**Gamekeeper’s Thumb**

**Definition**
Avulsion of ulnar collateral ligament of first MCP joint.

**Etiology**
- Forced abduction of the thumb.
- Can be associated with an avulsion fracture of the metacarpal base.

**Signs and Symptoms**
Inability to pinch.

**Diagnosis**
Application of valgus stress to thumb while MCP joint is flexed will demonstrate laxity of ulnar collateral ligament.

**Treatment**
- Rest, ice, elevation, analgesia.
- Thumb spica cast for 3–6 weeks for partial tears.
- Surgical repair for complete tears.

---

**EXAM TIP**
Gamekeeper’s thumb is commonly associated with ski pole injury (see Figure 28-7).
Carpal Tunnel Syndrome

**Definition**

Compression of the median nerve resulting in pain and sensory disturbances along the distribution of the nerve (see Figure 28-8).

**Etiology**

- Idiopathic/overuse (most common).
- Tumor (fibroma, lipoma).
- Ganglion cyst.
- Tenosynovitis of flexor tendons secondary to rheumatoid arthritis or trauma.
- Edema due to pregnancy, thyroid, or amyloid disease.
- Trauma to carpal bones.
- Gout.

**Risk Factors**

Repetitive hand movements.

**Epidemiology**

More common in women 3:1.

**Signs and Symptoms**

- Pain and paresthesia of volar aspect of thumb, digits 2 and 3, and half of digit 4.
- Activity and palmar flexion aggravate symptoms.
- Thenar atrophy: Uncommon but irreversible and indicates severe long-standing compression.
- Sensory deficit (two-point discrimination > 5 mm).

**Figure 28-8.** Carpal tunnel syndrome. (Reproduced, with permission, from LeBlond RF, DeGowin RL, Brown DD. DeGowin’s Diagnostic Examination, 8th ed. New York: McGraw-Hill, 2004: 736.)

**Exam Tip**

Carpal tunnel syndrome is the most common entrapment neuropathy.

**Typical scenario:** A 37-year-old female presents with pain in her right wrist and fingers, accompanied by a tingling sensation. The pain awakens her from sleep, and she is unable to perform her duties as a word processor. Think: Carpal tunnel syndrome.
**diagnosis**

- Tinel’s test: Tapping over median nerve at wrist reproduces pain and paresthesia.
- One minute of maximal palmar flexion reproduces pain and paresthesia.
- Consider erythrocyte sedimentation rate (ESR), thyroid function tests (TFTs), serum glucose, and uric acid level to look for underlying cause.

**Treatment**

- Treat underlying condition.
- Rest and splint.
- Nonsteroidal anti-inflammatory drugs (NSAIDs) for analgesia.
- Surgery for untreateable pain, thenar atrophy, and failure of nonoperative management.

---

**Ganglion Cyst**

**Definition**

A synovial cyst, usually present on radial aspect of wrist.

**Etiology**

Idiopathic.

**Signs and Symptoms**

- Presence of mass that patient cannot account for.
- May or may not be painful.
- Pain aggravated by extreme flexion or extension.
- Size of ganglia increases with increased use of wrist.
- Compression of median or ulnar nerve may occur (not common).

**Diagnosis**

Radiographs to ascertain diagnosis; since a ganglion cyst is a soft tissue problem only, no radiographic changes should be noted.

**Treatment**

- Reassurance for most cases.
- Wrist immobilization for moderate pain.
- Aspiration of cyst for severe pain.
- Surgical excision for cases involving median nerve compression and cosmetically unacceptable ganglia.

---

**Mallet Finger**

**Definition**

Rupture of extensor tendon at its insertion into base of distal phalanx (see Figure 28-9).

---

**Exam Tip**

Twenty to 50% of the population normally have a positive Phalen’s and Tinel’s test.

**Exam Tip**

Differential diagnosis includes bone tumor, arthritis, and intraosseous ganglion.

**Exam Tip**

Left untreated, mallet finger results in permanent boutonniere deformity.
eTiology

- Avulsion fracture of distal phalanx.
- Other trauma.

signs and syMPToMs

Inability to extend DIP joint.

TreatMent

- Splint finger in extension for 6–8 weeks.
- Surgery may be required for large avulsions of distal phalanx and for injuries that were not splinted early.

Trigger Finger
definition

Stenosis of fexor digitorum tendon sheath (A1 pulley) leading to nodule formation within the sheath (see Figure 28-10).
**Risk Factors**
- Rheumatoid arthritis
- Middle-aged women
- Congenital

**Signs and Symptoms**
Severe pain and snapping sensation or click when flexing and extending the digit.

**Treatment**
- Splinting of MP joint in extension.
- Injection of corticosteroid into tendon sheath.
- Surgical release of the AI pulley if above fail.

**Amputation Injuries**
**Prognosis is best for:**
- Sharp vs. crush or avulsion amputations.
- Children vs. adults.
- Clean vs. dirty.

**Indications for Surgery**
- Amputations with good prognosis as listed above.
- Thumb amputations (unlike other single digits, amputation of the thumb leaves a significant deficit in function of the hand).
- Multiple-digit amputations.

