FIRST AID FOR THE®

SECOND EDITION

EMERGENCY MEDICINE clerkship

A STUDENT-TO-STUDENT GUIDE

Completely revised and updated

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High-yield topics help you ace the in-service, shelf, and USMLE Step 2 exams

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Succeed in the clerkship, impress in the Emergency Department

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Includes tear-out cards to help you excel in a busy ED

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FIRST AID

FOR THE®

Emergency Medicine Clerkship

Second Edition

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INTRODUCTION

This clinical study aid was 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; however, a stronger clinical emphasis was placed on its content. You will find that rather than simply preparing you for success on an exam, this resource will also help guide you in the clinical diagnosis and treatment of many of the problems seen by emergency physicians.

The content of the book is based on the American College of Emergency Physicians (ACEP) and Society of Academic Emergency Medicine (SAEM) recommendations for the Emergency Medicine curriculum for fourth-year medical students. It also contains information derived from the Core Curriculum, an outline developed by the Residency Review Committee, which details the information that EM residents are expected to learn and will ultimately be responsible for on their oral and written board exams. Each of the chapters contains the major topics central to the practice of EM and has been specifically designed for the medical student learning level. In addition, special chapters such as Diagnostics and Procedures have been included to emphasize the more clinical nature of EM.

The content of the text is organized in the format similar to other texts in the *First Aid* series. Topics are listed by bold headings, and the "meat" of the topic provides essential information. The outside margins contain mnemonics, diagrams, exam and ward tips, summary or warning statements, and other memory aids. Exam tips are marked by 📝, ED tips by the symbol 🩺, and typical scenarios by the symbol 🧍.

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How to Succeed in the Emergency Medicine Clerkship

There is very little you will need to have on your person while working in the department. A basic list of equipment to carry with you includes:

1. Several black pens
2. Stethoscope
3. Trauma shears
4. Small notepad to track patients and record important teaching points
5. Penlight
6. A pocket-sized drug reference (e.g., Tarascon's *Pharmacopeia*)
7. EMRA's *Guide to Antibiotic Use in the Emergency Department* or Sanford's *Guide to Antimicrobial Therapy*

EM is a specialty with many unique aspects, which makes this clerkship a popular favorite. These include:
- A large variety of presenting complaints
- Being the first one to see a patient, which means being the first one to come up with a diagnosis
- Opportunity to do a number of procedures
- Opportunity to function as a real member of the resuscitation teams
- Opportunity for close interaction with attendings
- Constant ongoing teaching, with the opportunity to pick up many "pearls"

Many of the things that make EM so enjoyable may also pose a challenge at times:
- For many, the emergency department (ED) is the only place to obtain care, so what they perceive as an emergency may not be what you perceive as one. Often, eliciting the underlying issue requires a little finesse and remaining nonjudgmental. For example, the patient who presents with a rash of 3 weeks' duration at 4 A.M. may actually be a victim of domestic violence. The patient who presents multiple times with a complaint of pain with negative workup may be drug seeking. Because so many patients who present to the ED have underlying social and psychological issues, history taking can be quite challenging. It is important to remain nonjudgmental and provide the best possible care under sometimes less-than-optimal circumstances.
- The 24-hour open door policy of the ED, long waiting times, and uncomfortable waiting environment, along with the stress of high-acuity complaints, predispose to violence in the ED. Students should be aware of their environment and practice personal safety behavior as they would in any other potentially dangerous environment.
- While resuscitations are an exciting opportunity for students to learn and practice procedures, students often forget about universal precautions because of all the excitement, putting themselves at risk for needle-stick injury. Remember, ALWAYS wear gloves, NEVER recap needles. Report any exposure to body fluids to the ED attending immediately.

► HOW TO DRESS

Every ED will have a dress code. It is in your best interest to find out prior to your first shift what you are expected to wear. If for some reason this is not possible, men should wear any color shirt with tie, pants (not jeans), shoes (not sneakers), and a short white med-student coat. Women should also wear professional attire with the short white coat (no jeans, no sneakers). Although most people wear scrubs, it is not a universal rule that one can wear scrubs in the ED!

However, most departments are usually relaxed about what is acceptable and do allow scrubs, and sneakers/clogs.

► WHAT TO DO (HOW TO BEHAVE)

There are a few things we can say about what makes a medical student look good. Generally, a medical student who can handle a resident's/intern's workload in the ED already demonstrates his or her value to the department at an early stage. However, not having the extra year or two of clinical experience a resident may have, this presents a challenge.

It is best to try to emulate an efficient and thorough resident that you may work beside. Every institution has its particular procedures for assigning patients, charting, "starting up" patients, admitting and discharging patients, and ordering labs or radiologic studies. Your efficiency in the department will be markedly improved if you can familiarize yourself with these administrative hurdles early on.

A few general pointers:
- Punctuality speaks for itself. Be the first one to arrive for sign-over rounds, codes, lectures, and grand rounds.
- Know all the important numbers for the department (door and copier codes, pager numbers, etc.).
- Ask the nurses for their help should you need it. They can be valuable allies and generally have the ear of the attendings.
- Be thorough in your history and physical, but presentations should include only the essentials (quick bullet).
- Show an interest in what you are doing (fecal disimpactions can be fun!!).
- Charting: "The chart is your life raft in a sea of litigation!" We guarantee if your document includes these things, you are already ahead of the game:
  - Times of initial evaluation, all orders, repeat exams, radiology/lab results, discharge instructions.
  - Chief complaint and a good history *with review of systems*.
  - Exam (don't forget the neuro/mental status/rectal exams) and *repeat* exam data as things change.
  - Orders must be countersigned by a resident or attending.
  - *Write* the lab, radiology, and electrocardiogram results on the chart.
  - Discharge instructions must include *follow-up* arrangements and instructions for what to *return* for.
  - If a patient wants to sign out against medical advice, notify the attending.

▶ **WHAT NOT TO DO**

- Be late
- Make up an answer to a question you might not know (just say you don't know)
- Look sloppy
- Seem uninterested
- Turn down the opportunity to do a procedure (even if you've done it before)

▶ **WHAT TO READ**

Most EDs have a small collection of texts (usually locked up in a rack). If you have the time to read (i.e., the ED isn't that busy), you can read a little in one of the texts on each patient you see. However, *this book* should ultimately be the only one you'll need for the clerkship. If you feel the inevitable need to pore through page after page of medical minutia, you should do it when you are not working in the department. We advise that you write a little bit about each patient you see during your shift and then read up about them when you get home or at the library.

Suggested resources include:
- *Emergency Medicine (A Comprehensive Study Guide)*, 5th ed., Judith Tintinalli, 2127 pages. Designed as board review for residents.
- *Pediatric Emergency Medicine (A Comprehensive Study Guide)*, 1st ed., Gary Strange, 728 pages. Designed as board review for residents.
- *Clinical Procedures in Emergency Medicine*, 3rd ed., Roberts/Hedges, 1297 pages. A how-to for nearly all procedures you might be doing within the department.
- *Emergency Medicine (Concepts and Clinical Practice)*, 4th ed., Rosen/Barkin, 2930 pages. A text resource for reading on the pathophysiology as well as clinical aspects of disease.

▶ **THE EXAM**

The last hoop you will need to jump through before finishing the clerkship will be the exam. Many departments will have their own exam designed for the fourth-year medical student rotator (almost always multiple guess). Some will use a "shelf exam" or one that is almost identical to the one used for resident yearly "in-service" exams. These are sometimes more difficult, but expectations of your performance are a bit lower. Some programs will further torture you by giving you an oral exam. This is a lot like taking the oral board exam as a graduating resident, only less strenuous and with less at stake.

The best strategy for doing well on the written, multiple-choice exam is to do A LOT of practice questions, beforehand. *PEER VI* is a collection of questions put out by the ACEP as a board preparation (most like the shelf exam). This is probably your best source of questions, as they are written by EM physicians for EM physicians, cover the specialty in appropriate scope and detail, and contain extensive explanations for each question. Your best bet to

obtain a copy of *PEER VI* is to ask the residents in the program; someone is bound to have a copy you may borrow. There are a number of commercially published question-and-answer texts that are available but not as good. However, going through this many resources is probably overkill, as this book, *First Aid for the Clinical Clerkship in Emergency Medicine,* will have all the facts you need for both the test and the clerkship.

Seeing as many patients as possible and presenting cases to the attendings and senior residents is the best form of preparation for the oral exam.

A WORD ABOUT RESIDENCIES

ED attendings, residency directors, and department chairpersons will be observing you as a potential resident. You are, in a sense, auditioning for a position in the match. Residents you may work with can be your allies and help you "look good" to the attendings and ultimately attain a residency position (if this is your goal).

You are generally expected to do a rotation in your home hospital's department (the one affiliated with your medical school). Outside of that, it is always a good idea to do a rotation in the hospital where you would most like to do your residency. Fall is the best season for this, as it is the beginning of interview season. You will most likely get an interview, barring any medical disasters you may precipitate or gross personality conflicts with the staff. Interviewing after your rotation usually is more of a formality since most of the attendings have already worked with you and know you (see the advantage?).

OK, good luck . . . enjoy the book.

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High-Yield Facts

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Resuscitation

*Based on 2005 American Heart Association Guidelines
for CPR and Emergency Cardiovascular Care*

Goal of BLS: Increasing the survival rates of cardiac and respiratory arrest through the training of laypersons to:
- Recognize the symptoms of inadequate circulation or respiration.
- Immediately activate the Emergency Medical Services (EMS).
- Support the circulation and respiration via cardiopulmonary resuscitation (CPR) and rescue breathing.

BLS Protocol (3As)
- **A**ssessment: Determine unresponsiveness of the patient.
- **A**ctivate the EMS system immediately by calling 9-1-1.
- **A**BCs of CPR (airway, breathing, and circulation).

Airway with C-Spine Control

- Position the patient supine on a flat surface using "logroll" technique.
- Open the airway using head tilt–chin lift maneuver or the jaw thrust maneuver.

Breathing

- Look, listen, and feel for adequate breaths (approximately 3 to 5 seconds).
- Perform **rescue breathing** (mouth-to-mouth, etc.):
  - Give two initial breaths over 1 second.
  - Deliver 10 to 12 breaths per minute; 8 to 10 if advanced airway in place.

Circulation

- Determine pulselessness by checking carotid artery pulse. If there is no pulse, begin chest compressions:
  - Proper hand position is on the lower half of the sternum.
  - **Push hard, push fast. Allow the chest to recoil after each compression.**
  - Rate of chest compressions should be 80 to 100 per minute.
  - For one-rescuer CPR, ratio is 20 compressions to 2 breaths.
  - For two-rescuer CPR, ratio is 15 compressions to 2 breaths.

Risk Factors

- Large, poorly chewed pieces of food.
- Excessive alcohol intake.
- Dentures.
- Children swallowing small objects (toys, beads, marbles, thumbtacks).
- Children eating foods that require adequate chewing (hot dogs, peanuts, popcorn, candy).
- Children running/playing while eating.

The most common cause of sudden cardiac death in adults is ventricular fibrillation (V-fib).

Survival rates from cardiac arrest are highest when BLS is initiated within 4 minutes and advanced cardiac life support (ACLS) is initiated within 8 minutes.

The **tongue** is the most common cause of airway obstruction in the unconscious victim.

Foreign body airway obstruction should be considered in any victim who suddenly becomes cyanotic and stops breathing, especially children.

HIGH-YIELD FACTS

Resuscitation

In adults, poorly chewed meat is the most common cause of foreign body obstruction.

The **Heimlich maneuver** is the recommended method of expelling a foreign object from the airway.

A **finger sweep** should be attempted only in an unconscious victim, and never attempted in a seizure patient.

ACLS is a continuum of BLS.

Remember your **ABC**s.
- **A**irway with C-spine control
- **B**reathing
- **C**irculation
Don't forget to **D**efibrillate.

Management of Partial Airway Obstruction

Do not interfere with any choking victim who is able to cough or speak. Coughing is the most effective way to clear a foreign body from the airway, and the ability to speak indicates that adequate ventilation is still occurring.

Signs of Complete Airway Obstruction

- High-pitched, stridorous sounds during inhalation
- Weak and ineffective coughing
- Respiratory distress
- Inability to speak
- Cyanosis

Heimlich Maneuver

In a Standing (Conscious) Victim
- Stand behind victim and wrap arms around waist.
- Make fist and place thumb of fist slightly above the navel of the victim's abdomen.
- Grasp fist with the other hand and quickly thrust inward and upward into victim's abdomen.
- Repeat until object is dislodged or patient becomes unconscious.

In an Unconscious Victim
- Lay victim supine.
- Straddle victim, place heel of palm just above navel (well below the xiphoid), and deliver quick inward and upward abdominal thrusts (up to five).
- Open the mouth of the unconscious victim and perform a **finger sweep** using a hooking motion of the index finger along the base of the tongue to dislodge the foreign body.
- Reposition the head and attempt rescue breathing.
- Repeat the sequence of the Heimlich maneuver, finger sweep, and rescue breathing attempts until victim resumes breathing or definitive help arrives.

► **ADVANCED CARDIAC LIFE SUPPORT (ACLS)**

Goals

To provide rapid assessment and definitive management of the cardiac arrest situation using cardiac monitoring equipment, advanced airway management, as well as electrical and pharmacologic therapy.

Primary Survey

Focus on the ABCs of CPR and keep in mind defibrillation.

First "A-B-C-D"
- **A**irway—open the airway (maintaining C-spine control).
- **B**reathing—assess breathlessness and provide rescue breathing.

- **C**irculation—give chest compressions (CPR).
- **D**efibrillation—shock ventricular fibrillation and pulseless ventricular tachycardia.

Secondary Survey

Secondary survey of ACLS focuses on the same ABCs in more detail: Establishing a definitive airway, establishing access to the circulation, assessing cardiac rhythms, pharmacologic interventions, etc.

Second "A-B-C-D"
- **A**irway—laryngeal mask airway (LMA) or endotracheal intubation.
- **B**reathing—assess bilateral chest rise and bilateral breath sounds.
- **C**irculation—establish intravenous (IV) access, determine the cardiac rhythm, and give the appropriate medication for that rhythm.
- **D**ifferential diagnosis—why did the arrest occur? Are there any causes that are reversible and have a specific therapy?

Airway

- **Nasal airway**—rubber nasal trumpet inserted into the nostril and passed into the posterior pharynx keeps the tongue from falling back and obstructing the airway.
- **Oral airway**—curved rigid airway, inserted using a tongue blade so that the distal edge prevents the tongue from falling backward. Often *incorrectly* used as a "bite block." Should be used only in unconscious patients with absent gag reflexes (i.e., it will cause gagging if any gag reflex remains).
- **Laryngeal mask airway**—a supraglottic airway management device. Distal tip of LMA cuff presses against upper esophageal sphincter, upper border rests against tongue (see Figure 2-1). Cuff is then inflated, forming a seal over the larynx and permiting positive pressure ventilation.
- **Endotracheal intubation**—establishes a definitive airway that also protects against aspiration of blood, vomit, and pharyngeal secretions. Several cardiac medications can be given directly through the endotracheal tube (ETT). Usual ETT dose is 2 to 2.5 times the IV dose followed by
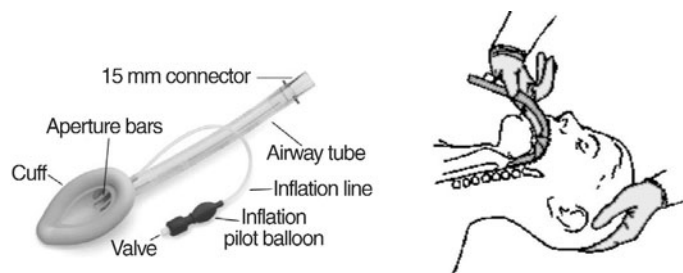
F I G U R E   2 - 1 .   **Laryngeal mask airway (LMA).**

10 mL of normal saline flush and several ventilations by bag-valve ventilation. Intravenous and intraosseous routes are preferred for administration of resuscitation medications.

Breathing

- Assess the status of ventilations after intubation (listen for equal breath sounds over both lung fields and make sure there are no sounds of gastric insufflation) and adjust the tube as necessary.
- Assess the movement of the chest wall with ventilations.
- If in a hospital setting, obtain a stat portable chest x-ray (CXR).
- Confirm ETT placement with an end-tidal CO_2 monitor.
- If there is any doubt of placement, consider extubation and reintubation under direct visualization with a laryngoscope.

Circulation

- Establish IV access (easiest access is usually the antecubital vein).
- *Normal saline* is the fluid of choice in the resuscitation setting.
- Determine cardiac rhythm.

Differential Diagnosis

- Continually ask yourself, "What caused this arrest?"
- Examine the rhythm and consider all the possible causes.
- Treat each of those possible causes that are reversible and/or have a specific therapy.

Classification of Therapeutic Interventions

2000 National Conference on CPR and Emergency Cardiovascular Care (ECC)
- **Class I**—a therapeutic option that is usually indicated, always helpful; considered useful and effective.
- **Class II**—a therapeutic option that is acceptable, is of uncertain efficacy; may be controversial:
  - **Class IIa**—a therapeutic option for which the weight of evidence is in favor of its usefulness and efficacy.
  - **Class IIb**—a therapeutic option that is not well established by evidence but may be helpful and probably not harmful.
- **Class III**—a therapeutic option that is inappropriate, is without specific supporting data, and may be harmful.

► **VENTRICULAR FIBRILLATION OR PULSELESS VENTRICULAR TACHYCARDIA (VF/VT)**

VF and Pulseless VT Algorithm

- It is essential to remember that early defibrillation is the most important therapy for this rhythm. (See Figures 2-2 and 2-3.)

Remember, a "shock" is administered after each medication given, and CPR is continued.
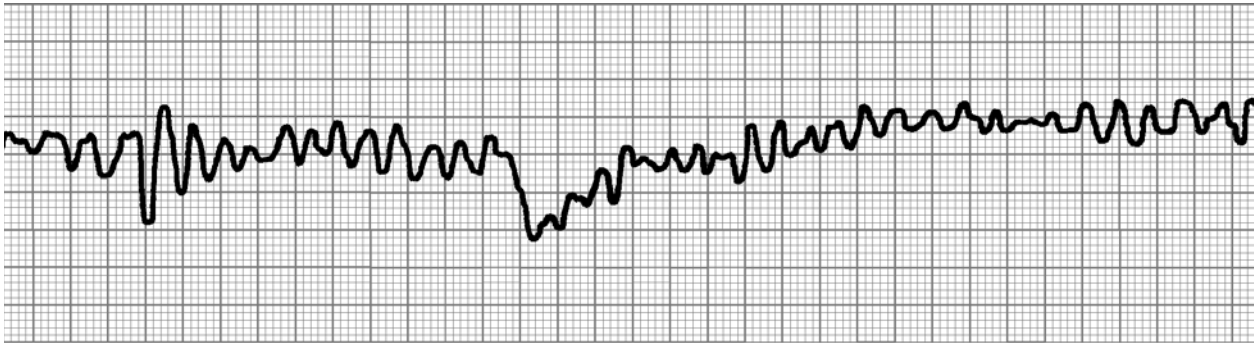
FIGURE 2-2. Ventricular fibrillation.

- Defibrillation should take precedence over establishing IV access, intubation, or the administration of any drug!
- ABCs (always begin with assessing your ABCs!!!).
- Initiate and continue CPR until defibrillator is attached.
- Defibrillate (shock), 360J (biphasic) or 150 to 200J (monophasic).
- Perform five CPR cycles in sequence.
- **Epinephrine** 1 mg IV q 3–5 minutes or **vasopressin** 40 U IV × 1.
- **Amiodarone** 300 mg IV for VF/pulseless VT, then infusion.
- **Lidocaine** 1 to 1.5 mg/kg IV; can repeat once.
- **Magnesium sulfate** 1 to 2 g IV, then infusion.
- **Procainamide** 30 mg/min, max total dose 17 mg/kg.
- **Sodium bicarbonate** 1 mg/kg IV if prolonged arrest, tricyclic antidepressant (TCA) overdose, or known acidosis.
- Consider **calcium** if hyperkalemia suspected.

> Magnesium sulfate is especially useful in *torsades de pointes* and suspected hypomagnesemia; it should be given whenever these etiologies are suspected.

▶ PULSELESS ELECTRICAL ACTIVITY (PEA)

Definition

- Any normally perfusing rhythm in which there is no detectable pulse.
- The differential diagnosis for PEA is key because certain etiologies of PEA have specific treatments and therefore the arrhythmia may be easly reversible.

> PEA = no pulse + electrical activity
> (just like it sounds)
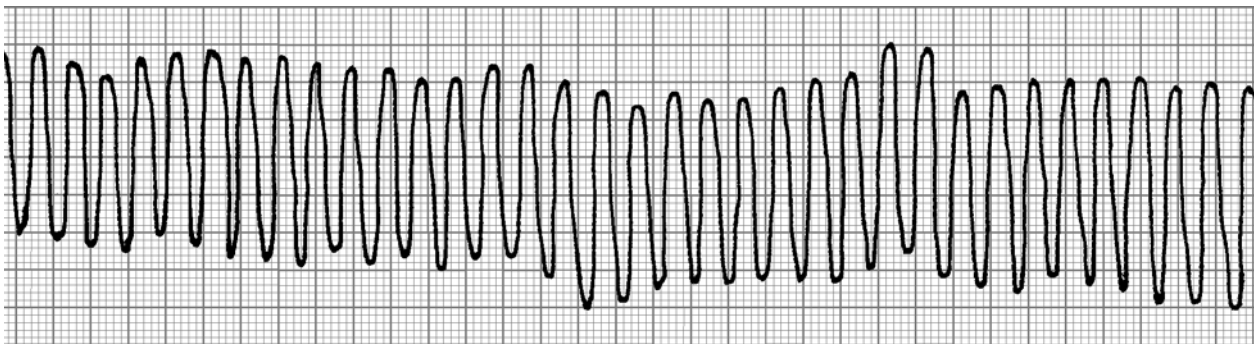
FIGURE 2-3. Ventricular tachycardia.

Causes of PEA:
Remember that cute lab partner in gross anatomy?
HOT MATCH MD
- **H**ypovolemia (volume—normal saline infusion)
- Hyp**o**xia (oxygen, intubation, ventilation)
- Hypo**t**hermia (warmed normal saline infusion)
- **M**assive pulmonary embolism (thrombolytics)
- **A**cidosis (sodium bicarbonate)
- **T**ension pneumothorax (needle decompression)
- **C**ardiac tamponade (pericardiocentesis)
- **H**yperkalemia (calcium, sodium bicarbonate)
- Massive acute **M**yocardial infarction
- **D**rug overdose from TCAs, digoxin, beta blockers, calcium channel blockers

Asystole = no pulse + no electrical activity.
Always confirm flatline in more than one lead.
Never shock asystole.

"Unstable bradycardia" = ↓ LOC, ↓ BP, CHF, ischemic chest pain.

PEA Algorithm

1. ABCs, O$_2$, IV access, cardiac monitor, pulse oximetry, ECG, portable CXR.
2. Confirm pulselessness by Doppler ultrasound (if available).
3. CPR.
4. Consider possible causes of PEA (and specific treatments).
5. Epinephrine 1 mg IV, repeat q 3–5 minutes. One dose of vasopressin can be given to replace first or second dose of epinephrine.
6. If patient is bradycardic, atropine 1 mg IV, repeat q 3 minutes, max total dose 0.03 to 0.04 mg/kg.

▶ **ASYSTOLE**

Definition

- A "flatline" rhythm is indicative of the absence of any electrical activity of the heart.
- The most common cause of a flatline tracing on ECG is a detached lead or malfunctioning equipment, not asystole; therefore, always confirm asystole in more than one lead!
- Asystole is always pulseless.
- Never shock asystole (no matter what you see on TV).

Asystole Algorithm

1. ABCs, O$_2$, IV access, cardiac monitor, pulse oximetry, ECG, portable CXR.
2. CPR.
3. Consider possible causes.
4. Consider immediate transcutaneous pacing (Class IIb).
5. **Epinephrine** 1 mg IV push q 3–5 minutes.
6. **Atropine** 1 mg IV push q 3–5 minutes.
7. Consider **sodium bicarbonate** (1 mEq/kg) if known preexisting bicarbonate-responsive acidosis, if TCA overdose suspected, or if attempting to alkalinize urine for appropriate drug overdoses.
8. Consider termination of efforts.

▶ **BRADYCARDIA**

Definition

- Defined as heart rate < 60 beats per minute.
- It is considered symptomatic or "unstable" when accompanied by hypotension, shock, congestive heart failure (CHF), pulmonary edema, shortness of breath, cyanosis, lethargy, or chest pain.

Bradycardia Algorithm

- ABCs, O$_2$, IV access, cardiac monitor, pulse oximetry, ECG, portable CXR.
- Call for transcutaneous pacer to bedside earlier rather than later.

Treatment of Unstable Bradycardia

1. **Atropine** 0.5 mg IVP q 3–5 minutes, max dose 3 mg (remember that transplanted hearts are *denervated* and will not respond to atropine, go straight to pacing).
2. **Transcutaneous pacing (TCP)** (this is *painful*, use sedation and/or analgesia as needed) verifying electrical capture and mechanical contractions.
3. **Dopamine** 5 to 20 µg/kg/min—titrate to acceptable heart rate (HR) and blood pressure (BP).
4. **Epinephrine** 2 to 10 µg/min—titrate to acceptable HR and BP.
5. Prepare for **transvenous pacing.**

> Atropine may convert Type II second-degree block into complete heart block; thus, TCP is indicated.

▶ TACHYCARDIA

Definition

-   Any rhythm in which the heart is beating faster than 100 times per minute.
-   As with bradycardia, treatment of tachydysrhythmia is largely dictated by the severity of the signs and symptoms.
-   If serious signs and symptoms are present, you should ask whether the tachycardia is causing the symptoms or an underlying symptom is causing the tachycardia.

▶ ATRIAL FIBRILLATION AND ATRIAL FLUTTER

> Tachycardia = HR > 100

See Figures 8-8 and 8-9 (Cardiovascular Emergencies chapter) for ECGs of atrial fibrillation and atrial flutter.

Atrial Fibrillation/Flutter Algorithm

1. ABCs, O_2, IV access, cardiac monitor, pulse oximetry, ECG, portable CXR.
2. Decide if *stable* or *unstable*.
3. If unstable, administer **synchronized cardioversion** (start at 50J for atrial flutter, 100J, 200J, 300J, 360J).
4. If stable, pharmacologic interventions include:
    -   **Diltiazem** 0.25 mg/kg IV slowly over 2 minutes.
    -   Wait 15 minutes.
    -   **Diltiazem** 0.35 mg/kg IV slowly over 2 minutes, then infusion.
    -   Wait 15 minutes.
    -   If no result, consider short-acting beta blocker (cautiously use beta blockers only after enough time has passed since last dose of calcium channel blockers; many prefer to stick with either a calcium channel blocker or a beta blocker to avoid blocking both channels):
        -   **Metoprolol** 5 mg IV q 5 minutes × 3.
        -   **Esmolol** 500 µg/kg IV over 1 minute (loading dose), then 50 to 200 µg/kg/min infusion.
        -   **Atenolol** 2.5 to 5.0 mg IV over 2 minutes.
    -   **Digoxin** 0.5 mg IV or PO × 1.

> Awake patients should be sedated prior to synchronized cardioversion.

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- **Verapamil** 5 to 10 mg IV, wait 30 minutes, may repeat.
- **Procainamide** 20 to 30 mg/min IV, max dose 17 mg/kg.
- **Quinidine** 200 to 400 mg IV or PO.
- Anticoagulation.

PSVT = narrow regular QRS and rate > 160

Avoid *carotid massage* if bruits present.
Avoid *ice bath* if history of myocardial infarction (MI).
Push adenosine *rapidly* with an immediate saline flush.
The biggest mistake when using adenosine is it is *not pushed rapidly enough.*

Adenosine feels like a *mule kick* to the chest — warn patients prior to giving it.

► **PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT)**

Definition

- Heart rate usually > 160 beats per minute.
- Usually demonstrates a narrow regular QRS complex (< 0.10 second) on ECG (see Figure 2-4).

PSVT Algorithm

1. ABCs, O₂, IV access, cardiac monitor, pulse oximetry, ECG, portable CXR.
2. Decide if *stable* or *unstable*.
3. If unstable, synchronized cardioversion (50J, 100J, 200J, 300J, then 360J).
4. If stable, proceed as follows:
   - **Vagal maneuvers:** Valsalva; carotid massage (listen first for bruits); ice water bath (not if history of MI).
   - **Adenosine** 6 mg *rapid* IV push.
   - Important: Wait 1 to 2 minutes.
   - **Adenosine** 12 mg rapid IV push, repeat after 1 to 2 minutes.
5. If tachycardia persists, analyze QRS complex on ECG and rhythm strip:
   - If **wide complex tachycardia** (and patient remains stable), treat as ventricular tachycardia.
   - If **narrow complex tachycardia** (and patient remains stable), several agents can be used for cardioversion:
   - Diltiazem 20 mg or verapamil 3 to 10 mg over 2 minutes; can repeat in 30 minutes. Hypotension from these calcium channel blockers can be treated with IV calcium chloride 35 mg.
   - Esmolol followed by infusion, or metoprolol 5 to 10 mg IV.
   - Digoxin 5 mg IV.
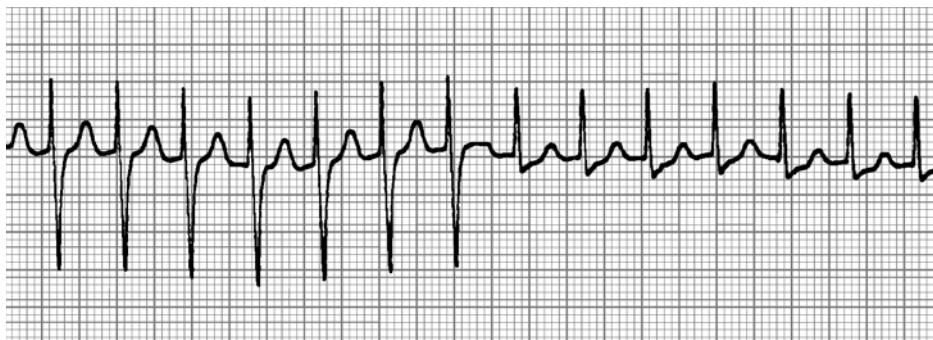
FIGURE 2-4. Paroxysmal ventricular tachycardia.

6. If at any time the patient becomes unstable, proceed directly to synchronized cardioversion.

Definition

- Rate > **100 beats per minute.**
- **Wide QRS** complexes that are **regular.**
- Constant QRS axis.
- Readily converts to ventricular fibrillation (BAD!).
- If pulseless VT, proceed immediately with defibrillation (remember VF/pulseless VT algorithm).

VT = wide QRS + rate > 100
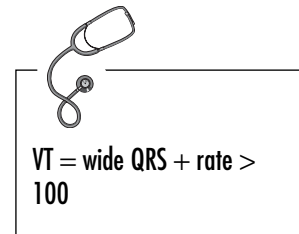
VT with Pulse Algorithm

1. ABCs, O$_2$, IV access, cardiac monitor, pulse oximetry, ECG, portable CXR.
2. Decide if stable or unstable.
3. If unstable, immediate synchronized cardioversion (100J, 200J, 300J, 360J).
4. If stable:
   - **Amiodarone** 150 mg IV bolus over 10 minutes or **lidocaine** 0.5 to 0.75 mg/kg IVP q 3–5 minutes (total max dose 3 mg/kg).
   - Procainamide 20 to 30 mg/min (max dose 17 mg/kg) may be used in patients with normal ejection fraction only.
   - Synchronized cardioversion (100J, 200J, 300J, 360J).

Definitions

- **Hypotension** is generally defined as an SBP < 100 and a DBP < 60.
- **Shock** is defined as inadequate tissue perfusion.

Shock = inadequate tissue perfusion

These topics will be covered elsewhere, but they are included in the ACLS course and will therefore be discussed (briefly).
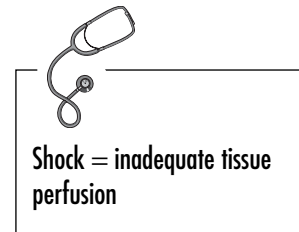
Causes

In order to rapidly assess a hypotensive patient, it is often helpful to divide the causes of shock into three etiologies.

Rate Problems
- Bradyarrhythmias:
  - Sinus bradycardia
  - Second- and third-degree heart block
  - Pacemaker failures
- Tachyarrhythmias:
  - Sinus tachycardia

- Atrial flutter
- Atrial fibrillation
- PSVT
- Ventricular tachycardia

Pump Problems
- Primary pump failure:
  - Myocardial infarction
  - Myocarditis
  - Cardiomyopathies
  - Ruptured chordae or papillary muscle damage
  - Aortic or mitral regurgitation/failure
  - Septal defect/damage
- Secondary pump failure:
  - Cardiac tamponade
  - Pulmonary embolism
  - Superior vena cava syndrome
  - Cardiodepressant drugs

Volume Problems
- Volume loss:
  - Blood loss
  - Gastrointestinal (GI) losses (vomiting, diarrhea, etc.)
  - Urine output
  - Third-space losses
- Decreased vascular resistance:
  - Central nervous system (CNS) or spinal injury
  - Sepsis
  - Vasodilatory drugs
  - Adrenal insufficiency

► **ADVANCED AIRWAY MANAGEMENT**

Rapid Sequence Intubation Algorithm

Prepare = equipment
Pretreat = drugs
Position = sniffing position
Preoxygenate = pulse oximetry of 100%
Pressure = Sellick maneuver
Paralyze = drugs
Placement of the tube
Position of tube = confirm by two methods

1. Prepare the necessary equipment:
   - IV access, cardiac monitor, pulse oximetry.
   - Bag-valve mask (Ambu bag).
   - Suction equipment (make sure it works!).
   - Laryngoscope with blade (check lightbulb!).
   - ETT (7.0 adult female/8.0 adult male).
   - Insert ETT stylet (if desired).
   - Medications.
   - Prepare adjunct airway (laryngeal mask airway, cricothyroidotomy tray, etc.) in case ETT is unsuccessful.
2. Pretreat:
   - Lidocaine for head injury patients (decreases intracranial pressure).
   - Atropine for children (prevents bradycardia).
3. Position the patient:
   - Raise bed to height appropriate for intubation.
   - Place head in "sniffing position" with neck extended (except when C-spine injury suspected).

4. Preoxygenate the patient:
   - Bag-valve mask with 100% oxygen.
   - Pulse oximetry should read 100%.
   - Hyperventilate patient to accomplish nitrogen washout.
5. Pressure on cricothyroid cartilage:
   - Sellick maneuver compresses esophagus to limit risk of aspiration.
6. Sedation: Many agents are available including:
   - Etomidate (does not cause hypotension, quite safe).
   - Thiopental (barbiturate, can cause hypotension).
   - Midazolam (benzodiazepine, quite safe).
7. Paralyze the patient:
   - Succinylcholine (1.5 mg/kg IVP) onset 45 to 60 seconds, duration 5 to 10 minutes. Do not use in hyperkalemia, crush injuries, or history of neuromuscular diseases.
   - Vecuronium (0.1 mg/kg IVP) onset 2 to 3 minutes, duration 25 to 30 minutes.
8. Place the tube:
   - Open the mouth and displace the jaw inferiorly.
   - Holding the laryngoscope in the left hand, insert the blade along the right side of the tongue, and the tongue is swept toward the left.
   - If using a curved (Macintosh) blade, the tip should be inserted to the vallecula (the space between the base of the tongue and the epiglottis).
   - If using a straight (Miller) blade, the tip is inserted beneath the epiglottis.
   - The laryngoscope is the used to *lift* the tongue, soft tissues, and epiglottis to reveal the vocal cords (remember, it is a *lifting* motion, not a *rocking* motion).
   - Upon direct visualization of the cords, the tube is directed through the cords, the stylet (if used) is removed, the tube is connected to an oxygen source, and it is secured after proper placement is confirmed.
9. Confirm position of the tube by two methods:
   - Bilateral breath sounds (check both apical lung fields!)
   - Absence of breath sounds in abdomen
   - End-tidal carbon dioxide detection
   - Portable CXR
   - Condensation in ETT corresponding to bag-valve mask breaths

▶ **NEEDLE CRICOTHYROIDOTOMY**

Definition

- Temporizing measure to provide oxygen to a patient emergently after a failed or impossible endotracheal intubation.
- The procedure entails inserting a large-bore angiocatheter through the cricothyroid membrane (see Figure 2-5) and providing oxygen through the catheter.
- It is important to note that while oxygen delivery can be established with this procedure, adequate elimination of carbon dioxide is not achieved.

Thyroid Cartilage

Cricothyroid Membrane

Cricoid Cartilage

Hyoid Bone

Thyroid Gland

Clavicle

A   Manubrium   Trachea
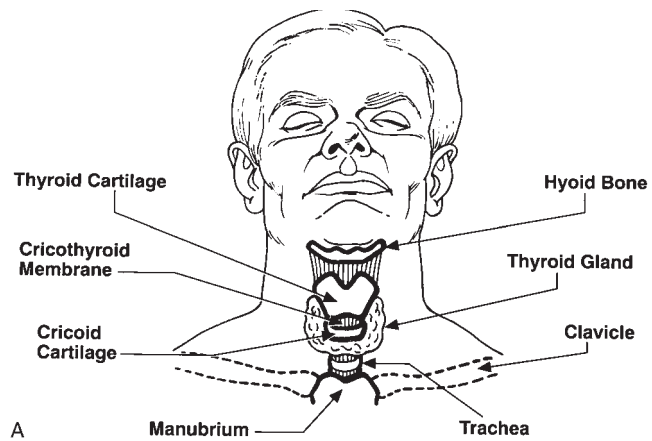
F I G U R E   2 - 5 .   Anatomical landmarks for needle and surgical cricothyroidotomy.

Needle Cricothyroidotomy Algorithm

1. Prep area with alcohol and povidone–iodine (Betadine).
2. Hyperextend neck (if no C-spine injury is suspected).
3. Identify cricothyroid membrane.
4. Insert 14G angiocatheter on a syringe at a 45° angle (toward feet) through cricothyroid membrane.
5. Advance with negative pressure until air is freely aspirated.
6. Remove needle and advance angiocatheter.
7. Use a syringe to verify placement in trachea.
8. Attach adapter from ETT and ventilate with bag-valve delivery system.

▶ **SURGICAL CRICOTHYROIDOTOMY**

Definition

- Allows for rapid establishment of an airway when endotracheal intubation has failed or is impossible (e.g., severe facial trauma, burns, impacted obstruction).
- Permits both oxygen delivery and ventilation for elimination of carbon dioxide.

Indications and Contraindications

Indications
- Inability to obtain an airway by orotracheal or nasotracheal intubation due to anatomic distortion, massive hemorrhage, or severe aspiration.
- Presence of severe maxillofacial trauma renders other airways impossible.
- Upper airway obstruction due to foreign body.
- Massive upper airway edema.

Resuscitation

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Contraindications
- Age under 5 to 10 years, depending on child's size.
- Significant injury to larynx or cricoid.
- Tracheal transection.
- Expanding hematoma over the cricothyroid membrane.
- Preexisting laryngeal pathology.

Procedure

1. Prep area with alcohol and povidone–iodine (Betadine).
2. Hyperextend neck (if no C-spine injury is suspected).
3. Identify cricothyroid membrane.
4. Holding a #10 scalpel at the hub of the blade, make a horizontal stab through the skin and cricoid membrane (hold at the hub to ensure the stab incision does not go too deep!).
5. Enlarge the stab incision to approximately 1.5 to 2.0 cm with a horizontal motion of the scalpel.
6. Keeping the scalpel in place, insert a tracheal hook next to the scalpel and retract the larynx.
7. Remove the scalpel.
8. Using the scalpel handle, or a dilator, dilate the surgical opening.
9. Place a tracheostomy tube into the opening, secure the airway, and ventilate with bag-valve oxygen delivery system.

Complications

Esophageal perforation, hemorrhage, subcutaneous emphysema, vocal cord injury.

HIGH-YIELD FACTS IN

Diagnostics

- Laboratory examinations are perhaps the most variable aspect of emergency medicine.
- The labs ordered for a patient differ greatly from one physician to another.
- In general, in the emergency department (ED), a test should not be ordered unless it will change your management of a patient.

Electrolytes

CALCIUM

- Over 99% of the calcium in the body is found in the bony skeleton.
- Remaining calcium is either protein bound (albumin, etc.), polyvalent (bound to phosphate, etc.), or ionized.
- Parathyroid hormone and calcitonin are counterregulatory hormones that respond to levels of ionized Ca.
- Vitamin D metabolites (calcitriol) are synthesized in liver/kidney in response to decreased calcium levels.

IONIZED CALCIUM

- Is most important physiologically.
- Hypoalbuminemia will decrease total calcium, but ionized calcium is unaffected.
- Acid–base disorders (alkalosis) will decrease ionized calcium.

Hypercalcemia

SIGNS AND SYMPTOMS

- Neurologic: Weakness, fatigue, ataxia, altered mental status, seizures (rare).
- Gastrointestinal (GI): Decreased motility (constipation), vomiting.
- Renal: Osmotic diuresis (polyuria), polydipsia, nephrolithiasis, potassium/magnesium losses.
- Cardiovascular: Bradycardia, heart blocks, shortened QT interval, potentiates digoxin toxicity.

TREATMENT

Aimed at correcting dehydration and promoting urinary excretion of calcium:
- Rehydrate with large amounts of intravenous (IV) saline until volume status is restored.
- Furosemide to promote diuresis once volume status is restored is controversial.
- Electrolytes must be monitored carefully (hypokalemia/hypomagnesemia).
- Dialysis in the setting of renal failure.

Signs of hypercalcemia:
- Bones (bony pain)
- Stones (kidney stones)
- Groans (abdominal pain)
- Psychiatric overtones (change in mental status)

Causes of hypercalcemia:
SPLITTing HEADACHE
Sarcoid
Pheochromocytoma
Lithium
Immobilization
Tuberculosis
Thiazides

1° **H**yperparathyroidism
Estrogens
Vitamin **A** excess
Vitamin **D** excess
Adrenal insufficiency
Cancers (lung, breast, kidney, leukemia, myeloma)
Hyperthyroidism
Estrogen antagonists (therapy for breast cancer)

HIGH-YIELD FACTS

Diagnostics

Chvostek's sign: Tapping facial nerve below zygomatic arch induces tetany of facial muscles.

Trousseau's sign: Inflating blood pressure (BP) cuff above systolic BP for 3 minutes induces carpal spasm.

Interpret potassium levels in the context of serum pH: Acidosis causes potassium shift into serum; alkalosis causes potassium shift into cells.

Hypocalcemia

CAUSES

- Paget's milk alkali immobilization.
- Hypoalbuminemia (cirrhosis, nephrotic syndrome); does not lower the ionized Ca^{2+}.
- Hypoparathyroidism (intrinsic or post-thyroid surgery), malnutrition (rickets/osteomalacia).
- Pancreatitis (saponification).
- Drugs (cimetidine).

PATHOPHYSIOLOGY

Neuronal membranes become more excitable secondary to increased sodium permeability.

SIGNS AND SYMPTOMS

- Perioral and digital paresthesias.
- Decreased myocardial contractility (relaxation is inhibited) can predispose to congestive heart failure (CHF).

DIAGNOSIS

Electrocardiogram (ECG) characteristically shows prolonged QT intervals.

TREATMENT

Supplement calcium:
- Asymptomatic patients should be given oral calcium (with or without vitamin D).
- Symptomatic patients should be treated with IV calcium (Ca gluconate or Ca chloride).

Potassium

- Potassium is the intracellular cation (98% of total body potassium is intracellular).
- Potassium is excreted primarily in urine (small amount in feces, sweat).
- Renin–angiotensin–aldosterone axis regulates potassium secretion in distal tubules.

Hypokalemia

CAUSES AND PATHOPHYSIOLOGY

Three mechanisms for decreased potassium:
- Intracellular shifts (alkalotic states, administration of insulin and glucose).
- Reduced intake (malnutrition).
- Increased losses (renal—diuretics, hyperaldosteronism; GI—vomiting, diarrhea, fistulas).

SIGNS AND SYMPTOMS

- Muscle weakness
- Hyporeflexia

- Intestinal ileus
- Respiratory paralysis
- Nephrogenic diabetes insipidus
- Dehydration

DIAGNOSIS

Hypokalemia results in hyperpolarization of cell membrane potential leading to:

- Cardiac abnormalities on ECG include flattened T waves, U waves (see Figure 3-1), low-voltage QRS, prolonged QT and PR, and premature ventricular contractions (PVCs).
- Hypokalemia potentiates digitalis and increases likelihood of digitalis toxicity (arrhythmias and atrioventricular blocks).

TREATMENT

Supplement potassium:

- Mild hypokalemia: Potassium-rich foods (bananas, avocados, passion fruit, orange juice) or oral KCl supplements.
- Severe hypokalemia: Treat with IV KCl (10 mEq/hr, max 40 mEq/hr).

Hyperkalemia

CAUSES

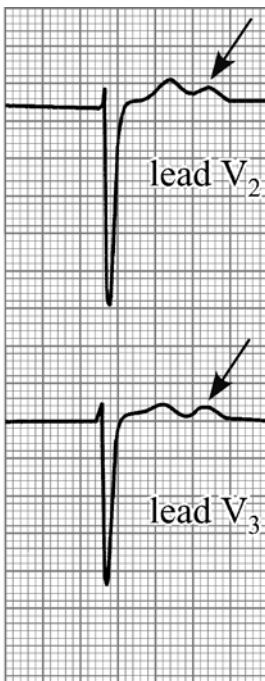- Lab error: Hemolysis, thrombocytosis, leukocytosis, polycythemia ("pseudohyperkalemia").
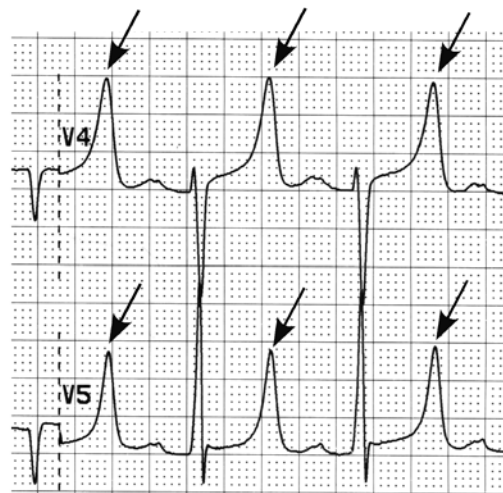
F I G U R E   3 - 1 .   U waves (arrows) of hypokalemia.