**Relative Contraindications To Surgical Reconstruction**
- Amputations in smokers (poor healing)
  - This is especially true if patient cannot commit to abstain from smoking for a 3-month period post reimplantation.
- Severe crush or avulsion injury.
- Grossly contaminated injuries.
- Amputations at multiple levels along the amputated limb.

**Complications**
- Stiffness.
- Cold intolerance.
- Decreased sensibility.
- Patients often require long-term hand therapy following surgery.

**Preoperative Management**
- IV antibiotics.
- Tetanus prophylaxis.
- NPO for possible surgery.
- Cleansing of wound site and amputated part with saline or lactated Ringer’s.
- Wrap amputated part in sterile gauze.
- X-ray both limb and amputated part.
High-Pressure Injection Injuries

- Commonly associated with grease guns, spray guns, and diesel fuel injectors.
- Usually occurs to index finger of nondominant hand.

**Signs and Symptoms**

- Seemingly innocuous puncture wound at initial presentation.
- Edema and minimal pain progress to more severe pain, discoloration, and swelling, then to intense tissue necrosis within 24 hours.

**Diagnosis**

Hand radiograph may reveal path of injected material if radiopaque.

**Treatment**

- IV antibiotics.
- Hospital admission for close observation.
- I&D—often operative; obtain hand surgery consult.
- Amputation may be necessary if part not salvageable.
- Physical therapy after acute management.

---

**Other Common Hand and Wrist Injuries**

See Table 28-4.

---

**Table 28-4. Common Hand and Wrist Injuries**

<table>
<thead>
<tr>
<th>Injury</th>
<th>Description</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| Boxer's fracture   | Fracture of neck of fifth metacarpal sustained in a closed fist injury       | - Ulnar gutter splint with MCPs flexed to 90° and PIP fully extended for 3–6 weeks  
|                    |                                                                             | - Surgical repair for:  
|                    |                                                                             |   - Any rotational deformity  
|                    |                                                                             |   - Angulation of fourth/fifth metacarpal > 40°  
|                    |                                                                             |   - Angulation of second/third metacarpal > 10–15°  
| Bennett fracture   | Fracture-dislocation of base of thumb                                       | - Initially immobilization in thumb spica cast  
|                    |                                                                             | - Definitive treatment is with surgical fixation  
| Rolando fracture   | Comminuted fracture of the base of the thumb                                | - Initially immobilization in thumb spica cast  
|                    |                                                                             | - Definitive treatment is surgical fixation  
| Scaphoid fracture  | - Most commonly caused by fall on outstretched hand  
|                    |   - Snuf box tenderness is classic                                          | - Immobilization in thumb spica cast with wrist in neutral position for 12 weeks  
|                    |   - May take up to 2 weeks to be seen on radiographs                       | - If no healing is seen by 4 weeks, likely surgical intervention since nonunion rate is very high due to tenuous blood supply  
|                    |   - Often requires scaphoid view or MRI to see fracture                     |                                                                           

(continues)
**Table 28-4. Common hand and Wrist Injuries** (continued)

<table>
<thead>
<tr>
<th>Injury</th>
<th>Description</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colles’ fracture</td>
<td>- Distal radius fracture with dorsal angulation</td>
<td>- Short arm cast for 4–6 weeks with volar flexion and ulnar deviation</td>
</tr>
<tr>
<td></td>
<td>- Most commonly caused by fall on outstretched hand</td>
<td>- Surgical repair for:</td>
</tr>
<tr>
<td></td>
<td>- “Dinner fork deformity” is classic</td>
<td>- Open fracture</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Comminuted fracture</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Intra-articular displacement &gt; 2 mm</td>
</tr>
<tr>
<td>Smith fracture</td>
<td>- Distal radius fracture with volar angulation</td>
<td>Surgical repair needed for most cases</td>
</tr>
<tr>
<td></td>
<td>- Most commonly caused by direct trauma to dorsal forearm</td>
<td></td>
</tr>
<tr>
<td>Galeazzi fracture</td>
<td>- Distal one third radial fracture with dislocation of distal radioulnar joint</td>
<td>Surgical repair needed for most cases</td>
</tr>
<tr>
<td></td>
<td>- Commonly caused by fall on outstretched hand with forearm in forced pronation or direct blow to back of wrist</td>
<td></td>
</tr>
<tr>
<td>Monteggia fracture</td>
<td>- Proximal one third ulnar fracture with dislocation of the radial head</td>
<td>Surgical repair for adults</td>
</tr>
<tr>
<td></td>
<td>- Commonly caused by fall on outstretched hand with forearm in forced pronation or direct blow to posterior ulna</td>
<td>- Closed reduction for children (children can tolerate a greater degree of displacement)</td>
</tr>
<tr>
<td></td>
<td>- May note injury of radial nerve</td>
<td></td>
</tr>
<tr>
<td>Nightstick fracture</td>
<td>- Isolated fracture of the ulnar shaft</td>
<td>- Long arm cast for 3–6 weeks</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Surgical repair for:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Angulation &gt; 10°</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Displacement &gt; 50%</td>
</tr>
</tbody>
</table>
Section IV