F I G U R E   3 - 2 .   Peaked T waves (arrows) of hyperkalemia.

- Decreased excretion: Oliguric renal failure, angiotensin-converting enzyme inhibitors, K-sparing diuretics, type IV renal tubular acidosis.
- Increased release: Metabolic acidosis, trauma, burns, rhabdomyolysis, tumor lysis, succinylcholine.
- Increased intake: Iatrogenic, dietary, salt substitutes.

SIGNS AND SYMPTOMS

- GI: Nausea, vomiting, diarrhea.
- Neurologic: Muscle cramps, weakness, paresthesias, paralysis, areflexia, tetany, focal neurologic deficits, confusion.
- Respiratory insufficiency.
- Cardiac arrest.

DIAGNOSIS

ECG Findings
- At K = 5.0 to 6.0 mEq/L, rapid repolarization causes peaked T waves (most prominent in precordium) (see Figure 3-2).
- At K = 6.0 to 6.5 mEq/L, decrease in conduction causes prolonged PR and QT intervals.
- At K = 6.5 to 7.0 mEq/L, P waves are diminished and ST segment may be depressed.
- At K = 7.0 to 8.0 mEq/L, P waves disappear, QRS widens, and irregular idioventricular rhythm appears.
- At K = 8.0 to 10.0 mEq/L, QRS merges with T wave to produce classic sine wave.
- At K = 10.0 to 12.0 mEq/L, ventricular fibrillation and diastolic arrest occur.

TREATMENT

Patients on digitalis should be given calcium only in emergencies; calcium in the setting of digitalis toxicity may induce tetany and "stone heart."

- Calcium gluconate: Stabilizes cardiac membrane, onset of action 1 to 3 minutes.
- Sodium bicarbonate: Alkalosis shifts potassium into cells, onset 5 to 10 minutes.
- Insulin and glucose: Insulin drives potassium and glucose into cells, onset 30 minutes.
- Lasix: Promotes renal excretion of potassium, onset with diuresis.
- Kayexalate: Cation exchange resin (potassium for sodium in GI tract), onset 1 to 2 hours.
- Dialysis: Peritoneal or hemodialysis removes potassium at time of dialysis.
- Dialysis is indicated for patients in renal failure with hyperkalemia that does not respond to above.

Sodium

- Ninety-eight percent of total body sodium is in extracellular fluid.
- Sodium is the major contributor to serum osmolarity.
- $S_{osm} = 2(Na) + (glucose/18) + (BUN/2.8)$.
- Balance between sodium, water, and osmolarity is regulated by kidney (excretion and reabsorption of sodium and water), posterior pituitary (secretion of antidiuretic hormone), and the hypothalamus (thirst center).

- Understanding the relationship between osmolarity (tonicity) and volume is essential:
  - Volume status is a clinical diagnosis.
  - Tonicity is a laboratory diagnosis.
- Understanding where you are in terms of volume and tonicity allows you to guide therapy appropriately (see Table 3-1).

Hyponatremia

CAUSES

Hyponatremia is subdivided into three categories based on the serum osmolarity:

1. **Hypotonic hyponatremia** is further subdivided into three categories:
   - Isovolemic/hypotonic hyponatremia: Renal failure, syndrome of inappropriate antidiuretic hormone (SIADH), glucocorticoid deficiency (hypopituitarism), hypothyroidism, and medications.
   - Hypovolemic/hypotonic hyponatremia:
     - Renal losses (diuretics, partial urinary tract obstruction, salt-wasting nephropathies).
     - Extrarenal losses (vomiting, diarrhea, extensive burns, third spacing, pancreatitis, peritonitis).
   - Hypervolemic/hypotonic hyponatremia: CHF, nephrotic syndrome, cirrhosis.
2. **Isotonic hyponatremia** (normal serum osmolarity):
   - Pseudohyponatremia (discussed later).
   - Isotonic infusions (glucose, mannitol).
3. **Hypertonic hyponatremia** (increased serum osmolarity):
   - Hyperglycemia: Each 100 mL/dL increase in serum glucose above normal decreases plasma sodium concentration by 1.6 mEq/L.
   - Hypertonic infusions: Mannitol, glucose.

PATHOPHYSIOLOGY

Severity is dependent on both the magnitude and rapidity of the fall in serum sodium:
- Initial response to low serum sodium is to shift water across blood–brain barrier into the central nervous system (CNS).

The most common cause of hyponatremia is hemodilution.

Calculation of sodium deficit:
(Wt in kg) (0.6) (140 − measured [Na⁺])
= sodium deficit in mEq

Pseudohyponatremia is present when glucose levels are elevated. Correct 1.6 mEq/L for every 100 mg/dL over 200.

TABLE 3-1. **Sodium Balance: Volume vs. Tonicity**

VOLUME	TONICITY		
	Hypertonic	**Isotonic**	**Hypotonic**
Hypervolemic	Iatrogenic	Early CHF, cirrhosis, nephrotic syndrome (no ADH stimulus)	Late CHF, cirrhosis, nephrotic syndrome (with ADH stimulus)
Euvolemic	Early stages of "dehydration"	Normal	Psychogenic polydipsia, SIADH "reset osmostat"
Hypovolemic	Late stages of "dehydration" (H₂O loss > salt loss)	Acute volume loss (i.e., burns, bleeding) (no ADH stimulus)	Chronic volume loss (with ADH stimulus) diuretics, Addison's

Pseudohyponatremia is caused by:
- Hyperglycemia
- Hyperlipidemia
- Hyperproteinemia

The serum osmolarity will be normal or high in these cases.

Correcting the sodium too quickly can result in central pontine myelinolysis, seizures, and cerebral edema.

Water deficit

$$= TBW \left(\frac{1 - NA^+_m}{140 \text{ (Not desired)}} \right)$$

The most common cause of hypernatremia is decrease in total body water (i.e., dehydration).

- CNS responds by shifting sodium and other osmotic agents from brain into cerebrospinal fluid to systemic circulation.
- If hyponatremia is corrected too quickly, CNS loses its ability to retain water (loss of osmotic agents).
- Brain rapidly becomes dehydrated, leading to osmotic demyelination syndrome, also known as central pontine myelinolysis (seen days after therapy; presents with bulbar dysfunction, quadriparesis, delirium, death).

SIGNS AND SYMPTOMS

- Early: Nonspecific headache, vomiting.
- Late: Confusion, seizures, coma, bradycardia, or respiratory arrest.

TREATMENT

- In symptomatic (CNS) patients with severe (< 120 mEq/L) hyponatremia, consider hypertonic (3%) saline at 25 to 100 mL/hr. Usually, normal saline is good enough as it is "hypertonic" relative to the patient's current state.
- Furosemide should be given with 3% saline to blunt ADH stimulus (maintain negative H_2O balance).
- Patients with acute hyponatremia should be corrected no faster than 1.0 mEq/L/hr.
- Patients with chronic hyponatremia should be corrected no faster than 0.5 mEq/L/hr.
- Electrolytes must be checked every 1 to 2 hours; never correct above Na = 120 mEq/L.

Hypernatremia

CAUSES

- GI losses: Vomiting, diarrhea, decreased thirst.
- Renal losses: Diabetes insipidus, osmotic diuretics, adrenal/renal disease.
- Insensible losses: Respiratory, skin, hyperthermia.
- Inability to respond to thirst (due to lack of access).

SIGNS AND SYMPTOMS

- Early signs (Na > 158 mEq/L) include irritability, lethargy, anorexia, and vomiting.
- As serum osmolarity rises (350 to 400 mOsm/L), begin to see ataxia, tremulousness, hypertonicity, and spasms.
- At serum osmolarity > 430, death usually ensues.

TREATMENT

- First step is to address fluid status: Hydrate with normal saline (NS) until volume is restored. (Remember, NS will actually be "hypotonic" with hypernatremia.)
- Once perfusion is established, hydrate with hypotonic (0.45%) saline (rarely indicated in the ED).
- Monitor urine output (0.5 mL/kg/hr) and check electrolytes every few hours. If adequate urine output cannot be achieved, switch to 1/2 NS for every liter of water and administer diuretic. This serves to unload

excess sodium. For every liter of water lost, the sodium rises by 3 to 5 mEq/L.
- Target should be to correct sodium over 48 to 72 hours (max rate of 0.5 mEq/L/hr).

SIADH

PATHOPHYSIOLOGY

- Thirst mechanism and ADH work together to control intake/excretion of water.
- Both mechanisms are usually impaired before SIADH becomes clinically apparent (excess ADH → water retention, thirst → increased fluid intake).

CAUSES

CNS
- Head trauma, tumors, abscesses, meningitis, subarachnoid hemorrhage.

Tumors
- Lung, pancreas, ovaries, lymphoma, thymoma.

Pulmonary
- Pneumonia, chronic obstructive pulmonary disease (COPD), tuberculosis, cystic fibrosis, abscess.

Drugs
- Opiates, nonsteroidal anti-inflammatory drugs, monoamine oxidase inhibitors, tricyclic antidepressants.

Other
- Hypothyroidism, adrenal insufficiency, porphyria, idiopathic.

DIAGNOSIS

SIADH is a diagnosis of exclusion (must rule out other causes of hyponatremia):
- Serum Na < 135 and serum osmolarity < 280.
- Urine is not maximally diluted (urine osmolarity > 100).
- No evidence of dehydration, edema, hypotension.
- No evidence of renal, cardiac, thyroid, or adrenal dysfunction.

TREATMENT

- Treatment generally consists of fluid restriction. (Remember, clinical manifestations of SIADH usually become apparent only when the thirst mechanism leads to increased fluid intake!)
- Hypertonic (3%) saline is appropriate only for patients with neurologic symptoms of hyponatremia.

Acid–Base Disorders

Assess the acid–base disorder step by step (Figure 3-3):
- Is the primary disorder an acidosis (pH < 7.40) or alkalosis (pH > 7.40)?
- Is the disorder respiratory (pH and P_{CO_2} move in opposite directions)?
- Is the disorder metabolic (pH and P_{CO_2} move in same direction)?
- Is the disorder a simple or mixed disorder?

Normally, a 2% rise in osmolarity triggers the thirst response in the brain.

Diabetes Insipidus
Central: Hypothalamus does not make ADH.
Nephrogenic: Kidneys do not *respond* to ADH.

Neither respiratory (12 hours) nor metabolic compensation (24 to 48 hours) will return pH completely to normal.

Causes of elevated anion gap metabolic acidosis:
MUDPILES
Methanol, metabolism (inborn errors)
Uremia
Diabetic ketoacidosis
Paraldehyde
Iron, isoniazid
Lactic acidosis
Ethylene glycol
Salicylates, strychnine

$AG = [Na^+(Cl^- + HCO_3)] > 12$

metabolic acidosis

Non AG

$HCO_3 < 24$

compensation → $pCO_2 = 1.5 (HCO_3) + 8 +/- 2$
Max: $pCO_2 = 10$ mmHg

Acute: $\Delta pH/\Delta pCO_2 \le .8$ — compensation → Max: $HCO_3 = 30$ mEq/L

respiratory acidosis

$pCO_2 \ge 45$

Chronic: $\Delta pH/\Delta pCO_2 \le .3$ — compensation → Max: $HCO_3 = 45$ mEq/L

acidemic

pH < 7.38

normal pH — examine ABG and serum chemistry for abnormal values

alkalemic

pH > 7.42

metabolic alkalosis

$HCO_3 \ge 30$

compensation → $pCO_2 = 0.9 (HCO_3) + 9$
Max: $pCO_2 = 55$ mmHg

Acute: $\Delta pCO_2 \ge 0.8$ — compensation → HCO_3 decreases by 2-4 mEq/L
Max: $HCO_3 = 15$ mEq/L

respiratory alkalosis

$pCO_2 \le 35$

Chronic: $\Delta pCO_2 \ge 0.3$ — compensation → for each mmHg decrease in pCO_2, get 0.5 mEq/L decrease in HCO_3
Max: $HCO_3 = 15$ mEq/L

If compensation is not appropriate, then there is a concomitant acid-base disturbance
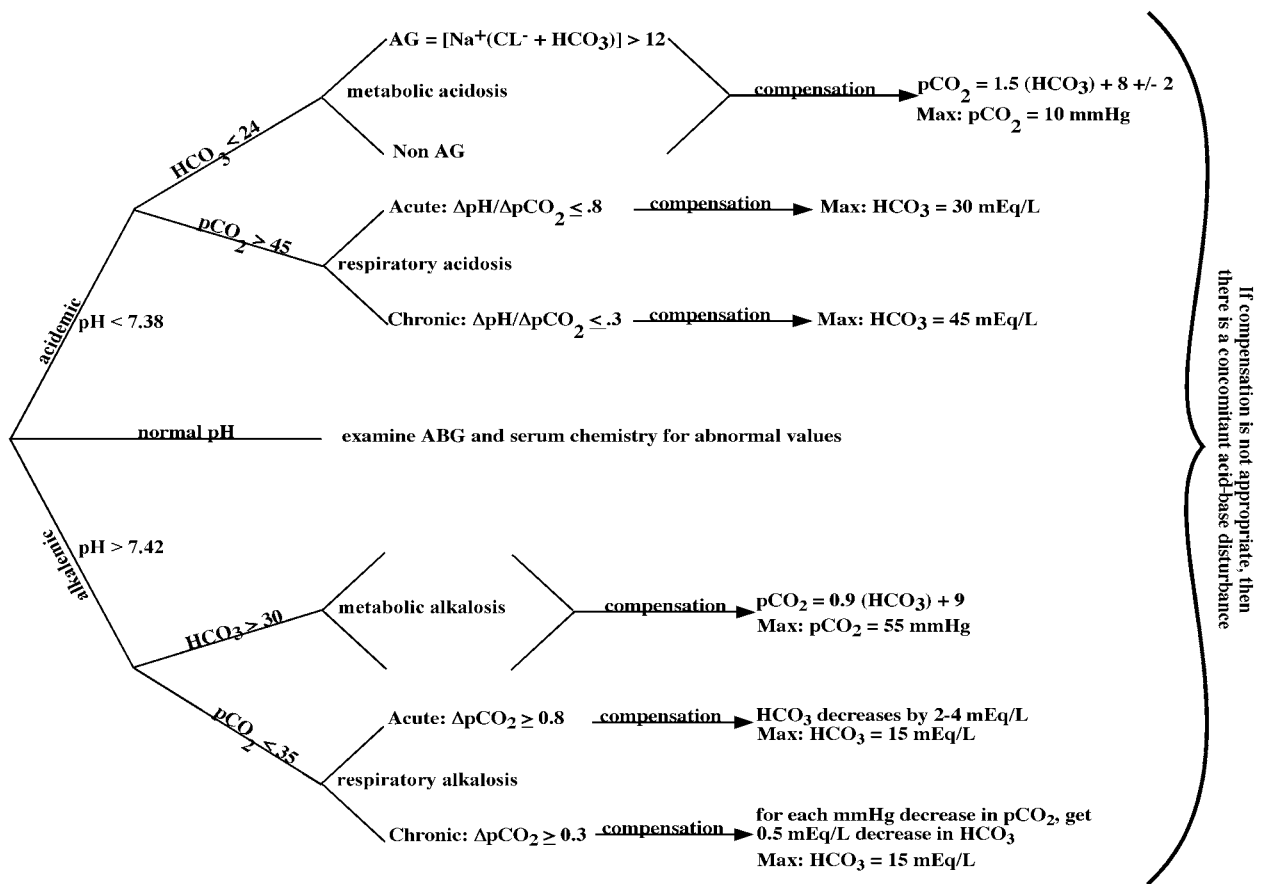
FIGURE 3-3. Acid–base algorithm.

(Reproduced, with permission, from Stead L. *BRS Emergency Medicine.* Philadelphia, PA: Lippincott Williams & Wilkins, 2000.)

Use the following general rules of thumb for acute disorders:
- Metabolic acidosis: P_{CO_2} drops ~1.5 (drop in HCO_3)
- Metabolic alkalosis: P_{CO_2} rises ~1.0 (rise in HCO_3)
- Respiratory acidosis: HCO_3 rises ~0.1 (rise in P_{CO_2})
- Respiratory alkalosis: HCO_3 drops ~0.3 (drop in P_{CO_2})

Compensation beyond above parameters suggests mixed disorder.

METABOLIC ACIDOSIS

Two varieties: Anion gap and nonanion gap.

Calculating the Anion Gap
$AG = Na - [Cl + HCO_3]$
Normal $AG \le 12$ to 14

METABOLIC ALKALOSIS

Two mechanisms:
- Loss of H^+:
  - Renal: Mineralocorticoid excess, diuretics, potassium-losing nephropathy.
  - GI: Vomiting, gastric drainage, villous adenoma of colon.
- Gain HCO_3: Milk–alkali syndrome, exogenous $NaHCO_3$.

Causes of normal anion gap metabolic acidosis:
HARD UP
Hyperparathyroidism
Adrenal insufficiency, anhydrase (carbonic anhydrase) inhibitors
Renal tubular acidosis
Diarrhea

Ureteroenteric fistula
Pancreatic fistulas

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RESPIRATORY ACIDOSIS

Hypercapnia secondary to one of two mechanisms:
- Hypoventilation (brain stem injury, neuromuscular disease, ventilator malfunction).
- Ventilation–perfusion (V/Q) mismatch (COPD, pneumonia, pulmonary embolism, foreign body, pulmonary edema).

RESPIRATORY ALKALOSIS

- Hyperventilation secondary to 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.

Vitamin Disorders

WATER-SOLUBLE VITAMINS

Thiamine

- Deficiency primarily seen in chronic alcoholics.
- "Dry beriberi"—sensorimotor neuropathy.
- "Wet beriberi"—high-output cardiac failure.
- Wernicke's encephalopathy—confusion, ataxia, altered mental status.
- Korsakoff's syndrome—impaired memory with intact cognition, psychosis.

Riboflavin (B_2)

- Cofactor for oxidation/reduction reactions.
- Deficiency leads to angular stomatitis and cheilosis.

Niacin (B_3)

- Cofactor for oxidation/reduction reactions.
- Deficiency leads to pellagra (3 Ds—diarrhea, dementia, dermatitis).

Pyridoxine (B_6)

- Deficiency leads to convulsions.
- Excess leads to neuropathy (usually sensory).

Cobalamin (B_{12})

Produced by microorganisms, binds to intrinsic factor in stomach, absorbed in ileum.

Causes of Deficiency
- Pernicious anemia—macrocytic anemia with mean corpuscular volume > 100 and hypersegmented neutrophils. Antibodies to intrinsic factor prevent binding and absorption of B_{12}.
- Resection of distal ileum.
- Tropical sprue.
- Crohn's disease.

Cause of respiratory alkalosis: **MIS[HAP]³S**
- **M**echanical overventilation
- **I**ncreased ICP
- **S**epsis
- **H**ypoxemia, **H**yperpyrexia, **H**eart failure
- **A**nxiety, **A**sthma, **A**scites
- **P**regnancy, **P**ain, **P**neumonia
- **S**alicylates

Thiamine should be given with dextrose in alcoholics to avoid precipitating Wernicke's encephalopathy.

Three doctors drank a nice Pellegrino.

Result of Deficiency

- Neuropathy typically involving dorsal columns (position and vibration sense).

Folic Acid

- Required for synthesis of nitrogen bases in DNA/RNA.
- Deficiency causes macrocytic anemia without neurologic symptoms.

Vitamin C

- Required for hydroxylation of proline and lysine (cross-linking) in collagen synthesis.
- Deficiency causes scurvy (bleeding gums, poor wound healing, hyperkeratosis).

FAT-SOLUBLE VITAMINS

Vitamin A

- Deficiency causes night blindness and dry skin.
- Excess causes neurologic symptoms, osteolysis, yellow skin, and alopecia.

Vitamin D

- Absorbed by GI (D_2) or synthesized in skin (D_3).
- Converted by liver/kidney to active form 1,25 (OH) D_3.
- Deficiency causes rickets (children), osteomalacia (adults), symptoms of hypocalcemia.
- Excess causes symptoms of hypercalcemia.

Vitamin K

- Synthesized by intestinal flora, cofactor in synthesis of clotting factors.
- Deficiency leads to disorders of bleeding and hemostasis.
- Excess can cause hemolytic anemia and hepatotoxicity.

Vitamin E

- Provides defense against lipid peroxidation.
- Protects membranes of intracellular organelles from damage.
- Found in green leafy plants and seeds.
- No consistent ill effects are noted with deficiency or excess states, which are uncommon to begin with.

▶ **ECGs**

ECG paper runs at 25 mm/s.
One small box = .040 s
One large box = 200 ms

See Cardiovascular Emergencies chapter for a more complete discussion:

- Electrocardiograms provide a tremendous amount of information in both the acutely ill patient and for a long-term view of cardiac function.
- To become proficient, one must have a system so as not to miss anything, and one must practice, practice, practice.
- Don't believe the machine's reading.

Rate

- Normal
- Bradycardic (< 60 bpm)
- Tachycardic (> 100 bpm)

Rhythm

- Sinus: P waves before every QRS, P upright in I and aVF, all P waves are of same shape (see Figure 3-4 for ECG lead placement).
- Atrial fibrillation: No P waves, irregularly irregular rhythm.
- Atrial flutter: No P waves, sawtooth-shaped waves.
- Ventricular tachycardia: No P wave, no discernible QRS, regular undulating smooth waves.
- Ventricular fibrillation: Grossly irregular waves of varying amplitude, no discernible P or QRS.

Axis

- Normal: 0 to +90
- Rightward: +90 to +270
- Leftward: 0 to –90

See Figure 3-5 for ECG axes.

Rightward axis frequently seen in asthma and COPD patients.

Diagnostics

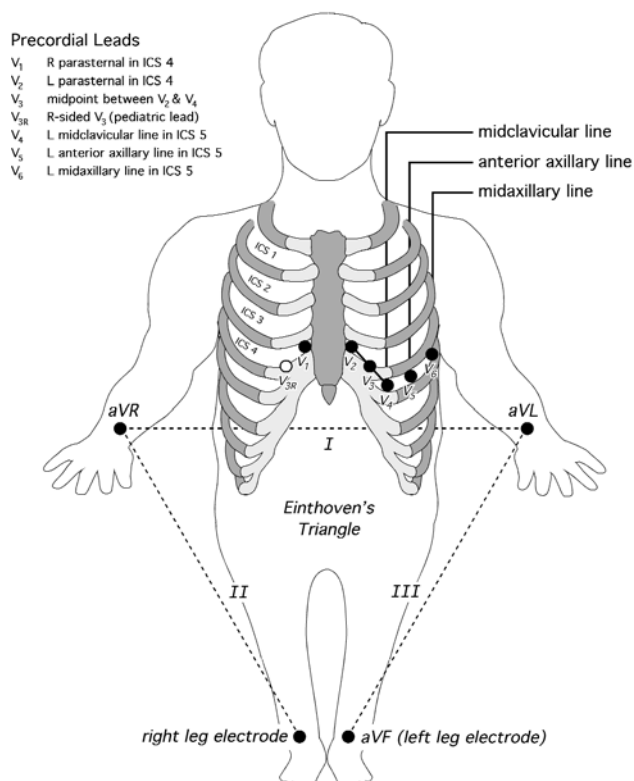**Precordial Leads**
V₁   R parasternal in ICS 4
V₂   L parasternal in ICS 4
V₃   midpoint between V₂ & V₄
V₃ᵣ   R-sided V₃ (pediatric lead)
V₄   L midclavicular line in ICS 5
V₅   L anterior axillary line in ICS 5
V₆   L midaxillary line in ICS 5

midclavicular line
anterior axillary line
midaxillary line

aVR   aVL

I

Einthoven's Triangle

II   III

right leg electrode   aVF (left leg electrode)

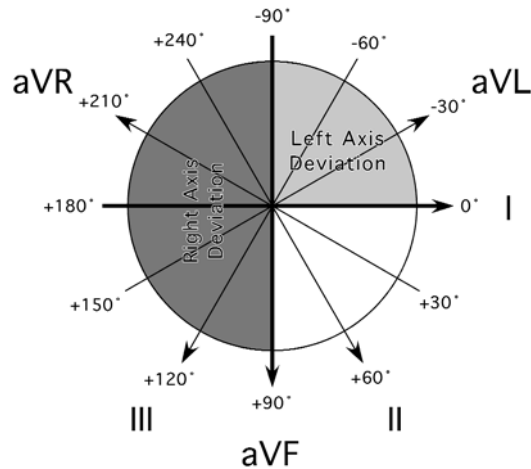**F I G U R E   3 - 4 . ECG lead placement.**

FIGURE 3-5. ECG axes.

Intervals

Intervals are important to determine if there is an atrioventricular (AV) nodal block, intraventricular conduction delay, or prolonged Q:

- PR interval—normal = 200 ms:
  - Consistently > 200 ms is first-degree AV block.
  - Progressively longer and eventually dropping a beat with a repeated pattern is Mobitz type I.
  - Consistent PR with dropped beats in a repeating pattern is Mobitz type II.
  - No association between P wave and QRS is third-degree AV block.
- QRS duration—normal < 120 ms. Greater than 120 ms indicates an intraventricular conduction delay or a left or right bundle branch block.
- QT interval—normal varies with rate but corrected QT (QT/√RR) < 450 ms:
  - Must take patient's age and sex into consideration.
  - Prolonged by hypokalemia, hypomagnesemia, hypocalcemia, and certain medications.
  - Risk of torsade de pointes with prolonged QT.

Morphology

Morphology of the waves is important as well:

- P wave morphology may indicate right or left atrial enlargement (tall P waves) or ectopic atrial focus (different looking P waves).
- QRS complex morphology may indicate right ventricular or left ventricular hypertrophy (tall QRS), right bundle or left bundle branch block (M-shaped QRS complex), or paroxysmal supraventricular tachycardia (wide).
- T waves are helpful to determine ischemic changes or hyperkalemia:
  - In general, T waves should have the same deflection as the QRS. If they're flat or inverted, this may be a sign of ischemia or hypertrophic repolarization changes.
  - Peaked T waves are an early ECG change in hyperkalemia.

Beta blockers and Ca²⁺ channel blockers may cause first-degree block.

A new left bundle branch block is considered an acute myocardial infarction (MI) unless proven otherwise.

ST Segments

ST segments are helpful in determining injury or ischemia. In general:
- Elevation means injury.
- Depression means ischemia.

Q Waves

Q waves are pathologic except in aVR, lead III and V1:
- Most commonly used criteria for significant Q waves is > 40 ms wide and at least one fourth of R wave in same lead.
- Can develop within hours.
- May not be seen in subendocardial infarctions (non–Q wave MI).

U Waves

- U waves are sometimes seen after T waves.
- May indicate hypocalcemia or hypokalemia.

Criteria for Thrombolysis

Criteria for thrombolysis in acute MI barring contraindications:
- Elevated ST segments > 1 mm in two consecutive leads.
- Chest pain or anginal equivalent consistent with MI.
- Contraindications to thrombolysis include recent surgery, active bleeding, recent stroke, suspected dissection, uncontrolled hypertension, or prolonged cardiopulmonary resuscitation.
- New left bundle branch block (LBBB).

Miscellaneous

- Look for pacemaker spike, may be very subtle.
- S in I, Q in III, inverted T in III—think pulmonary embolus.
- If injury is evolving, repeat ECGs are very helpful.

▶ X-RAYS

- X-rays have become a routine part of the evaluation of emergency patients.
- They are used to detect fractures, foreign bodies, pneumonias, CHF, pneumothoraces, and bowel obstruction.
- They are relatively inexpensive and readily available, making them an excellent adjunct when used properly.
- The emergency physician must be comfortable reading his or her own radiographs.

Chest X-Ray

- Use the ABCs for a systematic approach:
  - A —airway. Evaluate the trachea for deviation.
  - B—bones. Evaluate the ribs and other visible bones for evidence of fractures or bony pathology.

Diffuse (across all leads) ST elevations are seen in pericarditis.

Remember, have a system and practice, practice, practice. Even senior cardiologists don't agree with each other on some ECGs, so don't be discouraged.

In radiology, "one view is no view." Try to get a lateral when possible.

- C—cardiac. Look at the cardiac silhouette. It is considered enlarged when greater than one half the thoracic diameter.
- D—diaphragms. Look for free air beneath, flattening, or rising of one side as well as loss of the costophrenic angles.
- E—everything else. Now look at the lung fields for pathology.
- Know the technique used. You may be fooled thinking of cardiomegaly, which shouldn't be read on a portable anteroposterior (AP) chest x-ray.
- Small pneumothoraces may be very subtle and require a hot lamp evaluation.

Obstruction Series

- Consist of upright chest and abdominal x-rays, as well as a supine abdominal x-ray.
- Look for free air, bowel gas patterns, air–fluid levels, and stool in the intestine.

Facial Films

- Useful for trauma and sinus evaluation.
- Complicated fractures are usually followed up with computed tomography (CT) scan.
- Look for opacification of sinuses, air–fluid levels, and mucosal thickening, which are indicative of sinusitis.

Neck Films

- Cervical spine series consists of lateral, AP, and open mouth views:
  - Must see from C1 to top of T1 for complete lateral film.
  - May try shoulder pull or Swimmer's view for larger patients to expose bottom cervical vertebrae.
  - Open mouth used to evaluate dens and lateral masses.
- Soft tissue of neck useful if suspecting epiglottitis or foreign body.

Extremity Films

- Ordered when suspecting fracture or foreign body.
- Multiple views are better.
- Post-reduction views to check for proper positioning are necessary when extremity has been manipulated.
- Can also be used to evaluate for effusions and soft-tissue swelling.

► **CT SCANS**

CT scans have become widely available in the United States and have proven to be invaluable in the diagnosis and treatment of many emergency conditions. A good CT scan, when correlated with the history and patient's condition, can help save a life.

Loss of costophrenic angle indicates approximately 250 cc of fluid accumulation.

Most sensitive film for free air is an upright chest x-ray.

When suspecting epiglottitis, don't send patients to radiology by themselves!

Use **BLT RATS** *when describing fractures:*
Bone
Location
Type of fracture

Rotation
Angulation
Transposition
Shortening

Head CTs

- Useful in atraumatic and traumatic patients.
- Noncontrast CTs can help to see new-onset strokes, bleeds, masses, hydrocephalus, and edema.
- Can also be used to diagnose skull fractures, facial bone fractures, and sinus disease.
- Usually done prior to lumbar puncture to rule out increased intracerebral pressure.
- If HIV+ and infection is suspected, it is best to do without and with contrast to look for toxoplasmosis, cryptococcus, and lymphoma.
- C-spine/neck CTs are useful for penetrating trauma to the neck and to further delineate fractures and subluxations seen on plain C-spine films.

Chest CTs

- Newer-generation, high-resolution spiral CTs with IV contrast are useful for diagnosing pulmonary emboli.
- Have high sensitivity for small pleural effusions and small pneumothoraces not picked up by plain films.
- Useful in evaluating aorta and aortic root in suspected dissection or rupture.

Abdominal/Pelvic CTs

- Useful to detect free fluid in the abdomen.
- With PO and IV contrast administered appropriately, excellent test for infectious processes in the abdomen such as appendicitis, diverticulitis, and abscesses.
- Also useful in evaluating intestinal pathology, although not good for penetrating intestinal trauma.
- Sensitivity not great for pelvic organs. Ultrasound is more useful for gynecologic pathology.

IV Contrast

- Adds a tremendous amount of information.
- Risk of allergy, renal impairment, or asthma exacerbation. Must weigh risks against benefits.
- Exercise caution in patients with renal disease.

▶ ULTRASOUND

- Ultrasound studies have gained an increasing role in emergency medicine.
- Portable machines have found their way to the bedside, and increased training of residents and attendings in ultrasonography has helped patients throughout the country.
- Approved uses in the ED are listed in Table 3-2.
- The two most common uses in the ED currently are pelvic sonography and Focused Abdominal Sonogram for Trauma (FAST) studies.

Acute ischemic strokes may initially present with a negative CT scan.

Chest CT will miss small, peripheral emboli.

PO contrast must be given time to reach the end of the GI tract (minimum 1 to 3 hours).

Intrauterine pregnancy (IUP) on transvaginal sonogram is seen at beta-hCG of 1,000 to 1,500 IU/L. IUP on transabdominal seen at beta-hCG of 6,000 IU/L.

HIGH-YIELD FACTS

Diagnostics

TABLE 3-2. Uses of Ultrasonography in Emergency Medicine

EXAM	FOR DETECTION OF
FAST	Free intraperitoneal fluid in trauma
Cardiac	Pericardial effusion or heart motion in PEA
Pelvic	Early IUP and/or free pelvic fluid
Renal	Hydronephrosis
Aortic	Abdominal aortic aneurysm
Biliary	Cholelithiasis
Obstetric	Live fetus in second/third trimester

Pelvic Ultrasound

- Useful in evaluating the pregnant female with pain or bleeding, ruling out ectopic pregnancies, or evaluating the nonpregnant female with pelvic complaints.
- Order of appearance of structures in pregnancy:
  - Double ring sign
  - Double gestational sac
  - Intrauterine fetal pole
  - Fetal heart activity
- Should be able to visualize uterus, ovaries, bladder, and Douglas' pouch for free fluid.

FAST

- Used to detect free peritoneal blood following blunt trauma to the abdomen.
- Four sites of visualization:
  - Hepatorenal interface (Morison's pouch)
  - Splenic–renal interface
  - Pericardial sac
  - Bladder (Douglas' pouch)
- Sensitivity and specificity vary with experience of user and patient factors.
- Disadvantages:
  - Poor in obese patients or those with lots of bowel gas.
  - Poor in evaluating solid-organ or bowel injury.
  - May not pick up small amounts of fluid.

- Nuclear medicine studies involve the use of hormones or cells that are labeled radioactively to evaluate the function of different organ systems.
- The two most common studies ordered in the ED are hepato-iminodiacetic acid (HIDA) scans and V/Q scans.

HIDA Scan

- IDA is labeled and taken up by hepatocytes and secreted into bile canaliculi.
- Failure to visualize the gallbladder despite seeing the hepatic and common ducts indicates cystic duct obstruction.
- Ninety-eight percent negative predictive value for cholecystitis.
- Sensitivity as high as 97%.

HIDA scan loses sensitivity as bilirubin levels rise above 5 mg.

V/Q Scan

- Indicated for patients when pulmonary embolism is suspected and other diagnoses can't be proven.
- Perfusion scan done by labeling albumin.
- Eight views need to be obtained for complete scan.
- Perfusion scans alone are not sensitive or specific.
- Ventilation scan performed with radioactive aerosols.
- Four results are reported by radiology:
  - Normal scans have specificity of 96% and sensitivity of 98%.
  - Low-probability and intermediate-probability scans are considered nondiagnostic:
    - Correlate with clinical suspicion.
    - Intermediate probability—41% sensitive, positive predictive value (PPV) only 30%.
    - Low probability—16% sensitive, 14% PPV.
  - High-probability scans—41% sensitive, 87% PPV.
- If your clinical suspicion is high enough, go further in workup than V/Q scan (lower extremity Doppler study, spiral chest CT, empiric treatment).

Famous PIOPED study: "Low-probability" V/Q studies miss 16% of pulmonary emboli.

HIGH-YIELD FACTS

Diagnostics

HIGH-YIELD FACTS IN

Trauma

"Golden Hour" of Trauma

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

Prehospital Phase

Control of airway and external hemorrhage, immobilization, and rapid transport of patient to nearest appropriate facility.

Preparation

- Gown up, glove up, face shields on!
- Standard precautions!
- Set up: Airway equipment, monitor, O_2, urinary catheter (Foley), IV and blood tubes (complete blood count, chemistry, prothrombin time/partial thromboplastin time, type and cross, human chorionic gonadotropin, +/− toxicologies), chest tube tray, etc.

Trauma History

Whenever possible, take an **AMPLE** history:
- **A**llergies
- **M**edications/**M**echanism of injury
- **P**ast medical history/**P**regnant?
- **L**ast meal
- **E**vents surrounding the mechanism of injury

Primary Survey

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

ABCs

- **A**irway (with cervical spine precautions)
- **B**reathing and ventilation
- **C**irculation (and **C**ontrol of hemorrhage)
- **D**isability (neurologic status)
- **E**xposure/**E**nvironment control
- **F**oley

ALWAYS follow your ABCs!

Airway and C-Spine

- Assess patency of airway.
- Use jaw thrust or chin lift initially to open airway.
- Clear foreign bodies.
- Insert oral or nasal airway when necessary. Obtunded/unconscious patients should be intubated. Surgical airway—cricothyroidotomy is used when unable to intubate airway.

Assume C-spine injury in trauma patients until proven otherwise.

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 (14 or 16G) IVs.
- Assess circulatory status (capillary refill, pulse, skin color) (see Shock section below).
- Control of life-threatening hemorrhage using **direct pressure;** do not "clamp" bleeding vessels with hemostats.

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 the patient, most often with the help of your trauma shears.
- Hook up monitors (cardiac, pulse oximetry, blood pressure, etc.).

Foley Catheter

- Placement of a urinary catheter is considered part of the resuscitative phase, which takes place during the primary survey.
- Important for monitoring urinary output, which is a reflection of renal perfusion and volume status.
- Adequate urinary output:
  - Adult: 0.5 cc/kg/hr
  - Child (> 1 year of age): 1.0 cc/kg/hr
  - Child (< 1 year of age): 2.0 cc/kg/hr
- Foley is contraindicated when urethral transection is suspected, such as in the case of a pelvic fracture. If transection is suspected, perform retrograde urethrogram before Foley.

Signs of Urethral Transection
- Blood at the meatus
- A "high-riding" prostate
- Perineal or scrotal hematoma

Gastric Intubation

Placement of nasogastric (NG) or orogastric (OG) tube may reduce risk of aspiration by decompressing stomach, but does not assure full prevention.

All trauma patients should receive supplemental O_2.

Draw blood samples at the time of intravenous catheter placement.

AVPU *scale:*
Alert
Verbal
Pain
Unresponsive

Don't forget to keep your patients WARM.

Examine prostate and genitalia before placing a Foley.

Place OG tube rather than NG tube when fracture of cribriform plate is suspected.

Trauma resuscitation is a team sport with many different activities overlapping in both time and space.

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

Use **warmed** fluids whenever possible.

Body water:
⅔ intracellular
⅓ extracellular
¼ intravascular
¾ extravascular

- Crystalloids include saline, Ringer's lactate, and glucose.
- Colloids include blood products such as red blood cells (RBCs) and albumin.

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

Intravenous Catheters

The rate of maximal fluid administration is directly related to the internal diameter of the IV catheter (to the fourth power of the radius according to Poiseuille's law) and inversely related to the length of the tubing.

Intravenous Fluid

- Fluid therapy should be initiated with 1 to 2 L of an isotonic (either lactated Ringer's or normal saline) crystalloid solution (see below).
- Pediatric patients should receive an IV bolus of 20 cc/kg.

Crystalloid versus Colloid

- Crystalloids are sodium-based solutions that provide a transient increase in intravascular volume.
- Approximately one third of an isotonic solution will remain in the intravascular space. The remainder almost immediately distributes to the extravascular and interstitial spaces. This occurs because crystalloid solutions easily diffuse across membranes.
- Colloids have a harder time diffusing across membranes, thus remaining in the intravascular space for longer periods of time thereby requiring smaller volumes for resuscitation. However, it is costly and carries the risks of transfusion reactions and viral transmission.
- Neither crystalloids nor colloids have been shown to be superior for volume resuscitation. Therefore, volume resuscitation begins with crystalloids (see below).

"3 to 1 Rule"

Used as a rough estimate for the total amount of crystalloid volume needed acutely to replace blood loss.

Shock

- Inadequate delivery of oxygen on the cellular level secondary to tissue hypoperfusion.
- In traumatic situations, shock is the result of hypovolemia until proven otherwise.

Hypovolemic Shock

Caused by the acute loss of blood in most cases. Blood volume estimate based on body weight in kilograms:
- Adults: 7% of weight
- Peds: 8 to 9% of weight

For example, 70-kg adult ($70 \times 7\% = 4.9$ L of blood).

Classes of Hemorrhagic Shock

Table 4-1 lists the types of hemorrhagic shock.

Treatment of Hemorrhagic Shock

- Response to the initial fluid bolus (e.g., change in vital signs, urinary output, and/or level of consciousness) should direct further resuscitative efforts.
- Early blood transfusion and surgical intervention should be a consideration in patients who fail to respond to initial fluid resuscitation.

Hemorrhage is the most common cause of shock in the injured patient.

Nonhypovolemic Shock

- **Cardiogenic shock** occurring during trauma may occur secondary to blunt myocardial injury, cardiac tamponade, tension pneumothorax, air embolus, or an acute myocardial infarction.
- **Neurogenic shock** may occur secondary to sympathetic denervation in patients who have suffered a spinal injury.
- **Septic shock** is due to infection and may be seen when there is a significant delay in patients' arrival to the emergency department (ED) or in patients with penetrating abdominal injuries, for example.

Radiologic and Diagnostic Studies

- X-rays of the chest, pelvis, and lateral cervical spine usually occur concurrently with early resuscitative efforts; however, their procedure should never interrupt the resuscitative process.

A "trauma series" consists of radiographs of the C-spine, chest, and pelvis.

TABLE 4-1. Types of Hemorrhagic Shock

Class	Blood Loss (%)	Vol. Blood Loss (cc)	HR	Pulse Pressure	sBP	Urine Output	Altered Mental Status?	Treatment
I	Up to 15	Up to 750	< 100	N	N	N	No	Crystalloids (3 to 1 rule); no blood products necessary
II	15–30	750–1,500	↑	↓	↓	↓	No	Crystalloids initially, then monitor response; may or may not need blood products; can wait for type-specific blood.
III	30–40	1,500–2,000	↑↑	↓↓	↓↓	↓↓	Yes	Crystalloids followed by type-specific blood products
IV	> 40	> 2,000	↑↑↑	↓↓↓	↓↓↓	↓↓↓	Yes	2-L crystalloid bolus followed by uncrossed (O negative) blood; death is imminent.

N = normal; ↑ = increased; ↓ = decreased.

- 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 section on abdominal trauma).

Secondary Survey

- Begins once the primary survey is complete and resuscitative efforts are well under way.
- Includes a **head-to-toe evaluation** of the trauma patient and **frequent reassessment** of status.
- Neurologic examination, procedures, radiologic examination, and laboratory testing take place at this time if not already accomplished.

Tetanus Prophylaxis

Immunize as needed.

Anatomy and Physiology

- Scalp:
  - The scalp consists of five layers.
  - Highly vascular structure.
  - May be the source of major blood loss.
  - The loose attachment between the galea and the pericranium allows for large collections of blood to form a subgaleal hematoma.
  - Disruption of the galea should be corrected and may be done so with single-layer, interrupted 3.0 nonabsorbable sutures through the skin, subcutaneous tissue, and galea.
  - Prophylactic antibiotics are not indicated in simple scalp lacerations.
- Skull:
  - Rigid and inflexible (fixed volume).
  - Composed of the cranial vault and base.
- Brain:
  - Makes up 80% of intracranial volume.
  - Partially compartmentalized by the reflections of dura (falx cerebri and tentorium cerebelli).
  - *Note:* CN III runs along the edge of the tentorium cerebelli.
- Cerebrospinal fluid (CSF):
  - Formed primarily by the choroid plexus at a rate of approximately 500 cc/day with 150 cc of CSF circulating at a given moment.
  - Cushions the brain.
- Cerebral blood flow:
  - Brain receives approximately 15% of cardiac output.
  - Brain responsible for ~20% of total body O_2 consumption.
- Cerebral perfusion pressure (CPP):
  - $CPP = MAP - ICP$
  - MAP = mean arterial blood pressure
  - ICP = intracranial pressure

Layers of the scalp:
Skin
Connective tissue
Aponeurosis (galea)
Loose areolar tissue
Pericranium

Concept of CPP is important in a hypertensive patient. Lowering the BP too fast will also decrease the CPP, creating a new problem.

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 an decreased volume of the others. If not, the ICP will increase.
- Increased ICP can thus result in cerebral herniation, or when ICP = systolic blood pressure (BP), cerebral blood flow ceases and brain death occurs.

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: Hypertension and bradycardia in the setting of increased ICP.

Physical Exam

- Search for signs of external trauma such as lacerations, ecchymoses, and avulsions, as these may be clues to underlying injuries such as depressed or open skull fractures.
- Anisocoria (inequality of pupils) is found in a small percentage of normal people; however, unequal pupils in the patient with head trauma is pathologic until proven otherwise.

Glasgow Coma Scale (GCS)

GCS (Figure 4-1) may be used as a tool for classifying head injury:
- Severe head injury: GCS ≤ 8
- Moderate head injury: GCS 9 to 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:
  - Neurologic deficit.
  - Persisting depression or worsening of mental status.
  - Moderate to severe mechanism of injury.
  - Depressed skull fracture or linear fracture overlying a dural venous sinus or meningeal artery groove (as demonstrated with skull x-rays).

The Cushing reflex is the brain's attempt to maintain the CPP.

Hypotension is usually not caused by isolated head injury. Look for other injuries in this setting.

Skin staples interfere with CT scanning and should therefore not be used until after CT scanning is complete.

Eyes	Open spontaneously	4
	Open to verbal command	3
	Open to pain	2
	No response	1
Best motor response	Obeys verbal command	6
	Localizes pain to painful stimulus	5
	Flexion withdrawal	4
	Decorticate rigidity	3
	Decerebrate rigidity	2
	No response	1
Best verbal response	Oriented and converses	5
	Disoriented and converses	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
TOTAL		**15**

FIGURE 4-1. Glasgow Coma Scale.