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AAST provides medical student scholarships to attend the annual meeting. You may be sponsored by a member of the AAST to attend next year’s annual meeting. You would be required to send a letter of recommendation by a member of the AAST for approval by the AAST Board of Managers. Once approved, your registration fee and banquet fee will be waived. The AAST will also cover the cost of your hotel room. In addition, you will be provided with an approximately $75 stipend for food/miscellaneous items. You/your institution are responsible for your travel arrangements. Contact the AAST for the deadline for medical student scholarship applications (CV and letter of recommendation from an AAST member), generally June of each year. For consideration, medical students should send their letters of recommendation and curriculum vitae (CV) to:

Robert C. Mackersie, MD
Trauma/Critical Care
UCSF-San Francisco General Hospital
1001 Potrero Avenue, Ward 3A
San Francisco, CA 94110
Phone: (415) 206-4622
Fax: (415) 206-5484
E-mail: rmackersie@sfgsurg.ucsf.edu

Eligibility: Open to medical students who plan to conduct a New York-based research project in urology

Application Deadline/Start Date: Generally each February

Additional Information: www.nyam.org/

Eligibility: Open to students who have completed their M1 or M2 year who are interested in participating in a mentored neurosurgical research project in Portland, Oregon.

Application Deadline/Start Date: Generally each February

Additional Information: www.ohsu.edu/ohsuedu/academic/som/neurosurgery/news-and-events/campagna-scholarship.cfm

Open to students, residents, and fellows in a university, affiliated hospital, or in a surgical or vascular surgical residency in the United States or Canada. Submissions must be original papers on a subject related to vascular surgery that have not been previously published or presented. Abstracts may be based on either experimental or clinical observations or it may be analytical, based on analysis and review of previous published data on the anatomy, physiology, pathology, biochemistry, or genetics of the vascular system and its disease. Finalists for this award will be notified and must submit seven copies of the manuscript to the Society’s office before the deadline specified for the annual
symposium. The 2009 award was $1,000 cash prize, plus round-trip coach air-fare reimbursement and three nights’ hotel accommodations.

First-round winners will each receive a cash award ($500 in 2009). The Karmody Poster Competition winner will receive an additional cash award.

- The competition is limited to fellows, residents, and medical students.
- All abstracts accepted for poster presentation will be considered. No additional field needs to be marked during the abstract submission process.
- Submissions must be original papers on a subject related to vascular surgery that have not been previously published or presented.
- Abstracts may be based on either experimental or clinical observations, or they may be analytical, based on analysis and review of previously published data on the anatomy, physiology, pathology, biochemistry, or genetics of the vascular system and its disease.

Expand your horizons in the care and treatment of the cancer patient by participating in state-of-the-art clinical research. Explore the mysteries of the cancer cell by participating in a basic scientific research program. Or participate in both! Special emphasis is placed on cancer prevention through lectures and practical experiences. The Roswell Park program provides competitive stipend support (projected at $350 per week) for students in the health professions (medicine, dentistry, and osteopathy) to engage in clinical and/or basic scientific research for an 8-week period. Some funding is available to defray costs of room. The program runs from early June through the end of July (contact Roswell Park directly for specifics); however, dates of participation may be changed to accommodate your academic schedule. **Applications:** Generally due in February of each year

**Additional Information:**
Arthur M. Michalek, PhD, FACE
Senior Vice President
Department of Educational Affairs
Roswell Park Cancer Institute
Carlton and Elm Streets
Buffalo, NY 14263
Phone: (716) 845-2339
E-mail: arthur.michalek@roswellpark.org

Research educational appointments for college students at the University of Texas M. D. Anderson Cancer Center provide firsthand experience in the areas of cancer research and insight into the varied career opportunities available in the biomedical sciences. To qualify for a college student research appointment, the applicant must be classified as a college freshman, sophomore, junior, or nongraduate senior at the time of appointment. The applicant should be pursuing a career in sciences and have a transcript that demonstrates a record of academic achievement. The appointment policy limits an individual to a 1-year appointment, which may be renewed upon the
recommendation of the faculty mentor. College students seeking individual appointments should contact the faculty members or department administrators in the departments of interest to obtain information about available positions and the eligibility requirements of each program.

**SIGN AAN Neurology Medical Student Programs**

The Student Interest Group in Neurology (SIGN) program is a network of more than 150 chapters in medical schools across the United States and Canada. SIGN fosters medical student interest in neurology by providing opportunities to participate in clinical, research, and service activities in neurology, increasing the student’s neurologic knowledge, and creating an interest in the AAN.

Free SIGN membership will enable you to:
- Socialize with students, residents, and faculty who share your interest in neurology
- Shadow neurologists
- Attend patient presentations and seminars
- Develop experience, leadership, and valuable contacts
- Join the nationwide SIGN network
- Meet other SIGN members at the Annual Meeting

Apply for SIGN scholarships:
- $3,000 Summer Research Scholarship
- $1,000 AAN Annual Meeting Scholarship

www.aan.com/go/education/awards

**Medical Student Summer Research Scholarship in Neurology**

**Application Deadline:** Generally each February

Sponsored by the AAN’s Undergraduate Education Subcommittee, the Medical Student Summer Research Scholarship program offers members of the AAN’s Student Interest Group in Neurology (SIGN) program a summer stipend of approximately $3,000 to conduct a project in either an institutional, clinical or laboratory setting where there are ongoing programs of research, service or training, or a private practice. Only applicants from schools with established SIGN chapters are eligible to apply. The project is to be conducted through a U.S. or Canadian institution of the student’s choice and jointly designed by the student and sponsoring institution. More than one student from an institution may apply, but only one student will be selected from an institution. The scholarship program was established to stimulate individuals to pursue careers in neurology in either research or practice settings.