▶ **SKULL FRACTURES**

Linear (Nondepressed)

Becomes clinically important if it occurs over the middle meningeal artery groove or major venous dural sinuses (formation of an epidural hematoma), air-filled sinuses, or if associated with underlying brain injury.

Stellate

Suggestive of a more severe mechanism of injury than linear skull fractures.

Depressed

- Carries a much greater risk of underlying brain injury and complications, such as meningitis and post-traumatic seizures.
- Treatment involves surgical elevation for depressions deeper than the thickness of the adjacent skull.

Basilar

- Often a clinical diagnosis and sign of a significant mechanism of injury.
- Signs include periorbital ecchymoses (raccoon's eyes), retroauricular ecchymoses (Battle's sign), otorrhea, rhinorrhea, hemotympanum, and cranial nerve palsies.

Open

- A laceration overlying a skull fracture.

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.

- Requires careful debridement and irrigation. Avoid blind digital probing of the wound.
- Obtain neurosurgical consultation.

Cerebral Concussion

- Transient loss of consciousness that occurs immediately following blunt, nonpenetrating head trauma, caused by impairment of the reticular activating system.
- Amnesia and confusion are typical.
- 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, and admission to a neurosurgical intensive care unit.
- Patients are often comatose for prolonged periods of time.
- Mortality is approximately 33%.

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 usually have focal deficits; mental status ranges from confusion to coma.
- Common locations include frontal poles, subfrontal cortex, and anterior temporal lobes. Contused area is hemorrhagic.

Epidural Hematoma

- Collection of blood located between the dura and the skull.
- Majority are associated with tearing of the middle meningeal artery from an overlying temporal bone fracture.
- Typically biconvex or lenticular in shape (see Figure 4-2).
- Patients may have the classic "lucid interval," wherein they "talk and die." Requires early neurosurgical involvement.

Subdural Hematoma

- Collection of blood below the dura and over the brain (see Figure 4-3). Results from tearing of the bridging veins, usually secondary to an acceleration–deceleration mechanism.

A 20-year-old female sustains 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.*

No mass lesion is seen on CT in DAI.

A 19-year-old male with head injury has loss of consciousness followed by a brief lucid interval. He presents to the ED in a coma, with an ipsilateral fixed and dilated pupil and contralateral hemiparesis. *Think: Epidural hematoma.*
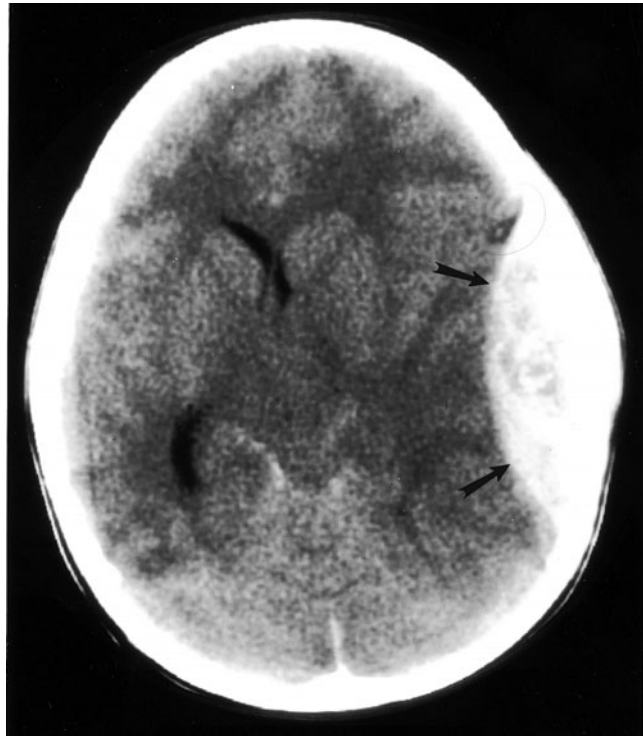
HIGH-YIELD FACTS

Trauma

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F I G U R E 4 - 2 . Epidural hematoma. Arrows indicate the characteristic lens-shaped lesion.

Arrows indicate the characteristic lens-shaped lesion. (Reproduced, with permission, from Schwartz SI, Spencer SC, Galloway AC, et al. *Principles of Surgery*, 7th ed. New York: Mc-Graw-Hill, 1999: 1882.)

- Classified as acute (< 24 hours), subacute (24 hours to 2 weeks), and chronic (> 2 weeks).
- Acute and subacute subdurals require early neurosurgical involvement.
- Alcoholics and the elderly (patients likely to have brain atrophy) have increased susceptibility.

Acute subdural hematomas have a high mortality: Approximately one third to two thirds.

When in doubt, admit the patient with head trauma for observation.

▶ **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, and reliable family members who can adequately observe the patient at home can often be discharged home 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

F I G U R E   4 - 3 .   Head CT showing left subdural hematoma overlying left frontal lobe (arrow) with associated midline shift.

- Inequality of pupils
- Confusion
- If any of the above signs are found, the patient should be brought to the ED immediately.

▶ MANAGEMENT OF SEVERE HEAD TRAUMA

- Patients must be treated aggressively starting with the ABCs.
- Secure the airway via endotracheal intubation using topical anesthesia, intravenous lidocaine, and paralytics when necessary to prevent any further increase in the ICP.
- Maintain an adequate BP with isotonic fluids. (Aim for mean arterial pressure [MAP] of 90 mm Hg.)
- Treatment of increased ICP:
  - Elevate head of the bed to 30°.
  - Hyperventilation to an arterial P_{CO_2} of 30 to 35 will decrease the ICP by approximately 25% acutely.
  - Mannitol (1 g/kg in a 20% solution) is an osmotic diuretic and lowers ICP by drawing water out of the brain. Contraindicated in the hypotensive patient.
- Corticosteroids (used in penetrating spinal trauma) have not been shown to be useful in the patient with head trauma.

Measures to lower ICP:
HIVED
- **H**yperventilation
- **I**ntubation with pretreatment and sedation
- **V**entriculostomy (burr hole)
- **E**levate the head of the bed
- **D**iuretics (mannitol, furosemide)

- Consider prophylactic anticonvulsant therapy with phenytoin 18 mg/kg IV at no faster than 50 mg/min (usually at the discretion of the neurosurgeon).
- Acute seizures should be managed with diazepam or lorazepam and phenytoin.
- Emergency decompression via trephination (burr holes) may be necessary.
- Treat the pathology whenever possible (e.g., surgical drainage of a hematoma).

General

Described in broad terms as penetrating versus blunt injuries even though considerable overlap exists between the management of the two.

Anatomy

The neck is divided into triangles (anterior and posterior) as well as zones (I, II, and III).

Anterior Triangle
- Bordered by the midline, posterior border of the sternocleidomastoid muscle (SCM) and the mandible.

Posterior Triangle
- Bordered by the trapezius, posterior border of the SCM, and the clavicle. There is a paucity of vital structures in its upper zone (above the spinal accessory nerve). In the lower zone lies the subclavian vessels and brachial plexus. The apices of the lungs are in close proximity.

Zones (Figure 4-4)
- Further division of the anterior triangle:
  - Zone I lies below the cricoid cartilage.
  - Zone II lies between I and III.
  - Zone III lies above the angle of the mandible.
- These divisions help to 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 cerebrovascular accidents (from transection of the carotid artery or air embolus, for example).

Within the anterior triangle lie the majority of the vital structures of the neck.

Fracture of the hyoid bone is suggestive of a significant mechanism of injury.

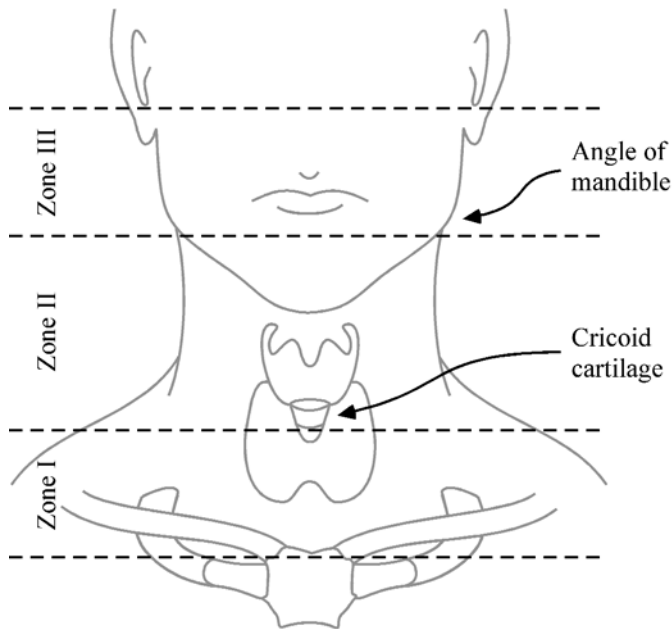C-spine injuries are much more common with blunt neck injury.

FIGURE 4-4. Zones of the neck.

Nonvascular Injuries
- Injury to the larynx and trachea including fracture of the thyroid cartilage and 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.

Resuscitation

AIRWAY

- Special attention should be paid to airway management of the patient with neck trauma.
- Anatomy may be distorted and an apparently patent airway can rapidly evolve into a compromised, difficult airway.
- Initial attempts at securing the airway should be via endotracheal intubation; however, alternative methods of airway management, such as percutaneous transtracheal ventilation and surgical airway, should be readily available.

BREATHING

- Inability to ventilate the patient after an apparently successful intubation should prompt rapid reassessment of that airway.
- Creation and/or intubation of a "false lumen" in the patient with laryngotracheal or tracheal transection may be a fatal error if not identified immediately.
- Look for pneumohemothorax, as the apices of the lungs lie in close proximity to the base of the neck.

Avoid unnecessary manipulation of the neck, as this may dislodge a clot.

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

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

Control of hemorrhage in the ED is via direct pressure (no blind clamping).

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

CIRCULATION

- If the patient remains unstable after appropriate volume resuscitation, he or she should be taken rapidly to the operating room (OR) for operative control of the bleeding.
- If injury to the subclavian vessels is suspected, IV access should be obtained in the opposite extremity, or more appropriately in the lower extremities.
- If a hemopneumothorax is suspected and central venous access is necessary, a femoral line is the first option, followed by placement of the access on the side ipsilateral to the "dropped lung" (because the patient doesn't like it when both lungs are down!).

Secondary Survey

- After stabilization, the wound should be carefully examined.
- Obtain soft-tissue films 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 deficits as well as Horner's syndrome.

Management

- Zone II injuries are taken to the OR for exploration.
- Injuries to zones I and III may be taken to the OR or managed conservatively using a combination of angiography, bronchoscopy, esophagoscopy, gastrografin or barium studies, and CT scanning.

▶ **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, 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 7 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 4 coccygeal vertebrae.

Patients on a backboard for a prolonged period of time are at risk for the formation of pressure ulcers.

- The cervical spine is the region that is most vulnerable to injury.
- The thoracic spine is relatively protected due to limited mobility from support of the rib cage (T1–10); 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 versus unstable).
- Level of neurologic (sensory and motor) and bony involvement.
- Severity (complete versus 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 include 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 VERSUS 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.

Physical Exam

- Classification of spinal cord injuries as complete or incomplete requires a proper neurologic exam.

Mechanisms suspicious for spinal injury:
- Diving
- Fall from > 10 feet
- Injury above level of shoulders (C-spine)
- Electrocution
- High-speed motor vehicle crash
- Rugby or football injury (tackling)

Deep tendon reflexes and sacral reflexes may be preserved in complete injuries.

HIGH-YIELD FACTS

Trauma

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- The exam should include testing of the three readily assessable long spinal tracts (see Figure 4-5):
  - 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 is tested as such.
  - Posterior (dorsal) columns:
    - Located in the posterior aspect of the spinal cord.
    - Responsible for ipsilateral position and vibratory sense and some light touch sensation.
    - Test 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.
- 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.
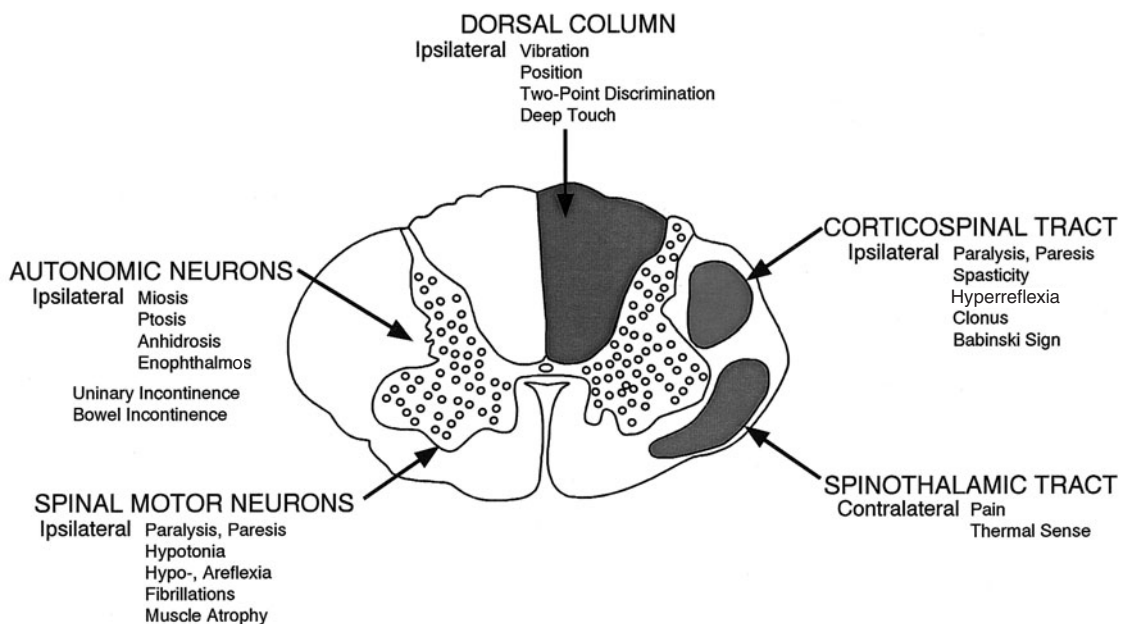
F I G U R E 4 - 5 . Effects of lesions in major spinal cord tracts.

(Reproduced, with permission, from Afifi AA, Bergman RA. *Functional Neuroanatomy: Text and Atlas.* New York: McGraw-Hill, 1998: 92.)

BROWN–SÉQUARD SYNDROME

- Pattern seen with hemisection of the spinal cord usually secondary to a penetrating injury, but may also be seen with disc 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 flavum 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).

A 70-year-old male presents to the ED after a whiplash injury. He is ambulating well but has an extremely weak handshake. *Think: Central cord syndrome.*

Treatment of Spinal Cord Syndromes

- 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.
- For penetrating spinal cord injury, administer prophylactic antibiotics and consider high-dose methylprednisolone in consultation with neurosurgeon:
  - Loading dose of 30 mg/kg over 15 minutes during hour 1, followed by a continuous infusion of 5.4 mg/kg/hr over the next 23 hours.
- Consider traction devices in consultation with the neurosurgeon.
- Consider early referral to a regional spinal injury center.

▶ C-SPINE FRACTURES AND DISLOCATIONS

General

As mentioned above, usually classified on the basis of mechanism (flexion, extension, compression, rotation, or a combination of these), location, and/or stability.

Imaging

- Three views of the cervical spine are obtained (lateral, anteroposterior [AP], and an odontoid view) for best accuracy.
- A lateral view alone will miss 10% of C-spine injuries.
- Adequate AP and lateral films will allow visualization of C1–T1.
- If C1–T1 can still not be adequately visualized, CT scanning is indicated.

NEXUS Low-Risk Criteria:
C-spine films are not indicated for patients with:
- No posterior midline cervical spine tenderness
- No evidence of intoxication
- Normal level of alertness
- No focal neurologic deficit
- No painful distracting injuries

- Most common level of fracture is C5.
- Most common level of subluxation is C5 on C6.

Reading a C-Spine Film

Alignment
- Evaluate the alignment of the four lordotic curves (see Figure 4-6):
  - Anterior margin of the vertebral bodies
  - Posterior margin of the vertebral bodies
  - Spinolaminar line
  - Tips of the vertebral bodies: In the adult, up to 3.5 mm of anterior subluxation is considered a normal finding.

Bones
- Assess the base of the skull and each vertebral body, pedicle, facet, laminae, and spinous and transverse process for fracture/dislocation.

Cartilage
- Assess the intervertebral spaces and posterolateral facet joints for symmetry.

Soft Tissue
- Assess the prevertebral soft tissue: Wider than 5 mm suggests hematoma accompanying a fracture.
- Assess the predental space: Wider than 3 mm in adults and 4 to 5 mm in children is suggestive of a torn transverse ligament and fracture of C1.
- Assess the spaces between the spinous processes: Any increase in distance between the spinous processes is likely associated with a torn interspinous ligament and a spinal fracture.

ATLANTO-OCCIPITAL DISLOCATION

- Results from severe traumatic flexion.
- Survival to the hospital setting is rare.
- Traction is not recommended.
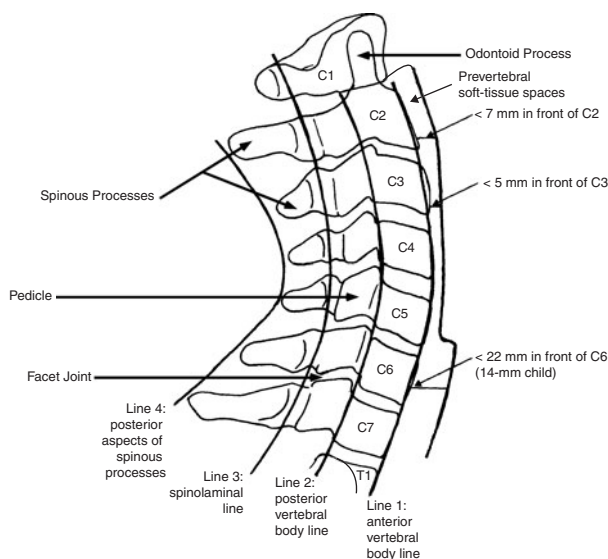
FIGURE 4-6. Lateral view of the cervical spine.

The four "lines" should flow smoothly, without step-up. The prevertebral soft-tissue spaces should be within normal.

Jefferson Fracture (Figure 4-7)

- 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.
- Often associated with C2 fractures.
- Consider all C1 fractures unstable even though most are not associated with spinal cord injury.
- Seen as an increase in the predental space on lateral x-ray and displacement of the lateral masses on the odontoid view.

C1 Rotary Subluxation

- Seen most often in children or in patients with rheumatoid arthritis.
- Seen as an asymmetry between the lateral masses and the dens on the odontoid view.
- Patients will present with the head in rotation and should not be forced to place the head in the neutral position.

Odontoid Fractures

- Type 1: Involves only the tip of the dens (stable).
- Type 2: Involves only the base of the dens (most common type).
- Type 3: Fracture through the base and body of C2.
- Generally unstable.

Some type 1 fractures may be stable when the transverse ligament remains intact.

Hangman's Fracture (Figure 4-8)

- Fracture of both pedicles ("posterior elements") of C2.
- 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.

FIGURE 4-7. Jefferson fracture.

FIGURE 4-8. Hangman fracture.

BURST FRACTURE OF C3–7

- An axial loading mechanism causing compression of a vertebral body with resultant protrusion of the anterior portion of the vertebral body anteriorly and the posterior portion of the vertebral body posteriorly into the spinal canal often causing a spinal cord injury (usually the anterior cord syndrome).
- Stable fracture when ligamentous structure remains intact.

SIMPLE WEDGE FRACTURE

- A flexion injury causing compression on the anterior portion of the vertebral body.
- Appears as a wedge-shaped concavity, with loss of vertebral height on the anterior portion of the vertebral body.
- Usually stable when not associated with ligamentous damage.

FLEXION TEARDROP FRACTURE (FIGURE 4-9)

- A flexion injury causing a fracture of the anteroinferior portion of the vertebral body.
- Appears as a teardrop-shaped fragment.
- Unstable fracture, as it is usually associated with a tearing of the posterior ligament and often neurologic damage.

EXTENSION TEARDROP FRACTURE

- Also appears as a teardrop-shaped fragment on the anteroinferior portion of the vertebral body
- However, occurs as an extension injury with avulsion of the fragment, rather than a compression mechanism
- The posterior ligaments are left intact, making this a stable fracture.
- However, differentiation between a flexion versus extension teardrop fracture may be difficult and should be treated initially as if it were unstable.

FIGURE 4-9. Flexion teardrop fracture.

Clay Shoveler's Fracture

- 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.

Unilateral Facet Dislocation

- Occurs as a flexion–rotation injury.
- Usually stable, but is potentially unstable as it often involves injury to the posterior ligamentous structures.
- Often identified on the AP view of the C-spine films when the spinous processes do not line up.

Bilateral Facet Dislocation

- Occurs as a flexion injury and is extremely unstable.
- Associated with a high incidence of spinal cord injury.
- Appears on lateral C-spine films as a subluxation of the dislocated vertebra of greater than one half the AP diameter of the vertebral body below it.

Subluxation

- Occurs with disruption of the ligamentous structures without bony involvement.
- Potentially unstable.
- Findings on C-spine films may be subtle, and flexion–extension views may be needed.

Thoracic Spine Fractures

- As mentioned above, the majority of injuries take place at the junction between the relatively fixed upper thoracic spine and the mobile thoracolumbar region (T10–L5).
- When thoracic fractures do take place, they can be devastating because the spinal canal through this region is relatively 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.
- The majority of fractures/dislocations in this area are considered stable because of the surrounding normal bony thorax.
- However, as mentioned, neurologic impairment resulting from injuries in this area is often complete.

> ► **THORACOLUMBAR JUNCTION AND LUMBAR SPINE FRACTURES AND DISLOCATIONS**

Compression (Wedge) Fracture

- Results from axial loading and flexion.
- Potentially unstable.

Frequency: L1 > L2 > T1

- Neurologic injury is uncommon.
- Treatment is symptomatic (patients usually experience pain and are at increased risk for the formation of an ileus).

Burst Fracture

- Fracture of the vertebral end plates with forceful extrusion of the nucleus pulposus into the vertebral body causing comminution of the vertebral body.
- Results from axial loading.
- See loss of vertebral height on lateral spine film.

Distraction or Seat Belt Injury

- Frequently referred to as a "chance fracture."
- Horizontal fracture through the vertebral body, spinous processes, laminae, and 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.

Fracture–Dislocations

- Result from flexion with rotation.
- Unstable and often associated with spinal cord damage.

Abdominal injuries frequently coexist with fracture–dislocations.

▶ **SACRAL AND COCCYGEAL SPINE FRACTURES AND DISLOCATIONS**

- Fractures in this area are relatively uncommon.
- Sacral injuries must often be diagnosed via CT scan.
- Neurologic impairment is rare; however, damage to the sacral nerve roots results in bowel/bladder and sexual dysfunction as well as loss of sensory and motor function to the posterior lower extremities.
- Fractures of the coccyx are usually caused by direct trauma.
- Diagnosis is made upon palpation of a "step-off" on rectal examination, and rectal bleeding must also be ruled out (severe fractures may lead to a rectal tear).
- Treatment of uncomplicated coccygeal fracture is symptomatic and includes pain management and a doughnut pillow.

▶ **THORACIC TRAUMA**

Cardiac 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 4-10).
- Diagnosis may be confirmed with cardiac sonogram if immediately available.

Beck's tamponade triad:
1. Hypotension
2. JVD
3. Muffled heart sounds
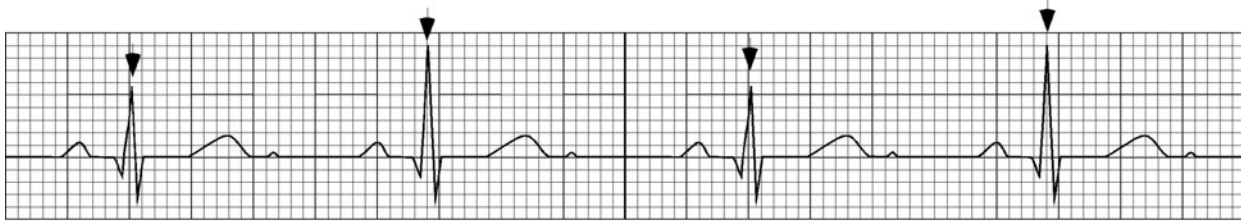
F I G U R E 4 - 1 0 . **ECG demonstrating electrical alternans. Note alternating heights of the R (arrow) in the QRS complexes.**

- Requires immediate decompression via **needle pericardiocentesis** (see Procedures chapter, Figure 17-2), pericardial window, or thoracotomy with manual decompression.

Pneumothorax

DEFINITION

Air in the pleural space.

SIGNS AND SYMPTOMS

- Chest pain
- Dyspnea
- Hyperresonance of affected side
- Decreased breath sounds of affected side

DIAGNOSIS

Upright chest x-ray is ~83% sensitive, demonstrates an absence of lung markings where the lung has collapsed (see Figure 4-11).

TUBE THORACOSTOMY

See Procedures chapter.

Tension Pneumothorax

- Life-threatening emergency caused by air entering the pleural space (most often via a hole in the lung tissue) but being unable to escape.
- Causes total ipsilateral lung collapse, mediastinal shift **(away from injured lung)** impairing venous return and thus decreased cardiac output, eventually resulting in shock.
- Signs and symptoms include dyspnea, hypotension, tracheal deviation, absent breath sounds, and hyperresonance to percussion.
- Requires immediate needle decompression followed by tube thoracostomy.

A 19-year-old male who was stabbed in the chest with a knife presents complaining of dyspnea. Breath sounds on the left are absent.
Think: Pneumothorax.

A diagnosis of tension pneumothorax via x-ray is a missed diagnosis. Do not delay treatment of a suspected tension pneumothorax in order to confirm your suspicion (i.e., tension pneumothorax is a **clinical** diagnosis).
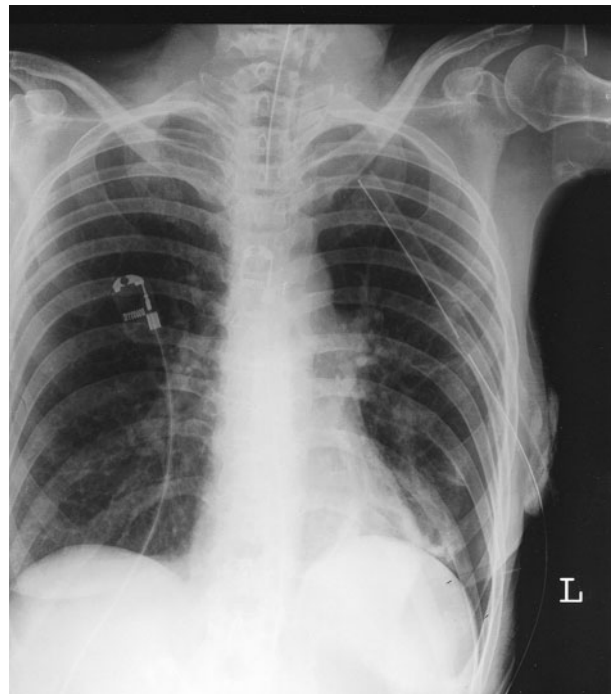
FIGURE 4-11. **A. CXR demonstrating left-sided pneumothorax. Note lack of lung markings. B. Same patient, after tube thoracostomy and endotracheal intubation.**

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 a tube thoracostomy (chest tube).

Hemothorax

- Defined as the presence of blood in the lungs.
- > 200 cc of blood must be present before blunting of costophrenic angle will be seen on CXR.
- Treatment involves chest tube placement and drainage.

Indications for Thoracotomy

- 1,500 cc initial drainage from the chest tube.
- 200 cc/hr continued drainage.
- Patients who decompensate after initial stabilization.
- > 50% hemothorax.

Traumatic Aortic Rupture

- Most often seen with sudden deceleration injuries (high-speed motor vehicle crash, falls from > 25 feet).
- Most frequent site of rupture is ligamentum arteriosum.
- High-mortality injury: Almost 90% die at the scene, and another 50% of those that survive the scene die within 24 hours.
- Signs and symptoms:
  - Retrosternal chest pain.
  - Dyspnea.
  - New systolic murmur.
  - Pseudocoarctation syndrome: Increased BP in upper extremities with absent or decreased femoral pulses.
  - Pulse deficits between upper and lower extremities.

- Findings on CXR (see Figure 4-12):
  - Widened mediastinum
  - Tracheal or NG tube deviation to the right
  - Depression of left main stem bronchus
  - Widening of paratracheal stripe to the right
  - Indistinct aortic knob
  - Indistinct space between pulmonary artery and aorta
  - Presence of left apical cap
  - Multiple rib fractures
- Diagnosis is via angiography.
- Management: Fluid resuscitation and stat surgical consultation for OR.

ED Thoracotomy

INDICATIONS

- Salvageable patient with postinjury cardiac arrest.
- Persistent severe hypotension secondary to tamponade, intrathoracic hemorrhage, or air embolism.

PROCEDURE BASICS

- Left anterolateral incision: Fourth to fifth intercostal space.
- Rib retractor.
- Pericardiotomy:
  - Evacuation of blood and clots.
  - Control bleeding with digital pressure and/or partially occluding vascular clamp on atria or great vessels.
- In a nonbeating heart, may suture lacerations with 3-0 nonabsorbable suture.
- In a beating heart, delay repair of defects until initial resuscitation is completed and patient is in OR.

- One fourth of cases of hemothorax have an associated pneumothorax.
- Three fourths of cases of hemothorax are associated with extrathoracic injuries.

Because of the low success rate for blunt injuries, ED thoracotomy is **not** indicated for blunt thoracic trauma.

F I G U R E 4 - 1 2 . **CXR illustrating wide mediastinum due to penetrating trauma of the ascending aorta.**

OUTCOME

- Overall survival for penetrating trauma—20%.
- Success rate for patients with signs of life—30 to 57%.
- Success rate if no signs of life—13%.
- Success rate for blunt injuries—1 to 2%.

Sucking Chest Wound

- Also known as a communicating pneumothorax.
- Caused by an open defect in the chest wall, often due to gunshot injuries.
- If the diameter of the defect is greater than two thirds the diameter of the trachea, air will preferentially enter through the defect.
- The affected lung will collapse on inspiration as air enters through the defect and expand slightly on expiration. This mechanism seriously impairs ventilation.
- Initial treatment involves covering wound with an occlusive dressing sealed on three sides. This will convert it to a closed pneumothorax while the unsealed side will allow air to escape, preventing conversion into a tension pneumothorax.

Pulmonary Contusion

- Damage to the lung parenchyma without pulmonary laceration.
- Most common mechanism is direct chest trauma in a rapid deceleration injury.
- Signs and symptoms:
  - Dyspnea
  - Tachypnea
  - Local ecchymosis
- Arterial blood gas (ABG) findings:
  - Hypoxemia
  - Widened A-a gradient
- Findings on CXR:
  - Local irregular patchy infiltrate that corresponds to site of injury. This develops usually immediately, and always within 6 hours.
- Treatment involves supplemental oxygen and pulmonary toilet.
- Most frequent complication is pneumonia.

> **ABDOMINAL TRAUMA**

General

Penetrating abdominal injuries (PAIs) resulting from a gunshot 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.

Potential causes of *iatrogenic* great vessel injury:
- CVP line or chest tube placement
- Intra-aortic balloon pump (IABP) placement
- Use of nonvascular clamp during ED thoracotomy
- Overinflation of Swan–Ganz balloon

A 25-year-old female presents after a high-speed motor vehicle crash with dyspnea and tachycardia. There is local bruising over the right side of her chest. CXR shows a right upper lobe consolidation. *Think: Pulmonary contusion.*

The most frequently injured solid organ associated with penetrating trauma is the liver, followed by the small bowel.

Blunt abdominal injury also has three general mechanisms of injury:

1. Injury caused by the direct blow
2. Crush injury
3. Deceleration injury that occurs

Anatomy

- Anterior abdominal wall—bordered laterally by the midaxillary lines, superiorly by a horizontal line drawn through the nipples and inferiorly by the symphysis pubis and inguinal ligaments.
- The "thoracoabdominal region" is that region below the nipples and above the costal margins and is the area within which the diaphragm travels. Penetrating injuries to this region are more likely to involve injury to the diaphragm.
- Flank—area between the anterior and posterior axillary lines.
- Back—area posterior to the posterior axillary lines, bordered superiorly by a line drawn through the tips of the scapulae and inferiorly by the iliac crests.
- Peritoneal viscera—liver, spleen, stomach, small bowel, sigmoid and transverse colon.
- Retroperitoneal viscera—majority of the duodenum (fourth part is intraperitoneal), pancreas, kidneys and ureters, ascending and descending colon, and major vessels such as the abdominal aorta, inferior vena cava, renal and splenic vessels.
- Pelvic viscera—bladder, urethra, ovaries and uterus in women, prostate in men, rectum, and iliac vessels.

The most frequently injured solid organ associated with blunt trauma is the spleen, followed by the liver.

Physical Examination

- **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.
- Inspect the abdomen for evisceration, entry/exit wounds, impaled objects, and a gravid uterus.

Diagnosis

- Perforation: Abdominal x-ray 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).

FAST

- Used as a rapid bedside screening study.
- Noninvasive and not time consuming.
- Positive if free fluid is demonstrated in the abdomen.

Serial abdominal examinations can and should be performed.

Trauma

Three abdominal views are utilized to search for free intraperitoneal fluid (presumed to be blood in the trauma victim), which collects in dependent areas and appears as hypoechoic areas on ultrasound:

- Morison's pouch in the right upper quadrant: Free fluid can be visualized between the interface of the liver and kidney.
- Splenorenal recess in the left upper quadrant: Free fluid can be visualized between the interface of the spleen and kidney.
- Pouch of Douglas, which lies above the rectum (probe is placed in the suprapubic region).
- Subxiphoid and parasternal views to look for hemopericardium.

DPL

Advantages	Disadvantages
▪ Performed bedside	▪ Invasive
▪ Widely available	▪ Risk for iatrogenic injury
▪ Highly sensitive for hemoperitoneum	▪ Relatively low specificity (many false positives)
▪ Rapidly performed	▪ Does not evaluate the retroperitoneum

DPL Technique: Open

Make vertical incision carefully from the skin to the fascia. Grasp the fascial edges with clamps, elevate, then incise through to the peritoneum. Insert the peritoneal catheter and advance toward the pelvis.

DPL Technique: Closed

- Using skin clamps or an assistant's sterile gloved hands, elevate the skin on either side of the site of needle placement and make a "nick" with a #11 blade.
- Insert the needle (usually an 18G needle comes with the kit) angled slightly toward the pelvis, through the skin and subcutaneous tissue, and into the peritoneum. Most often three areas of resistance are met (felt as "pops") as the needle is passed through: Linea alba, transversalis fascia, and peritoneum.
- Using the Seldinger technique, a guidewire is placed through the needle and advanced into the peritoneum.
- The needle is removed, leaving the guidewire in place.
- The peritoneal catheter is then threaded over the wire, and the wire is removed.
- The peritoneal catheter is in a syringe and is connected to the catheter and aspiration is performed. If gross blood appears (> 5 to 10 cc), the patient should be taken to the OR for exploratory laparotomy.
- If the aspiration is negative, instill 15 cc/kg of warmed normal saline or lactated Ringer's solution into the peritoneum through IV tubing connected to the catheter.
- Let the solution stand for up to 10 minutes (if the patient is stable), then place the IV bag from which the solution came from on the floor for drainage via gravity.
- A sample of the returned solution should be sent to the lab for STAT analysis.

DPL: If pelvic fracture is suspected, a supraumbilical approach should be used.

DPL: If the patient is pregnant, a suprafundal approach should be used.

CT Scanning

- Useful for the hemodynamically stable patient.
- Has a greater specificity than DPL and ultrasonography (US).
- Noninvasive.
- Relatively time consuming when compared with DPL and US.
- Diagnostic for specific organ injury; however, may miss diaphragmatic, colonic, and pancreatic injury.

Serial Hematocrits

Serial hematocrits should be obtained during the observation period of the hemodynamically stable patient.

Laparotomy

Indications for Exploratory Laparotomy
- Abdominal trauma and hemodynamic instability.
- Bleeding from stomach (not to be confused with nasopharyngeal bleeding).
- Evisceration.
- Peritoneal irritation.
- Suspected/known diaphragmatic injury.
- Free intraperitoneal or retroperitoneal air.
- Intraperitoneal bladder rupture (diagnosed by cystography).
- Positive DPL.
- Surgically correctable injury diagnosed on CT scan.
- Removal of impaled instrument.
- Rectal perforation (diagnosed by sigmoidoscopy).
- Transabdominal missile (bullet) path (e.g., a gunshot wound to the buttock with the bullet being found in the abdomen or thorax).

▶ GENITOURINARY (GU) TRAUMA

General

- Often overlooked in the initial evaluation of the multiply injured trauma victim.
- 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).

Anatomy

The GU tract injury is divided into upper (kidney and ureters) and lower tract (bladder, urethra, and genitalia) injury.

Signs and Symptoms

- Flank or groin pain
- Blood at the urethral meatus
- Ecchymoses on perineum and/or genitalia
- Evidence of pelvic fracture

Criteria for a positive DPL:
- > 100,000 RBCs
- > 500 white blood cells (WBCs)
- Gram stain with bacteria or vegetable matter
- Amylase > 20 IU/L
- Presence of bile

Contraindications to DPL:
Absolute
- Clear indication for laparotomy present

Relative
- Coagulopathy
- Previous abdominal surgeries
- Morbid obesity
- Gravid uterus

CT is the most sensitive test for retroperitoneal injury.

Suspect GU trauma with:
- Straddle injury
- Penetrating injury to lower abdomen
- Falls from height

- Rectal bleeding
- A "high-riding" or superiorly displaced prostate

Placement of Urethral Catheter

- A Foley or coudé catheter should be placed in any trauma patient with a significant mechanism of injury in the **absence** of any sign of urethral injury.
- Partial urethral tears warrant one careful attempt of a urinary catheter. If any resistance is met or a complete urethral tear is diagnosed, suprapubic catheter placement will be needed to establish urinary drainage.

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

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.

Retrograde Urethrogram

- Should be performed in any patient with suspected urethral disruption (before Foley placement).
- A preinjection KUB (kidneys, ureter, and bladder) film should be taken.
- A 60-cc Toomey syringe (versus a Luer-lok syringe) should be filled with the appropriate contrast solution and placed in the urethral meatus.
- With the patient in the supine position, inject 20 to 60 cc contrast over 30 to 60 seconds.
- A repeat KUB is taken during the last 10 cc of contrast injection.
- Retrograde flow of contrast from the meatus to the bladder without extravasation connotes urethral integrity and Foley may then be placed.
- May be performed in the OR in patients requiring emergency surgery for other injuries.

History of enlarged prostate, prostate cancer, urethral stricture, self-catheterization, or previous urologic surgery may make Foley placement difficult or can be confused with urethral disruption.

Bladder Rupture

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

Extraperitoneal
- Usually occurs due to pelvic fracture.
- Treatment is nonsurgical management by Foley drainage.

Retrograde Cystogram

- Should be performed on patients with gross hematuria or a pelvic fracture.
- Obtain preinjection KUB.
- Fill the bladder with 400 cc of the appropriate contrast material using gravity at a height of 2 feet.

HIGH-YIELD FACTS

Trauma

- Obtain another KUB.
- Empty the bladder (unclamp the Foley), then irrigate with saline and take another KUB ("washout" film).
- Extravasation of contrast into the pouch of Douglas, paracolic gutters, or between loops of intestine is diagnostic for intraperitoneal rupture and requires operative repair of the bladder.
- Extravasation of contrast into the paravesicular tissue or behind the bladder as seen on the "washout" film is indicative of extraperitoneal bladder rupture.

Ureteral Injury

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

Renal Contusion

- Most common renal injury.
- Renal capsule remains intact.
- IVP is usually normal and CT scan may show evidence of edema or microextravasation of contrast into the renal parenchyma.
- Often associated with a subcapsular hematoma.
- Management is conservative and requires admission to the hospital.
- Recovery is usually complete unless there is underlying renal pathology.

Renal Laceration

- Classified as either minor (involving only the renal cortex) or major (extending into the renal medulla and/or collecting system).
- Diagnosed by CT scan or IVP.
- Minor renal lacerations are managed expectantly.
- Management of major renal lacerations varies and depends on the surgeon, hemodynamic stability of the patient, and the extent of injury and its coincident complications (ongoing bleeding and urinary extravasation).

Renal Fracture ("Shattered Kidney")

- Involves complete separation of the renal parenchyma from the collecting system.
- Usually leads to uncontrolled hemorrhage and requires surgical intervention.

▶ EXTREMITY TRAUMA

Signs and Symptoms

- Tenderness to palpation
- Decreased range of motion
- Deformity or shortening of extremity

Rhabdomyolysis causes myoglobin release, which can cause renal failure. Maintaining a high urine output together with alkalinization of the urine can help prevent the renal failure by reducing precipitation of myoglobin in the kidney.

Signs of compartment syndrome: **The 6 Ps**
Pain
Pallor
Paresthesias
Pulse deficit
Poikilothermia
Paralysis

Endotracheal (ET) tube size based on size of cricoid ring rather than glottic opening because narrowest part of the child's airway is beyond the glottic opening.

Systolic BP = 80 mm Hg + 2 (age in years)

- Swelling
- Crepitus
- Laceration or open wound over extremity (open fracture)
- Temperature or pulse difference in one extremity compared to the other
- Loss of sensation in extremity
- Abnormal capillary refill

Treatment

- Reduction of fracture or dislocation under sedation.
- Splint extremity.
- Irrigation, antibiotics, and tetanus prophylaxis for open fractures.

Complications

- Compartment syndrome
- Neurovascular compromise
- Fat embolism
- Osteomyelitis
- Rhabdomyolysis (with prolonged crush injuries)
- Avascular necrosis
- Malunion
- Nonunion

► **PEDIATRIC TRAUMA**

Airway

- Smaller airway.
- Relatively large tongue.
- Anterior larynx.
- Narrowest portion is below the vocal cord at the level of the cricoid.

$$\text{ET tube size} = \frac{\text{age} + 16}{4}$$

$$\text{Depth} = \frac{\text{age (years)} + 12}{2}$$

$$\text{Depth} = \text{Internal diameter} \times 3$$

Breathing

Infants: 40 breaths/min
Children: 20 breaths/min
Tidal volume: 7 to 10 mL/kg

Circulation

Child blood volume 80 mL/kg:
- One fourth of blood volume must be lost before hypotension occurs.
- Hypovolemia causes tachycardia long before it causes hypotension.

	INFANT	CHILD	
Eye opening	Spontaneously	Spontaneously	4
	To speech	To speech	3
	To pain	To pain	2
	No response	No response	1
Best motor response	Spontaneous	Obeys commands	6
	Withdraws from touch	Localizes pain	5
	Withdraws from pain	Same	4
	Decorticate	Same	3
	Decerebrate	Same	2
	No response	Same	1
Best verbal response	Coos, babbles, smiles	Oriented	5
	Crying, irritable	Confused	4
	Cries, screams to pain	Inappropriate words	3
	Moans, grunts	Incomprehensible	2
	No response	No response	1
TOTAL			15

FIGURE 4-13. Pediatric Glasgow Coma Scale.

- Intraosseous cannulation < 6 years.
- Adequate urine output must be maintained:
  - Infant—2 mL/kg/hr
  - Child—1.5 mL/kg/hr
  - Adolescent—1 mL/kg/hr

Neurologic

- Separate Glasgow Coma Scale for infants and children (see Figure 4-13).
- CT head without contrast for any child with decreased level of consciousness or suspected loss of consciousness.
- Increased intracranial pressures may be masked in infants because cranium can expand via open fontanelles.

HIGH-YIELD FACTS

Trauma

HIGH-YIELD FACTS IN

Neurologic Emergencies

DEFINITION

Change in mental status is a term used to describe a spectrum of altered mentation including dementia, delirium, psychosis, and coma.

CAUSES

- Infection:
  - Meningitis
  - Neurosyphilis
  - Encephalitis
  - Urosepsis
  - CNS Lyme disease
  - Pneumonia
- Metabolic:
  - Uremia
  - B_{12} deficiency
  - Hepatic encephalopathy
  - Electrolyte imbalance
  - Hyper/hypoglycemia
  - Thyroid disease
  - Adrenal disease
- Neurological:
  - Cerebrovascular accident (CVA)
  - Central nervous system (CNS) space-occupying lesions (neoplasm)
  - Seizures/postictal state
  - CNS trauma
  - Hydrocephalus
- Vascular:
  - Hypertensive encephalopathy
  - Vasculitis
- Cardiopulmonary:
  - Hypoxic encephalopathy
  - Congestive heart failure (CHF)
  - Chronic obstructive pulmonary disease
  - Pulmonary embolism
- Toxic:
  - Drug overdose
  - Alcohol withdrawal
- Respiratory:
  - CO_2 retention
- Inflammatory/autoimmune:
  - Paraneoplastic syndrome, neurosarcoidosis, lupus, Hashimoto's encephalopathy
- Environmental:
  - Carbon monoxide exposure
  - Hypo/hyperthermia

HIGH-YIELD FACTS

Neurologic Emergencies

▶ DELIRIUM

DEFINITION

- Impairment of brain function secondary to another disease state.
- Delirium is usually transient in nature, reversing with removal or treatment of underlying cause.
- Patients with structural brain damage as cause of delirium may progress to chronic dementia.

CLINICAL PRESENTATION

- Patients usually have difficulty in focusing or sustaining attention.
- Clinical course is usually fluctuating, waxing and waning.
- Onset is usually rapid, from days to weeks.
- Symptoms usually worsen at night.
- Some patients may experience hallucinations, usually visual in nature.
- Patients usually have clinical signs and symptoms suggestive of underlying cause.

DIAGNOSIS

- Head CT and labs to identify underlying cause.

TREATMENT

- Coma cocktail: Thiamine, glucose, naloxone, oxygen.
- Sedation as needed for patient comfort.
- Treat underlying cause.