The AAN will award up to twenty $3,000 scholarships to first or second-year AAN medical student members who have a supporting preceptor and a project with clearly defined goals. Third-year AAN medical student members who are on an official summer break will also be considered with accompanying documentation. One graduating medical student from each institution is eligible to receive the award.

The project is to be conducted through a U.S. or Canadian institution of the student’s choice and jointly designed by the student and sponsoring institution. Only applicants from schools with established SIGN chapters are eligible to apply. More than one student from an institution may apply, but only one student will be selected from an institution.

Applicants must be AAN medical student members at the time of application submission.
An medical student prize for excellence in neurology

**Application Deadline:** Generally each February

This award recognizes excellence in clinical neurology among graduating medical students. Only one medical student per institution should be nominated for the Medical Student Prize for Excellence. Awarded annually to a graduating medical student who exemplifies outstanding scientific achievement and clinical acumen in neurology or neuroscience, and outstanding personal qualities of integrity, compassion, and leadership. A Certificate of Recognition and a check for $200 will be presented on behalf of the AAN during the graduation or awards ceremony at each institution.

**Application Procedure:** Each department chair will designate a faculty committee that will select the award winner. The award winner will be selected based on outstanding performance in the neurology clerkship and outstanding personal and professional qualities, as noted in the award description.

Preference should be given to students choosing neurology as a career.

Submit material to calementi@aan.com, fax to (651) 361-4837 or mail to:

Medical Student Prize for Excellence in Neurology
Cheryl Alementi
American Academy of Neurology
1080 Montreal Avenue
St. Paul, MN 55116

**Application Deadline:** The deadline to nominate a medical student for the Medical Student Prize for Excellence is generally in February of each year.

medical student essAy Awar d—r ollAnd p. mAckAy Awar d

This award seeks to stimulate interest in the field of neurology as an exciting and challenging profession by offering highly competitive awards for the best essay. Essays are judged on the basis of the quality of the scholarship and on suitability for an audience of general neurologists.

This award is given for the best essay in historical aspects.

**Presentation:** Recipients are expected to give a poster presentation based on the selected manuscript at the AAN Annual Meeting.

Recipient will receive:
- Certificate of Recognition and $350 prize
- Complimentary registration for 61st Annual Meeting
- One-year complimentary subscription to Neurology journal
- Reimbursement for 61st Annual Meeting travel, lodging, and meal expenses (up to 2 days)
- Recognition at Awards Luncheon at Annual Meeting

**Eligibility:**
- Must be enrolled and in good standing in an accredited medical school in North America
- Must submit an original, lucid essay targeted to general neurologists (essay cannot be a previously published manuscript for Mackay)
- Must have spent less than 1 year on a project leading to the submitted essay

Applicants should submit one complete set of the following materials:
- Completed application form
- Essay using the following guidelines (essays will not be returned):
- Must be typed, double-spaced using a standard font
- Maximum length of 30 pages
- Only deceased persons may be subjects of biographical papers
- Letter attesting to eligibility criteria and identifying award category
- Maximum 200-word abstract
- Letter from a faculty sponsor detailing the extent of technical or financial support received, the student’s individual contribution to the project, and verifying that the student is the sole author of the essay

For more information, please contact Kyle Krause at kkrause@aan.com or (651) 695-2733.

**michael s. pessin stroke leadership prize**

Sponsored by the AAN and endowed by Dr. Pessin’s family, friends, and colleagues, this award recognizes emerging neurologists who have a strong interest in and have demonstrated a passion for learning and expanding the field of stroke research. Applicants should have an active involvement in providing patients with the highest quality of compassionate care. This award is intended to stimulate and reward individuals in the developmental stages of their careers, who demonstrate a passion for stroke.

**Presentation:** Recipient is expected to give a 10-minute presentation on a topic of their choice during a scientific session at the AAN Annual Meeting.

Recipient will receive:
- Certificate of Recognition and $1,500 prize
- Complimentary registration for Annual Meeting
- Recognition at Awards Luncheon at Annual Meeting

**Eligibility:**
- Must be a medical student, resident, fellow, or junior faculty member involved in or considering a career in neurology, emphasizing the care of stroke patients
- Must be no more than 5 years from completion of most recent training program and no higher academic rank than assistant professor
- Additional consideration will be given to those involved in clinical research aimed at enhancing the understanding of stroke or improving acute treatment protocols

**Application Procedure:** Applicants should submit one complete set of the following materials:
- Completed application form
- Current curriculum vitae

For more information, please contact Franziska Schwarz at fschwarz@aan.com or (651) 695-2807.

**medical student essay award—extended neuroscience award**

This award seeks to stimulate interest in the field of neurology as an exciting and challenging profession by offering highly competitive awards for the best essay. Essays are judged on the basis of the quality of the scholarship and on suitability for an audience of general neurologists.

This award is given for the best essay in Neuroscience.

**Presentation:** Recipients are expected to give a poster presentation based on the selected manuscript at the AAN Annual Meeting.
Recipient will receive:
- $1,000 prize
- Complimentary registration for Annual Meeting
- One-year complimentary subscription to Neurology journal
- Reimbursement for Annual Meeting travel, lodging, and meal expenses (up to 2 days)
- Recognition at Awards Luncheon at Annual Meeting

Eligibility:
- Must be enrolled and in good standing in an accredited medical school in North America
- Must submit an original, lucid essay targeted to general neurologists
- Must have spent more than one year on a project leading to the submitted essay

Applicants should submit one complete set of the following materials:
- Completed application form
- Essay using the following guidelines (essays will not be returned):
  - Must be typed, double-spaced using a standard font
  - Maximum length of 30 pages
  - Only deceased persons may be subjects of biographical papers
  - Letter attesting to eligibility criteria and identifying award category
  - Maximum 200-word abstract
  - Letter from a faculty sponsor detailing the extent of technical or financial support received, the student's individual contribution to the project, and verifying that the student is the sole author of the essay

For more information, please contact Kyle Krause at kkrause@aan.com or (651) 695-2733.

**medical student essay award—g. milton shy award**

This award seeks to stimulate interest in the field of neurology as an exciting and challenging profession by offering highly competitive awards for the best essay. Essays are judged on the basis of the quality of the scholarship and on suitability for an audience of general neurologists.

This award is given for the best essay in clinical neurology.

**Presentation:** Recipients are expected to give a poster presentation based on the selected manuscript at the AAN Annual Meeting.

Recipient will receive:
- Certificate of Recognition and $350 prize
- Complimentary registration for Annual Meeting
- One-year complimentary subscription to Neurology journal
- Reimbursement for Annual Meeting travel, lodging, and meal expenses (up to 2 days)
- Recognition at Awards Luncheon at Annual Meeting

**Eligibility:**
- Must be enrolled and in good standing in an accredited medical school in North America
- Must submit an original, lucid essay targeted to general neurologists (essay cannot be a previously published manuscript for Shy)
- Must have spent less than 1 year on a project leading to the submitted essay

Applicants should submit one complete set of the following materials:
- Completed application form
- Essay using the following guidelines (essays will not be returned):
  - Must be typed, double-spaced using a standard font
- Maximum length of 30 pages
- Only deceased persons may be subjects of biographical papers
- Letter attesting to eligibility criteria and identifying award category
- Maximum 200-word abstract
- Letter from a faculty sponsor detailing the extent of technical or financial support received, the student’s individual contribution to the project, and verifying that the student is the sole author of the essay

For more information, please contact Kyle Krause at kkrause@aan.com or (651) 695-2733.

**Medical Student Essay Award—Saul R. Korey Award**

This award seeks to stimulate interest in the field of neurology as an exciting and challenging profession by offering highly competitive awards for the best essay. Essays are judged on the basis of the quality of the scholarship and on suitability for an audience of general neurologists.

This award is given for the best essay in experimental neurology.

**Presentation:** Recipients are expected to give a poster presentation based on the selected manuscript at the AAN Annual Meeting.

Recipient will receive:
- Certificate of Recognition and $350 prize
- Complimentary registration for Annual Meeting
- One-year complimentary subscription to Neurology journal
- Reimbursement for Annual Meeting travel, lodging, and meal expenses (up to 2 days)
- Recognition at Awards Luncheon at Annual Meeting

**Eligibility:**
- Must be enrolled and in good standing in an accredited medical school in North America
- Must submit an original, lucid essay targeted to general neurologists (essay cannot be a previously published manuscript for Korey)
- Must have spent less than 1 year on a project leading to the submitted essay

**Application Procedure:** Applicants should submit one complete set of the following materials:
- Completed application form
- Essay using the following guidelines (essays will not be returned):
  - Must be typed, double-spaced using a standard font
  - Maximum length of 30 pages
  - Only deceased persons may be subjects of biographical papers
  - Letter attesting to eligibility criteria and identifying award category
  - Maximum 200-word abstract
  - Letter from a faculty sponsor detailing the extent of technical or financial support received, the student’s individual contribution to the project, and verifying that the student is the sole author of the essay

For more information, please contact Kyle Krause at kkrause@aan.com or (651) 695-2733.

**University of California Davis Ophthalmology Summer Fellowship**

Department will offer summer fellowship stipend of $2,000 for expenses and supplies for a medical student to pursue a research project. Students first must find a project and a mentor for the summer project. Then the
A student will need to write up the project proposal with a budget and submit it to the Department of Ophthalmology Research Committee. The committee will award the stipends based on the quality of the submitted proposal.