Treatment of delirium:
CAST
Cocktail
Admission
Sedation
Tomography (CT)

▶ DEMENTIA

DEFINITION

- A chronic, progressive decline in mental capacity that interferes with a patient's normal psychosocial activity.
- The identification of *reversible dementia* is key because progression can be halted.

NONREVERSIBLE CAUSES

- Degenerative:
  - Alzheimer's disease
  - Parkinson's disease
- Vascular:
  - Multiple infarcts
  - Subarachnoid hemorrhage (SAH)
  - Anoxic brain damage

CLINICAL PRESENTATION

- Impairment is gradual and progressive.
- Attention is usually normal, without waxing or waning of consciousness.
- Distant memory is usually preserved.

Reversible causes of dementia: **DEMENTIA**
Drugs
Electrolyte disturbances, eye and ear problems
Metabolic abnormalities (uremia, thyroid)
Emotional problems (psych)
Neoplasm, nutritional (vitamin deficiency)
Trauma
Infection, inflammation (lupus)
Alcohol

DIAGNOSIS

Identify *reversible* causes of dementia.

TREATMENT

- Address *reversible* causes of dementia.
- Supportive environmental, psychosocial interventions.

▶ DIFFERENTIATING DELIRIUM, DEMENTIA, AND PSYCHOSIS

See Table 5-1.

TABLE 5-1. Differentiating Delirium, Dementia, and Psychosis

MANIFESTATIONS	DELIRIUM	DEMENTIA	PSYCHOSIS
Onset	Sudden	Insidious	Sudden
Duration	Days to weeks	Permanent	Years (history)
Arousal level	Fluctuating	Normal	Normal
Attention	Poorly maintained	Maintained	Varies
Memory	Usually not intact	Distant memory intact	Varies
Hallucinations	Usually visual	Usually absent	Usually auditory
Delusions	Transient	Absent	Sustained
Thought process	Poorly organized	Varies with degree	Varies

▶ COMA

DEFINITION

Diffuse brain failure leading to impaired consciousness.

MANAGEMENT

- ABCs:
    - **A**irway—intubate if necessary to protect airway.
    - **B**reathing—oxygen, oral airway.
    - **C**irculation—intravenous (IV) access, blood pressure.
    - **C-spine**—cervical collar unless absolutely sure no history of trauma.
- **Vitals**—temperature, oxygen saturation (fifth vital sign) frequent re-assessment.
- **Electrocardiogram (ECG)/cardiac monitor**—arrhythmias, myocardial infarction (MI).

Causes of coma:
AEIOU TIPS
Alcohol
Encephalopathy, endocrine (thyroid disease, etc.), electrolyte abnormality
Insulin-dependent diabetes
Opiates, oxygen deprivation
Uremia

Trauma, temperature
Infection
Psychosis, porphyria
Space-occupying lesion, stroke, SAH, shock

- **History:**
  - From patient, family member, bystander, or old chart.
  - Past medical history.
  - Past psychiatric history.
  - Medications.
  - Social history (drug or alcohol use).
- **Physical exam:**
  - General exam:
    - Check for signs of trauma.
    - Glasgow Coma Scale.
    - Respiratory pattern:
      - Cheyne–Stokes: Periodic fluctuations of respiratory rate and depth suggest CNS pathology.
  - Ocular exam:
    - Pupillary function: If pupils are **reactive** to light bilaterally, midbrain is probably intact:
      - **Pinpoint pupils** suggest opioid toxicity or pontine dysfunction.
      - **Fixed and dilated pupils** suggest increased intracranial pressure (ICP) with possible herniation.
  - Ocular motions:
    - **Doll's eyes reflex:** Turn patient's head quickly to one side and observe eye movement. In a normal response, the eyes move in the opposite direction. Absence of motion suggests dysfunction in hemisphere or brain stem function. (Note that this reflex is also absent in a conscious patient.)
  - Neurological exam: Refer to section on neurological examination.

Do not perform doll's eye maneuver in the presence of head or neck trauma.

DIAGNOSIS

- Arterial blood gas: Acid–base disorders can help point to etiology (remember MUDPILES).
- Routine labs: Look for infection or electrolyte abnormalities.
- Toxicology screen: Look for drugs and alcohol.
- X-rays: C-spine in suspected cases of trauma.
- Head CT: Look for intracranial pathology.
- Lumbar puncture (LP): Look for SAH or infection. Remember, CT before LP for mass lesion/cerebral edema/hydrocephalus.

TREATMENT

Coma cocktail: **DON'T**
Dextrose (1 amp)
Oxygen
Naloxone
' (To remind you to give thiamine before dextrose)
Thiamine

- Coma cocktail.
- Supportive care.
- Monitoring (cardiac, oxygen saturation).
- Identify specific cause and apply appropriate treatment.
- Appropriate specialty consult as deemed necessary.

▶ STROKE

DEFINITIONS

- **CVAs:** Neurologic deficit caused by a disruption of blood flow to the brain.
- **Transient ischemic attacks (TIAs):** Neurological deficits that resolve within 24 hours.

- **Stroke in evolution:** Neurological deficits that fluctuate or worsen over time.
- **Completed stroke:** Neurological deficits that have remained stable for over 24 hours.

ANATOMY

Anterior Circulation
- Originates from the carotid system, then leads to anterior and middle cerebral artery.
- Supplies blood to the eye, the frontal and parietal lobes, and majority of the temporal area.

Posterior Circulation
- Originates from the vertebral arteries, then forms the basilar artery, cerebellar arteries, then the posterior cerebral artery (PCA).
- Supplies the brain stem, ears, cerebellum, occipital cortex, and parts of the temporal lobe.

GENERAL CLASSIFICATION

Ischemic Stroke (Figures 5-1 and 5-2)
- 80% of all strokes
- Thrombotic stroke
- Embolic stroke
- Lacunar stroke
- Systemic hypoperfusion (results in "watershed" infarcts, boundary zones between middle anterior and posterior cerebral arteries; usually bilateral and symmetric)
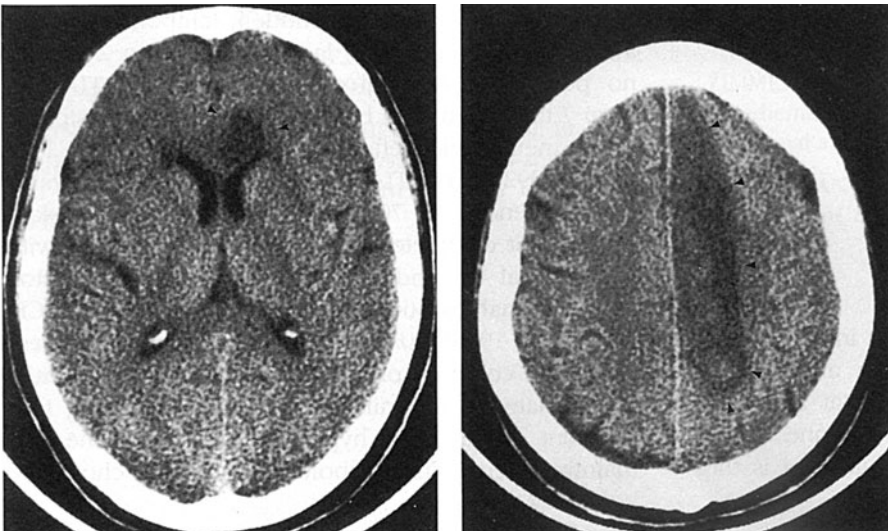
F I G U R E 5 - 1 . CT of ischemic stroke of the anterior cerebral artery (ACA).

Note the lesion is hypodense. (Reprinted, with permission, from Johnson MH. CT evaluation of the earliest signs of stroke. *The Radiologist* 1(4): 189–199, 1994.)
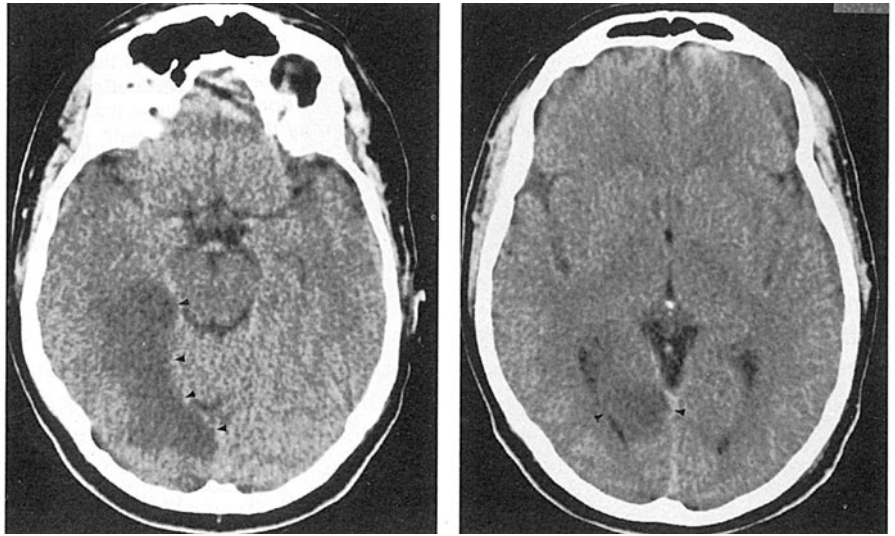
FIGURE 5-2. CT of ischemic stroke of the posterior cerebral artery (PCA).

Note the lesion is hypodense. (Reprinted, with permission, from Johnson MH. CT evaluation of the earliest signs of stroke. *The Radiologist* 1(4): 189–199, 1994.)

Hemorrhagic Stroke (Figure 5-3)
- 20% of all strokes
- Intracerebral hemorrhage
- SAH

SPECIFIC CLASSIFICATION

See Table 5-2.

Thrombotic Stroke
- Risk factors:
  - Atherosclerosis (most common cause)
  - Hypertension
  - Hyperlipidemia
  - Vasculitis
  - Often preceded by TIA
- Pathophysiology:
  - Vessel narrowing and occlusion secondary to plaque formation

Embolic Stroke
- Risk factors:
  - Atrial fibrillation
  - Dilated cardiomyopathy
  - Recent MI
  - Endocarditis
  - IV drug abuse
  - Smoking
  - Hyperlipidemia
- Pathophysiology:
  - Occlusion from intravascular material (clot, air bubble, fat, etc.) from distal sites.

FIGURE 5-3. **Hemorrhagic stroke due to hypertension.**

Note the lesion is hyperdense. Note bleeding into the ventricles. Effacement of sulci suggests edema. Mild mass effect is also present. (Reprinted, with permission, from Lee SH, Zimmerman RA, Rao KC. *Cranial MRI and CT*, 4th ed. New York: McGraw-Hill, 1999.)

- Sources of emboli:
  - Most common sources of emboli are from heart or ruptured plaque from major vessels.
  - Dislodged vegetation from cardiac valves (fibrin clots, septic vegetations, etc.).
  - Dislodged mural thrombi from atrial fibrillation, dilated cardiomyopathy, or recent MI.

Lacunar Stroke
- Risk factors: Chronic hypertension, diabetes
- Pathophysiology: Occlusion of small penetrating arteries

TABLE 5-2. **Differentiating Stroke Types**

	THROMBOTIC	**EMBOLIC**	**HEMORRHAGIC**
Timing	Upon awaking	Any time	Any time
Onset	Gradual, evolves	Sudden	Sudden
Associated symptoms	Prior TIA with similar symptoms	Palpitations Prior TIA with different symptoms	Severe, sudden headache Nausea, vomiting Stiff neck Coma Seizure

Intracerebral Hemorrhage
- Risk factors:
  - Age
  - History of prior stroke
  - Smoking
  - Hypertension
  - Anticoagulation (e.g., warfarin therapy)
  - Cerebral amyloidosis
  - Cocaine use
- Pathophysiology:
  - Vessel rupture with bleeding into brain parenchyma, causing increased ICP

SAH
- Risk factors:
  - Ruptured berry aneurysms
  - Arteriovenous malformations
- Pathophysiology:
  - Vessel rupture with blood leaking into subarachnoid space
  - Usually occurs at bifurcation of vessels

PHYSICAL EXAM

- Vitals:
  - Cushing reflex: Hypertension, bradycardia, and abnormal breathing could represent an increase in ICP.
- Eyes:
  - Do funduscopic exam.
  - Papilledema is a sign of increased ICP.
  - Subhyaloid hemorrhage is pathognomonic for SAH.
  - Note pupillary function and extraocular movements.
- Neurological exam: Localize lesion and differentiate between ischemic and hemorrhagic CVA.

LOCALIZATION

Signs of lacunar strokes:
DAMS
- **D**ysarthria—clumsy hand: Slurred speech with weak, clumsy hands
- **A**taxic hemiparesis: Ataxia with leg weakness
- Pure **M**otor hemiplegia: Motor hemiplegia without sensory changes
- Pure **S**ensory stroke: Sensory deficits of face, arms, legs without motor deficits

- Middle cerebral artery:
  - Most common
  - Contralateral weakness and numbness of arms greater than legs
  - Aphasia
  - Homonymous hemianopsia: Loss of vision on right or left side of both eyes
- ACA: Contralateral weakness of legs greater than arms
- PCA:
  - Vision changes
  - Sensory changes
  - Usually have subtle presentations
- Small penetrating arteries: Lacunar strokes present as "DAMS"
- Vertebrobasilar artery:
  - Syncope
  - Weakness
  - Cranial nerve changes
  - **Crossed findings** (ipsilateral cranial nerve changes with contralateral motor weakness)
  - Ataxia

- Cerebellar arteries:
  - **Central vertigo**
  - Headache
  - Nausea and vomiting
  - Loss of posture (inability to sit or stand without support)

DIAGNOSIS

- Routine labs
- ECG
- CT scan of head:
  - Helps differentiate ischemic from hemorrhagic CVA.
  - CT can be negative in ischemic strokes for 12 hours and may remain equivocal for small strokes.
  - Posterior fossa (brain stem and cerebellum) not as well visualized on CT.
  - Detects 1-cm and larger intracerebral hemorrhage (acute bleed looks white on CT scan).
  - Detects SAH 95% of time.
- Magnetic resonance imaging (MRI)
  - Can detect subtle ischemic infarcts, particularly diffusion-weighted sequences.
  - Good study for brain stem or cerebellar lesions.
  - Availability of MRI can be a limiting factor in the acute setting.

*Vertebrobasilar strokes present with **3 Ds:*** **D**izziness (vertigo) **D**iplopia **D**ysphasia

EMERGENCY DEPARTMENT MANAGEMENT OF ISCHEMIC STROKE

- ABCs: Give supplemental O_2.
- Blood pressure (BP) control:
  - Since autoregulation is lost in ishemic brain, perfusion is directly dependent on the cerebral perfusion pressure (CPP), which in turn is dependent on the mean arterial pressure (MAP).
  - CPP = MAP − ICP.
  - MAP = ⅓ SBP + ⅔ DBP. Ideally want MAP ≥ 90.
  - Use pressors for MAP < 60 or SBP < 90.
  - Do not treat MAP < 130.
- Serum glucose control:
  - Hyperglycemia provides more substrate for anaerobic metabolism, worsening acidosis.
  - It is recommended to keep serum glucose < 150 mg/dL.
  - Use sliding scale regular insulin for glucose > 300 mg/dL.
- Temperature control:
  - Hyperthermia increases oxygen demand when the ischemic brain is already hypoxic.
  - Administer acetaminophen for fever.
- Thrombolytic therapy:
  - Only approved specific therapy (Class I within 3 hours of symptom onset).
  - Very specific inclusion criteria: Need a clinical diagnosis of stroke with National Institute of Health stroke scale score < 22. Stroke needs to be moderate; massive strokes and very mild strokes are not eligible.
  - Contraindications include SBP > 180, history of hemorrhagic stroke, any stroke within past year, suspected aortic dissection, active bleeding.

Identification of strokes involving the cerebellum is important due to risk of edema and increased pressure to brain stem.

Optimization for ischemic stroke:
- Supplemental O_2
- BP: MAP ≥ 60, SBP ≥ 90
- Serum glucose: < 150
- Normal temperature
- Screen for thrombolytics
- For hypertension, do not treat until MAP > 130.

EMERGENCY DEPARTMENT MANAGEMENT OF HEMORRHAGE STROKE

- ABCs: Consider early intubation.
- BP control:
  - Keep MAP < 130 and SBP < 220.
  - Agents of choice: Labetalol, nitroprusside.
- Control seizures with lorazepam acutely, followed by phenytoin.
- Control cerebral edema with dexamethasone.
- Control elevated ICP with mannitol, hyperventilation (transiently), and by elevating the head of the bed.
- Nimodipine (Ca^{2+} channel blocker) to decrease vasospasm in SAH.
- Prompt neurosurgical evaluation.

PREVENTION

- Antiplatelet therapy for ischemic strokes
- Anticoagulation with warfarin for embolic strokes
- Smoking cessation
- Strict hypertension control
- Control of hyperlipidemia
- Control of diabetes

> **NEUROLOGICAL EXAMINATION**

Mental Status

- Orientation: Person (×1), place (×2), time (×3).
- Level of consciousness: Awake, lethargic, comatose.
- Affect: Appropriate, alert, confused.
- Speech: Presence of dysphasia or dysarthria, appropriateness of speech, and language content.

Definitions of Expression

- *Dysarthria:* Difficulty in speech secondary to muscle weakness or paralysis.
- *Aphasia:* Disorder in the comprehension or expression of language.
- *Expressive aphasia:* Difficulty in finding words or expression of language without a defect in comprehension.
- *Receptive aphasia:* Problems in understanding words or written language.
- *Fluent aphasia:* Normal rate, meter, quantity of speech, usually abnormal content, poor understanding.
- *Nonfluent aphasia:* Diminished quantity of speech, better understanding, more word-finding difficulties.

> **CRANIAL NERVES**

CN I: Olfactory

Distinguishing two odors (e.g., coffee and garlic powder).

Up to 25% of patients with a hemorrhagic stroke will seize in the first 72 hours.

Antiplatelet agents: **CAT**
Clopidogrel
Aspirin
Ticlopidine

Fracture of cribriform plate can disrupt odor sensation.

CN II: Optic

Test visual fields of each eye, visual acuity, funduscopy, pupillary reactions.

CN III: Oculomotor

Check pupillary reactions, ptosis, and extraocular movements.

CN IV: Trochlear

Controls **S**uperior **O**blique (**SO4**); predominant movement is down and in.

CN V: Trigeminal

- Sensation to face (V1, V2, and V3).
- Check motor function of muscles of mastication (masseter, pterygoids, temporalis).

CN VI: Abducens

Controls **L**ateral **R**ectus (**LR6**), abducts the eye.

CN VII: Facial

- Check motor function of face—ask patients to puff out cheeks (buccinator muscle), smile, close eyes, and raise eyebrows.
- Check for facial symmetry.

Peripheral versus Central CN VII Palsy
In patients with a seventh nerve palsy, ask patient to raise eyebrows and examine forehead for symmetry:
- Peripheral lesion: Asymmetrical or absent wrinkles on the side of the lesion.
- Central lesion: Symmetrical wrinkles due to crossed fibers (innervation from both sides of the cerebral hemispheres).

CN VIII: Vestibulocochlear

- Check auditory acuity.
- Look for nystagmus (onset, direction, fatigability).

CN IX: Glossopharyngeal

- Check gag reflex (shared with CN X).
- Check taste on posterior one third of tongue.

CN X: Vagus

Check for uvula deviation.

Corneal reflex: Touch side of cornea with cotton wisp. A blink is a normal response indicating normal reflex arc. This reflex requires V1 division of trigeminal nerve (V) for sensory input and motor response from intact facial nerve (VII).

If vertical nystagmus is found in a patient, a central lesion within the brain stem or cerebellum must be ruled out.

CN XI: Accessory

Check trapezius and sternocleidomastoid muscles. Ask patient to shrug shoulders, turn head against resistance.

CN XII: Hypoglossal

Check for tongue deviation. Deviation indicates ipsilateral lesion.

▶ **MOTOR SYSTEM**

Posturing

- **Flexor posture:** Abnormal flexion of the arm and wrist, with extension of the leg (lesion above red nucleus).
- **Extensor posture:** Abnormal extension of both the arms and legs (lesion below red nucleus).

Strength

5 = Normal strength
4 = Able to move against resistance
3 = Movement against gravity
2 = Movement with gravity eliminated
1 = Flickers of motion
0 = No movement

Pronator Drift

- Have patient hold arms outstretched, palms upward, with eyes closed.
- Pronation of the hand with downward drift of the arms is considered an abnormal sign, typically an upper motor neuron lesion.
- Normal strength and proprioception is required to prevent arms from drifting.

▶ **SENSORY SYSTEM**

Symmetry

- Right versus left
- Upper versus lower

Sensation

- Touch—large and small fiber
- Pain—small fiber
- Temperature—small fiber
- Position—large fiber
- Vibratory sensation—large fiber

Level of Reactivity

- Hyperactive reflexes are associated with upper motor neuron lesions.
- Hypoactive reflexes are associated with lower motor neuron lesions or acute spinal injury.

Symmetry

Visualize spinal roots starting from feet up to arms.

Spinal Roots	Reflex
S1–2	Ankle
L3–4	Knee
C5–6	Biceps
C7–8	Triceps

Cerebellar Tests

- Finger to nose.
- Heel to shin.
- Check rapid repetitive motions (dysdiadokinesis).
- Gait.

► HEADACHE

Primary Headache Syndromes

- Migraine
- Tension headache
- Cluster headache

Secondary Causes

- CNS infection:
  - Meningitis
  - Encephalitis
  - Cerebral abscess/tumor
- Non-CNS infection:
  - Sinusitis (overrated)
  - Fever
  - Herpes zoster
  - Ear infections
  - Dental infections
- Vascular:
  - SAH
  - Subdural hematoma
  - Epidural hematoma
  - Intracerebral hemorrhage
  - Temporal arteritis
  - Carotid or vertebral artery dissection

HIGH-YIELD FACTS

Neurologic Emergencies

- Ophthalmologic:
  - Glaucoma
  - Iritis
  - Optic neuritis
- Toxic/metabolic:
  - Carbon monoxide (CO) poisoning
  - Nitrates and nitrides
  - Hypoglycemia
  - Hypoxia
  - Hypercapnia
  - Caffeine withdrawal
  - Withdrawal from chronic analgesics (rebound headache)
  - Malignant hypertension
  - Preeclampsia
  - Pseudotumor cerebri
  - Post LP/CSF leak
  - Hypertension

DIAGNOSTIC APPROACH

- Differentiate between primary headache syndromes and secondary causes of headache.
- Recognize critical life-threatening causes of headache.
- Treat primary causes of headache.

HISTORY

- Pattern:
  - First episode or chronic in nature.
  - In chronic headache, assess duration, severity, or associated symptoms.
- Onset: Gradual versus sudden or severe
- Location:
  - Migraines typically unilateral
  - Tension headache usually bilateral
  - SAH headache usually occipital
- Associated symptoms:
  - Syncope
  - Changes in mental status
  - Fever
  - Vision changes
  - Seizures
  - Neck pain and stiffness
  - History of head trauma
- Past medical history:
  - Hypertension
  - CVA
  - Migraines
  - Human immunodeficiency virus (HIV)
- Medications:
  - Nitrates
  - Analgesics
  - Anticonvulsants

Consider CO poisoning if similar symptoms of headache, nausea, vomiting in other members of household (more common in winter/cold climates due to space heaters).