Additional funding besides a summer stipend for medical student research is available. Students first must find a project and a mentor for the project. Then the student will need to write up the project proposal with a budget and submit it to the Department of Ophthalmology Research Committee. The committee will award the stipends based on the quality of the submitted proposal.

The projects are of two types:

1. (Short Projects) These projects should be completed in a relatively short time frame (e.g., 2 to 3 months). A student could start it on a 4- to 8-week rotation and then write the project up at a later time. The type of projects that would be appropriate might be:
   a. Case reports
   b. Chart reviews on particular projects asking a limited and specific question

2. (Long Projects) These projects would be more long-range projects that would take 6 to 12 months. The student would be taking off from medical school to do the project.
   a. The projects may be prospective clinical projects with a well-defined question, mentor, research plan, and budget with funding defined.
   b. The project may be a laboratory project with a well-defined question, mentor, research plan, and budget with funding defined.

Web site: www.ucdmc.ucdavis.edu

Harvard-Longwood Research Training in Vascular Surgery Summer Research Fellowships in Vascular Surgery:

- This experience is supported by the William J. von Liebig Summer Research Fellowship program.
- Four student research fellowships available starting (generally) on June 1.
- Program runs 10–12 weeks over the summer, with research training in molecular and cell biology, coagulation and thrombosis, atherogenesis, intimal hyperplasia, prosthetic/host interactions, and thrombosis.
- Trainees will pursue a program of intense research activity, which will be carried out under the guidance of a selected faculty advisor based at one of our Harvard Medical School hospitals.
- Applicants should have a minimum of 1 year of medical school at an LCME accredited school.
- Student will receive a $5,000 stipend for the summer.
- Application deadline is generally January.

For more information, contact:

Leena Pradhan, PhD
William J. von Liebig Summer Research Fellowship
Harvard Institutes of Medicine
4 Blackfan Circle, Room 130
Boston, MA 02115
Phone: (617) 667-0096
Fax: (617) 975-5300
E-mail: lpradhan@bidmc.harvard.edu
nyu hospital for Joint diseases department of orthopaedics
(year-long fellowship)

- One-year fellowship for medical students interested in pursuing a career in orthopaedics.
- The program is centrally located at the Hospital for Joint Diseases and Bellvue Medical Center with some travel to Jamaica Hospital in Queens, New York.
- Responsibilities will include participation in ongoing studies, maintenance of the Orthopaedic Trauma Service database, and submission of IRB protocols.
- Applicants should be in medical school or a recent graduate.
- A monthly stipend is given to help offset the cost of New York City housing, which is not provided.
- For more information, go to: www.med.nyu.edu/orthosurgery/research/opp.html

For more information, please contact:
Kenneth Egol
E-mail: kenneth-egol@nyumc.org

American otological society medical student research trainings fellowships

**Purpose:** To further the study otosclerosis, Ménière’s disease, and related ear disorders.

The American Otologic Society, Inc., through its Research Fund, is offering Research Grant Awards and full time Research Training Fellowships to study otosclerosis, Ménière’s disease, and related ear disorders in U.S. or Canadian institutions only. Proposals may include investigation of the management and pathogenesis of these disorders, and underlying processes.

**Research Training Fellowship:** For physicians only (residents and medical students), fellowship will support 1–2 years’ full-time research conducted outside of residency training; $35,000 for stipend, $5,000 for supplies (and up to 10% indirect costs).

Applications must be accompanied by sponsoring institution documentation stating that facilities and faculty are appropriate for requested research. Research conducted during the Research Training Fellowship can be on any topic related to ear disorders.

**Deadline:** Grant and fellowship applications must be postmarked by January 31.

Information and materials may be obtained from www.americanotologicalsociety.org/forms.html or by contacting:
Lloyd B. Minor, MD, Executive Secretary
Research Fund of the American Otological Society, Inc.
Johns Hopkins University, School of Medicine
Department of Otolaryngology–Head & Neck Surgery
601 N. Caroline Street, JHOC 6210
Baltimore, MD 21287-0910
Phone: (410) 955-1080
Fax: (410) 955-6526
E-mail: lminor2@jhmi.edu

The nyu urology summer fellowship

This is an 8-week opportunity for two students between their first and second years of medical school to learn more about this exciting and diverse surgical subspecialty. This summer experience will allow students to spend time...
in the operating room observing procedures in urologic oncology, female and male incontinence, infertility, erectile dysfunction, stone disease, and pediatric urology. Students will also spend time in the clinic observing urological outpatient procedures and office visits with patients before and after surgery. Summer fellows will attend teaching conferences and journal clubs run by faculty and residents.

To round out this exciting summer experience, students will be assigned to work with one of the Urology attending physicians on a clinical research project. For highly motivated students, this is a great opportunity to take a project from the start and create abstracts and manuscripts eligible for submission to national conferences and peer-reviewed journals for publication. Students will have the opportunity and are strongly encouraged to continue this research following completion of the summer fellowship. Finally, at the end of the fellowship, students will give a short oral presentation to the Department of Urology on a topic to be decided. This summer program will provide students with great exposure to this surgical subspecialty and research opportunities that can lead to medical conference presentations and publications.

**Eligibility and Salary:** Students must be between their first and second years of medical school to apply.

A stipend of $800 is provided. In addition to the stipend, students may receive additional funds in the amount of $2,100 through the NYU School of Medicine Office of Financial Aid if they qualify for work study. However, work-study eligibility is not required to apply.

**How to Apply:** Please forward a one-page cover letter addressed to Dr. William Huang, Director of the Urology Summer Fellowship, describing your interest in the fellowship, along with your resume, to Sabine Gay, program coordinator, at Sabine.Gay@nyumc.org. For further information, call (646) 825-6310.

**Application Deadline and Important Dates:** Students must submit applications by February 15 for the upcoming summer.

Notification of acceptance into this program will be in mid-March. The fellowship program runs from mid-June to early August.

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Available June 1. Four medical student research fellowships are available for 10–12 weeks of summer research training in molecular and cell biology, biomechanics, coagulation and thrombosis, and angiogenesis, with a focus on clinically relevant problems such as atherogenesis, intimal hyperplasia, prosthetic/host interactions, and thrombosis. Trainees will pursue a program of intense research activity. This training program is designed to provide medical students an initial exposure to vascular surgery research.

Students will carry out their research projects under the guidance of a faculty advisor, selected from renowned vascular researchers based at four Harvard Medical School hospitals: Beth Israel Deaconess Medical Center, Brigham and Women’s Hospital, Children’s Hospital (Boston), and the Joslin Diabetes Institute, as well as the Massachusetts Institute of Technology.

Selection of trainees is based on candidates’ demonstrated ability. Applicants should be medical students who have completed at least 1 year of study at an LCME accredited school. Students must be U.S. citizens or permanent residents (green card holders).
Interested applicants are encouraged to submit a personal statement, together with a curriculum vitae, dean/advisor or program director’s letter, and two letters of recommendation. Selection is based on merit only, without bias to gender, race, color, or ethnic origin.

**Support:** A $5,000 stipend for the summer and appointment at Harvard Medical School as a Research Fellow in Surgery.

**Student Program Director:**
Frank W. LoGerfo, MD  
Chief, Division of Vascular Surgery  
Beth Israel Deaconess Medical Center  
William V. McDermott Professor of Surgery  
Harvard Medical School

**Contact:**
Leena Pradhan, PhD  
William J. von Liebig Summer Research Fellowship  
Harvard Institutes of Medicine  
4 Blackfan Circle, Room 130  
Boston, MA 02115  
Tel: 617-667-0096  
Fax: 617-975-5300  
E-mail: lpradhan@bidmc.harvard.edu

**The American Association of Neurological Surgeons (AANS) through the neurosurgery research and education foundation (nref)**

The new AANS Medical Student Summer Research Fellowship (MSSRF) program. The fellowship is open to medical students in the United States or Canada who have completed 1 or 2 years of medical school and wish to spend a summer working in a neurosurgical laboratory, mentored by a neurosurgical investigator who is a member of the AANS and will sponsor the student.

This year, 15 Medical Student Summer Research Fellowships will be awarded in the amount of $2,500 per award.

To be considered for this award, applications need to be received by February 1. Awardees will be notified and posted on the AANS website by March 31.

Submit completed applications to:
AANS Medical Student Summer Research Fellowship  
c/o American Association of Neurological Surgeons  
5550 Meadowbrook Drive  
Rolling Meadows, IL 60008-3852

or e-mail application and all supporting documents to nref@aans.org.

For more information about this medical student summer research fellowship program, contact the Development Department, toll free at (888) 566-2267 or info@aans.org.

**The Orthopaedic Research and Education Foundation**

The Orthopaedic Research and Education Foundation sponsors a Medical Student Summer Orthopaedic Research Fellowship. Our goal is to encourage medical students considering a career in orthopaedics to gain experience in basic, clinical, or translational research.
The Orthopaedic Research and Education Foundation (OREF) is an independent organization that raises funds to support research and education on diseases and injuries of bones, joints, nerves, and muscles. OREF-funded research enhances clinical care, leading to improved health, increased activity, and a better quality of life for patients.

Fellowship Description:
- Medical students with an interest in orthopaedics are eligible to apply.
- The medical student needs to identify an investigator at a U.S. institution with an ongoing orthopaedic research project who is willing to provide research training to the student and act as his/her mentor.
- OREF will provide $2,500 as salary support for the student, payable directly to the sponsoring institution. OREF will reimburse the institution for FICA taxes of up to $200 and up to $200 for supplies, if requested. No other fringe benefits are authorized.
- This program is intended to be a summer research fellowship. A minimum of 8 weeks’ full-time work on a specific project is required. The research project should be one that the student is not already involved in.
- At the completion of the program, the medical student is required to complete an evaluation form provided by OREF. The mentor will also complete an evaluation form.

Orthopaedic Research and Education Foundation (OREF)
6300 North River Road, Suite 700
Rosemont, IL 60018-4261
847-698-9980/847-698-9981
Web site: http://www.oref.org

The decrease in the number of physicians interested in pursuing a career as a clinician-scientist is well documented in lower numbers of NIH applications and in AAMC questionnaires. The Department of Surgery at the University of Wisconsin is uniquely qualified to direct a short-term summer research experience for medical students who are interested in research related to diabetes, obesity, endocrine disorders, nutritional disorders, digestive diseases, liver disease, kidney disease, and urologic disease. The overall program goal of this Department of Surgery training program is to provide six medical students with a focused, mentored 12-week research and training experience that will help students discern their career path, preferably toward a career that involves biomedical research. During this experience, all students will plan and complete a research project, complete a learning contract, attend an established curriculum in effective research and academic conduct, and write an abstract for submission and presentation at a medical meeting. The training program will foster the development of knowledge, competence, skills, professional attitudes, and experience required to understand what is involved in successful academic careers in laboratory or clinically based research related to the goals of the NIDDK. The four specific objectives of this program are to:

1. Expose medical students, early in their training, to the excitement and challenges of a research career through participation in an individual, mentored training experience.
2. Encourage students to pursue a research career through participation in a focused 12-week research experience in a research program of relevance to the mission of NIDDK.
3. Provide a didactic curriculum emphasizing research issues.
4. Increase the pool of medical students who pursue further research activities by partnering medical students with physicians and scientists who will serve as role models for a career combining patient care and scientific research. The training program includes plans to follow up with trainees later in their career to evaluate whether their summer surgical research experience in the Department of Surgery led to additional research training and a career as a clinician-scientist.

**Contact:** Herbert Chen, MD, University of Wisconsin–Madison

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**Award Amount:** $2,500

**Number of Awards:** 2

**Purpose:** The CNS/CSNS Medical Student Socioeconomic Fellowship supports a medical student conducting research on a socioeconomic issue impacting neurosurgical practice.

**Eligibility:** The fellowship is open to all medical students in the United States and Canada. The fellow will spend 8–10 weeks conducting supervised research on a socioeconomic topic of importance to neurosurgery.

**Requirements:** The fellow must submit a final report to the CNS Fellowships Committee by December 1 following completion of the summer fellowship. Any publications resulting from supported research must acknowledge the support.

**Eligible Expenses:** Financial support is exclusively for stipend support to the student.

**Application Requirements:**
- Complete the online CNS/CSNS Medical Student Fellowship Application.
- Two reference letters.
- Curriculum vitae of applicant.
- Curriculum vitae of proposed mentor.
- Attach a headshot photograph.

**Congress of Neurological Surgeons**
Elad I. Levy, MD
Chairman, CNS Fellowships Committee
10 North Martingale Road, Suite 190
Schaumburg, IL 60173
Phone: (847) 240-2500
Fax: (847) 240-0804
Toll Free: (877) 517-1CNS
E-mail: info@1CNS.org

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**Fight for Sight—Summer Student Fellowships**

Awards of $2,100 are offered to currently enrolled undergraduates, medical students, or graduate students who wish to explore ophthalmology or eye research as a career. Students are expected to complete a short, independent project during the summer months under the guidance of a senior scientist or clinician. The goal of this award is to advance the skills needed to initiate and carry out research in a scientific environment. Lab employees are not eligible for this fellowship.

Go to www.fightforsight.com/grant_IGP.php for more information.
This is an 8-week program of mentored research designed to introduce students who have completed their first year of medical school to research opportunities in orthopaedic basic science, translational science, and clinical research in orthopaedics.

The fellowship program is built around a summer research project that is directed by an orthopaedic surgeon or scientist at the Hospital for Special Surgery (HSS). Both clinical and basic science projects are offered, and these are detailed below.

The fellowship program includes the opportunity for weekly observation of orthopaedic surgical procedures with a variety of clinical faculty. In addition, a weekly seminar series and discussion group provides the opportunity for students to expand their understanding of the fundamentals of medical research and clinical orthopaedics.

The 8-week program runs from late June through mid-August (in 2009: June 22 to August 14) and includes a $2,400 stipend. Up to 15 fellowship awards will be granted.

To apply for the program, eligible students should first contact an orthopaedic surgeon or scientist who is offering a research project that is of interest (see list below). Once the student has been accepted by the surgeon or scientist, the student and mentor may apply for the fellowship together. Alternatively, if a student has an established working relationship with a faculty member, projects not listed can be submitted for consideration.

Students who have completed their first year in an accredited U.S. medical school are eligible to apply. Preference will be given to those students attending Weill Cornell Medical College.

Download the Fellowship Application Form.

The applications must be sent to Lizandra Portalatin (portalatinl@hss.edu) by April 3. Students and mentors will be notified of the award by May 1.

For questions, contact Chisa Hidaka, MD, Assistant Scientist, Hospital for Special Surgery, at hidakac@hss.edu or (212) 774-2384.

Fellowship Coordinators: Chisa Hidaka, MD, and Jo A. Hannafin, MD, PhD

The Summer Intern Scholarship Program was established in 2007 to introduce the field of cardiothoracic surgery to first- and second-year medical students from North American medical schools. In the 2 years since its inception, the Summer Intern Scholarship has offered 100 medical students the opportunity to broaden their educational experience by providing scholarships to spend 8 weeks during the summer working in an AATS member’s cardiothoracic surgery department.

Web site: www.aats.org/MSSR/medicalStudents.html
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