- Family history:
  - Migraines
  - SAH
- Age:
  - Look for secondary causes in elderly.

PHYSICAL EXAM

- Vitals: Fever, BP, Cushing reflex (systemic response to $\uparrow$ ICP, HTN, tachycardia, bradypnea).
- Eyes: Funduscopic exam may reveal absence of venous pulsations or papilledema, suggesting increased ICP.
- Neurological exam: Refer to section on neurological exam.

LABS/RADIOLOGY

- Routine labs:
  - Check glucose.
  - Erythrocyte sedimentation rate (ESR): Positive if over 50 mm/hr.
- CT scan of the brain:
  - Helps to identify:
    - Mass lesions
    - Midline shift of intracranial contents
    - CNS bleeds
    - Increased ICP (increased size of ventricles, cerebral edema)
  - Contrast is useful for:
    - Cerebral toxoplasmosis
    - Small brain mass
    - Intracranial abscess
- LP indications (do not perform LP until CT is negative for mass lesion or obstructive hydrocephalus):
  - Meningitis
  - Encephalitis
  - SAH

Remember LP required in patients suspected of SAH with negative CT of head.

▶ MIGRAINE HEADACHE

EPIDEMIOLOGY

- Onset in adolescence.
- Increased frequency in females.

PATHOPHYSIOLOGY

Not completely understood; involves:
- Trigeminal innervation of meningeal vasculature.
- Sterile inflammatory changes in meningeal vessel walls.
- In aura, posterior to anterior spreading depression of brain metabolism.

SIGNS AND SYMPTOMS

- Frequently associated with aura (should not last more than 1 hour). Aura can be any neurologic symptom; paresthesias are common.

- May have visual auras (scintillating scotoma, or flashing lights).
- Slow onset.
- Last 4 to 72 hours.
- Worsen with exertion.
- Unilateral and pulsating.
- Nausea, vomiting, photophobia, phonophobia, osmophobia.
- Neurological deficits (history of similar deficits in prior episodes). Focal deficits contraindicate treatment with triptans or dihydroergotamine (DHE).

PROPHYLAXIS

- Tricyclics (amitriptyline, nortriptyline)
- Gabapentin
- Valproic acid
- Topiramate
- Beta blockers (propranolol)
- Calcium channel blockers

TREATMENT

- Whatever worked in the past
- Metoclopramide
- Compazine
- DHE (not with focal deficit)
- Nonsteroidal anti-inflammatory drugs (NSAIDs) such as ketorolac
- Opioid analgesia
- Triptans (not with focal deficit)

▶ CLUSTER HEADACHE

EPIDEMIOLOGY

- More common in men.
- Onset usually > 20 years old.

PATHOPHYSIOLOGY

Mechanism unknown.

SIGNS AND SYMPTOMS

- Short lived.
- Severe, unilateral, lasting up to 3 hours.
- Appear in clusters, multiple attacks in same time of day or month.
- Patients appear restless.
- Associated with ipsilateral conjunctival injection, lacrimation, nasal congestion, rhinorrhea, miosis, and ptosis.
- Precipitated/exacerbated by EtOH.

PROPHYLAXIS

- Verapamil
- Lithium

- Topiramate
- Valproic acid
- Gabapentin

TREATMENT

- 100% oxygen
- NSAIDs
- Steroids
- EtOH cessation

► TENSION HEADACHE

PATHOPHYSIOLOGY

Muscle tension has been theorized as the causative factor.

SIGNS AND SYMPTOMS

- Bilateral
- Nonpulsating
- Not worsened with exertion
- Usually no nausea or vomiting
- Associated neck or back pain

PROPHYLAXIS

- Tricyclics
- Gabapentin

TREATMENT

NSAIDs

► SUBARACHNOID HEMORRHAGE (SAH)

CAUSES

- Arteriovenous malformation (AVM)
- Rupture of aneurysm
- Idiopathic
- Trauma

Head CT can miss up to 15% of SAH; therefore, an LP must be done to look for blood or xanthochromia if CT is negative.

SIGNS AND SYMPTOMS

- Sudden, severe occipital headache.
- Nausea, vomiting.
- CT of head detects up to 95% (see Figure 5-4).
- If CT negative, perform LP to check cerebrospinal fluid (CSF) for blood or xanthochromia (strive to avoid traumatic tap). Xanthochromia requires at least 6 hours to develop; may be negative before that time.
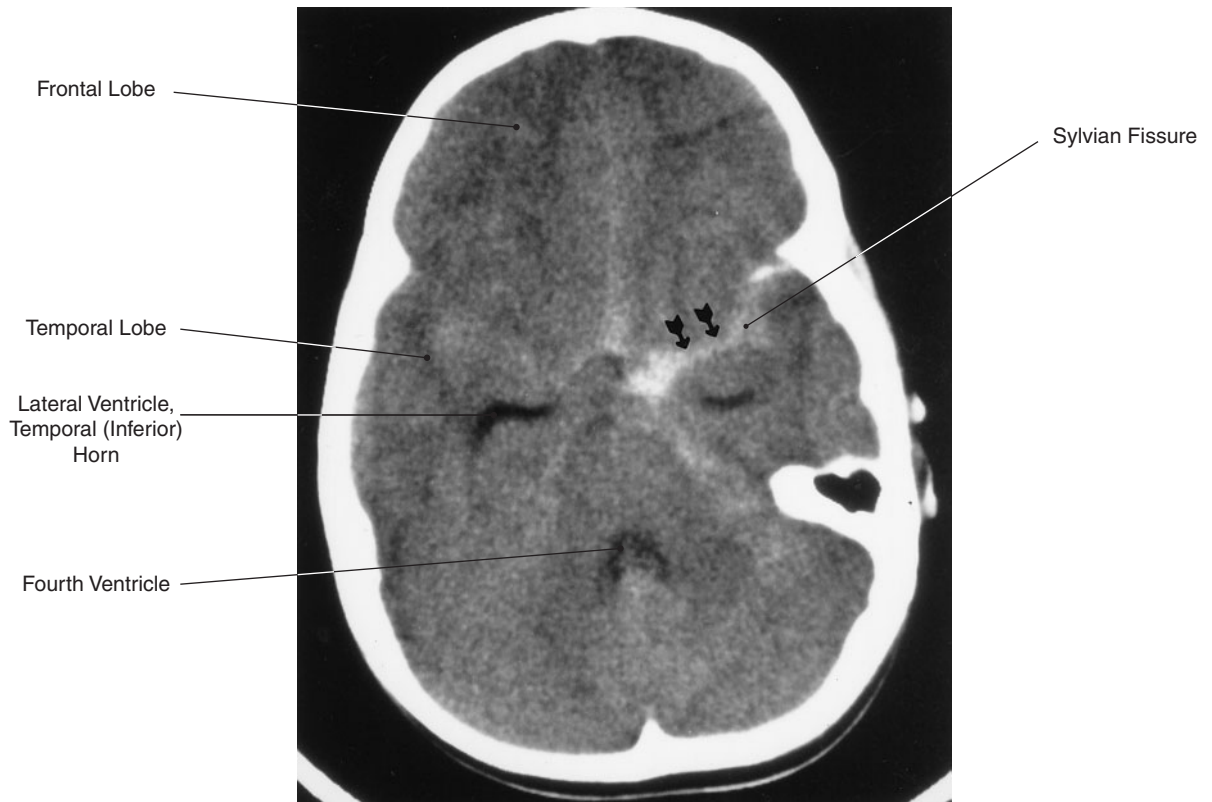
FIGURE 5-4. SAH.

Arrows indicate fresh blood in the Sylvian fissure. (Reproduced, with premission, from Afifi AK, Bergman RA. *Functional Neuroanatomy: Text and Atlas.* New York: McGraw-Hill, 1998: 554.)

TREATMENT

- To prevent vasospasm: Nimodipine 60 mg PO q 4 hours × 21 days, start within 96 hours of SAH.
- For cerebral edema: Dexamethasone 10 mg IV × 1.
- Prevent hypo- or hypertension.
- To prevent seizures: Phenytoin 15 to 18 mg/kg loading dose.
- Elevate head of the bed to 30° if C-spine is not a concern.
- Admit patients for observation.
- Unless traumatic, evaluate for AVM or aneurysm with angiogram.

> Sudden, severe headache described as "worst headache of my life" should be considered SAH until proven otherwise (do CT + LP).

▶ **TEMPORAL ARTERITIS**

EPIDEMIOLOGY

More common in women age > 50.

PATHOPHYSIOLOGY

Systemic panarteritis affecting temporal artery.

SIGNS AND SYMPTOMS

- Severe, throbbing in nature
- Frontal headache
- Tender temporal artery

DIAGNOSIS

- ESR > 50 mm/hr.
- Temporal artery biopsy showing giant cells is definitive—do not await biopsy results before initiating treatment.

TREATMENT

- Prednisone.
- If left untreated, can lead to **vision loss.**

▶ SUBDURAL HEMATOMA

- History of head trauma (see Trauma chapter).
- Disruption of bridging vessels intracranially.
- High-risk patients include alcoholics, elderly, and patients on anticoagulants.
- Can present like transient ischemic attack (TIA).

▶ CEREBRAL ISCHEMIA/INFARCT

Rarely produces headaches.

▶ INTRACEREBRAL HEMORRHAGE

- Commonly produces headache.
- Neurologic exam usually abnormal.
- Refer to section on CVA for more detail.

▶ BRAIN TUMOR

- Commonly presents with insidious headache.
- Headache positional and worse in morning.
- Neurologic abnormalities usually present on physical exam.

▶ PSEUDOTUMOR CEREBRI (IDIOPATHIC INTRACRANIAL HYPERTENSION)

EPIDEMIOLOGY

- Occur in young, obese females.
- History of headaches in past.

ETIOLOGY

Unknown; should exclude venous sinus thrombosis, antibiotics, vitamin A intoxication, endocrine dysfunction, chronic meningitis.

SIGNS AND SYMPTOMS

- Papilledema
- Absent venous pulsations on funduscopic exam
- Headache, nausea, vomiting
- Visual loss

DIAGNOSIS

- Usually normal CT of head.
- LP reveals elevated CSF opening pressures.
- Response of headachee and visual symptoms to large-volume LP.
- Visual field testing.

TREATMENT

- Acetazolamide +/– other diuretic
- LP to remove CSF
- Optic nerve fenestration
- CSF shunt in refractory cases

▶ **CAROTID OR VERTEBRAL ARTERY DISSECTION**

- Idiopathic.
- Secondary to trauma.
- Unilateral neck pain with headache.
- Diagnosed via angiography (conventional or magnetic resonance angiography [MRA]).

▶ **POST LP HEADACHE**

- Occurs within 24 to 48 hours post LP.
- Headache secondary to persistent CSF leak.
- Mild cases treated with analgesics.
- Severe cases treated with blood patch (epidural injection of patient's blood to patch leak).

▶ **VERTIGO AND DIZZINESS**

Definitions

- **Dizziness** is a nonspecific term that should be clarified. It can be used to describe true vertigo or other conditions such as syncope, presyncope, light-headedness, or weakness.
- **Vertigo** is the perception of movement when there is no movement. The patient typically describes the room as spinning or the sensation of falling.

- **Nystagmus** is the rhythmic movement of eyes with two components (fast and slow). The direction of nystagmus is named by its fast component. Activation of the semicircular canals causes the slow component of the nystagmus to move away from the stimulus. The fast component of nystagmus is the reflex counter movement back to the desired direction of gaze by the cortex.

Distinguishing Peripheral from Central Vertigo

See Table 5-3.

Peripheral Vertigo

Causes
- Benign paroxysmal positional vertigo
- Ménière's disease
- Vestibular neuronitis
- Labyrinthitis
- Ototoxicity (drugs)
- Eighth (vestibulocochlear) CN lesion
- Post-traumatic vertigo
- Middle ear disease
- Cerebellopontine angle tumors

> The approach in the emergency department is to first distinguish between *true* vertigo and syncope, presyncope or weakness. Once *true* vertigo is differentiated, one must distinguish between *central* and *peripheral* vertigo. Consider all clinical information, including age and comorbidities.

TABLE 5-3. Peripheral versus Central Vertigo

	PERIPHERAL VERTIGO	CENTRAL VERTIGO
Pathophysiology	Disorders affecting the vestibular apparatus or the eighth (vestibulocochlear) cranial nerve	Disorder affecting the brain stem or cerebellum
Severity	Intense	Less intense
Onset	Sudden	Slow, insidious
Pattern	Intermittent	Constant
Nausea/vomiting	Usually present	Usually absent
Positional (worsened by motion)	Usually	Usually not
Hearing changes or physical findings on ear exam	May be present	Usually absent
Focal neurologic findings	Absent	Usually present
Fatigability of symptoms	Yes	No
Nystagmus	Horizontal, vertical, rotary	Vertical

Benign Paroxysmal Positional Vertigo

EPIDEMIOLOGY

- Common
- Can occur at any age

PATHOPHYSIOLOGY

Transient vertigo precipitated by certain head motions.

SIGNS AND SYMPTOMS

- Sudden onset
- Nausea
- Worse in morning, fatigable
- Normal ear exam, no hearing changes

DIAGNOSIS

Dix–Hallpike maneuver:
- Have patient with eyes open go from sitting to a supine position with head rotated to the side you want to test.
- Positive test entails reproduction of vertigo and nystagmus that resolves within 1 minute.
- Not to be performed on patients with carotid bruits.

TREATMENT

- Antiemetics
- Antihistamines (meclizine)
- Benzodiazepines

Ménière's Disease

EPIDEMIOLOGY

Occurs between the ages of 30 and 60.

PATHOPHYSIOLOGY

Etiology unknown; postulated that symptoms are due to extravasation of endolymph into the perilymphatic space. Excess production of endolymph is also known as *endolymphatic hydrops*.

SIGNS AND SYMPTOMS

- Deafness, tinnitus, vertigo.
- Nausea, vomiting, diaphoresis.
- Recurrent attacks.
- Deafness between attacks.
- Attacks occur several times a week to months.

TREATMENT

Symptomatic treatment with antihistamines, antivertigo and antiemetic agents, hydrochlorothiazide.

Ménière's triad: **DVT**
Deafness
Vertigo
Tinnitus

Labyrinthitis

DEFINITION

Infection of labyrinth.

SIGNS AND SYMPTOMS

- Hearing loss
- Peripheral vertigo
- Middle ear findings

DIAGNOSIS

Head CT or clinical.

TREATMENT

Symptomatic treatment with antihistamines, antivertigo and antiemetic agents.

Post-Traumatic Vertigo

PATHOPHYSIOLOGY

- Injury to labyrinth structures
- History of head trauma

SIGNS AND SYMPTOMS

- Peripheral vertigo
- Nausea, vomiting

DIAGNOSIS

CT of head to check for intracranial bleed or hematoma.

TREATMENT

Symptomatic treatment with antihistamines, antivertigo and antiemetic agents.

Vestibular Neuronitis

PATHOPHYSIOLOGY

Viral etiology.

SIGNS AND SYMPTOMS

- Sudden onset, lasts several days
- Upper respiratory tract infection

TREATMENT

Symptomatic treatment with antihistamines, antivertigo and antiemetic agents.

Neurologic Emergencies

Obtain neurology consult in all cases of central vertigo or cases in which you are unsure.

Other Causes of Central Vertigo

- Cerebellar hemorrhage or infarct
- Lateral medullary infarct **(Wallenberg syndrome)**
- Vertebrobasilar insufficiency
- Multiple sclerosis
- Neoplasm

OTOTOXICITY

Many drugs such as aminoglycosides, furosemide, and PCP cause ototoxicity.

HERPES ZOSTER OTICUS (RAMSAY HUNT SYNDROME)

Presents as deafness, facial nerve palsy, and vertigo with vesicles present in auditory canal.

CEREBELLOPONTINE ANGLE TUMORS

Present with multiple findings (cerebellar signs, ataxia, vertigo, and loss of corneal reflex).

CEREBELLAR HEMORRHAGE OR INFARCTION

- Acute vertigo
- Profound ataxia or inability to stand or sit without support
- Cerebellar findings
- Headache

WALLENBERG SYNDROME

- Occlusion of posterior inferior cerebellar artery
- Acute onset
- Nausea, vomiting
- Nystagmus
- Ipsilateral facial pain or numbness, Horner's syndrome
- Contralateral pain and temperature loss

VERTEBROBASILAR VASCULAR DISEASE

- Vertebrobasilar vascular insufficiency can produce symptoms of vertigo.
- Other findings include diplopia, dysphagia, dysarthria, ataxia, **crossed findings** (refer to section on CVA—localizing the lesion).

MULTIPLE SCLEROSIS (SEE FIGURE 5-5)

- Demyelinating disease that can also affect the brain stem, causing vertigo.
- May present with optic neuritis, ataxia, weakness, incontinence, facial pain, or paresthesias.
- Best clue is inability to explain multiple neurologic symptoms and deficits by a single lesion.
- CSF may reveal oligoclonal banding.
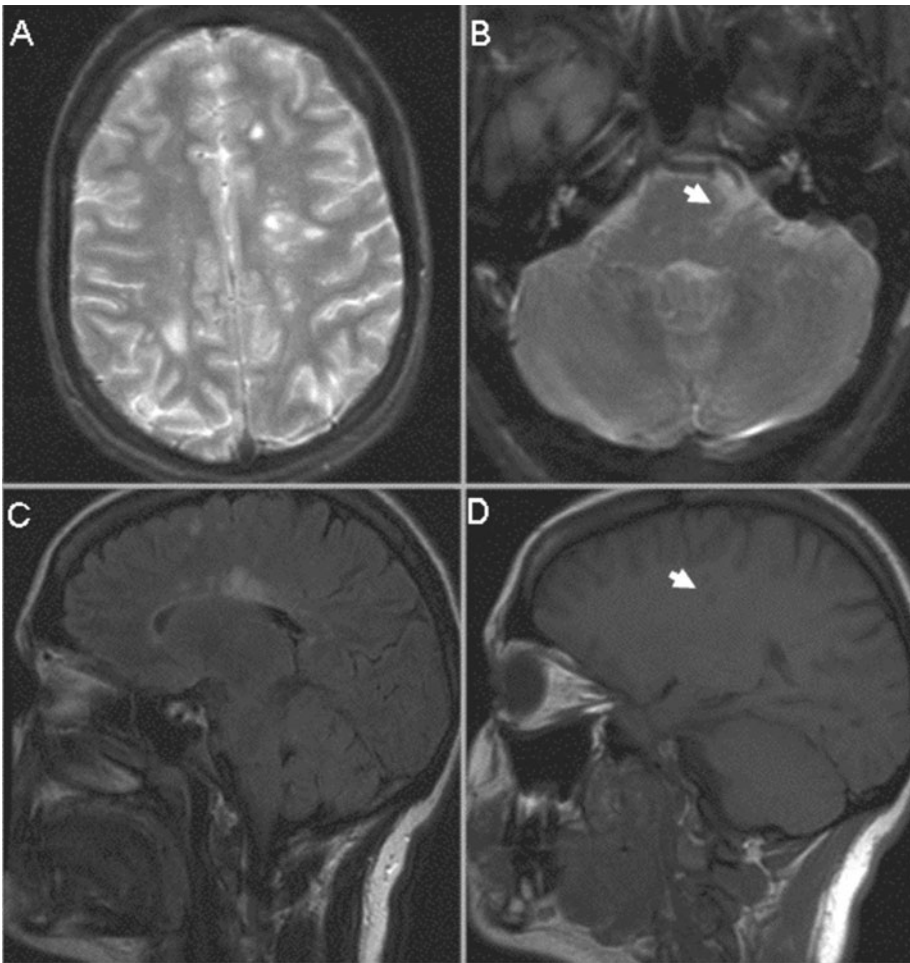
F I G U R E 5 - 5 . **MRI demonstrating findings associated with multiple sclerosis. Figure B shows Dawson's fingers. Figure D arrow shows black holes.**

Meningitis

DEFINITION

Inflammation of the membrane surrounding the brain and spinal cord.

CAUSES

- The majority of meningitides are caused by an infectious etiology, which varies according to age group (see Table 5-4).
- Noninfectious causes of meningitis include neoplasms and sarcoidosis.

SIGNS AND SYMPTOMS

Altered mental status, photophobia, headache, fever, meningeal signs (nuchal rigidity, Kernig and Brudzinski's signs).

	< 2 Months (Require Follow-up LP in 24–36 Hours)	2 Months to 50 Years	> 50 Years Debilitated or Immunocompromised
Organisms	■ Group B strep ■ *Listeria* ■ *Escherichia coli* ■ *Klebsiella* ■ *Enterobacter* ■ *Staphylococcus aureus* ■ *Haemophilus influenzae*	■ *Streptococcus pneumoniae* ■ *Neisseria meningitidis* ■ *H. influenzae*	■ *S. pneumoniae* ■ *Listeria* ■ Gram-negative bacteria
Treatment (IV)	■ Ampicillin 50 mg/kg q 8 h AND ■ Cefotaxime 50 mg/kg q 8 h ■ Generally considered good to treat early with steroids in any possible bacterial meningitis	■ Cefotaxime 2 g q 4–6 h OR ■ Ceftriaxone 2 g q 12 h ■ Vancomycin 15 mg/kg q 6 h (until sensitivity known)	■ Ampicillin 2 g q 4 h AND ■ Cefotaxime 2 g q 4–6 h OR ■ Ceftriaxone 2 g q 12 h
Penicillin-allergic Rx (IV)	■ TMP/SMZ 5mg/kg q 12 h AND ■ Vancomycin 15 mg/kg q 6 h	■ Vancomycin 15 mg/kg q 6 h AND ■ Gentamicin 2 mg/kg loading then 1.7 mg/kg q 8 h AND ■ Rifampin 10–20 mg/kg qd	

Kernig's sign:
Pain or resistance with passive extension of knee with hip flexed 90°.

Brudzinski's sign:
Passive flexion of neck causes flexion of the hips.

DIAGNOSIS

- Diagnosis made by LP: Obtain 5 tubes containing 1 to 2 mL of CSF.
- Make sure there is no risk of herniation prior to performing LP. (Head CT scan can support this.)
- CSF findings suggestive of bacterial meningitis include increased white blood cells with a high percentage of polymorphonuclear leukocytes, low glucose, and high protein.

TREATMENT

- Mainstay of treatment in adults is ceftriaxone, which has good CSF penetration.
- If resistance is an issue for *Streptococcus pneumoniae*, vancomycin or rifampin can be added to the regimen.
- Ampicillin should be added to any age group at risk for *Listeria monocytogenes*.

- Vancomycin and ceftazidime are used in post–head trauma patients, neurosurgical patients, or ventriculoperitoneal shunts.
- Antifungal agents should be considered in HIV⁺ and other immuno-compromised patients.

CNS Encephalitis

DEFINITION

Inflammation of the brain parenchyma secondary to infection.

CAUSES

Usually viral in origin.

SIGNS AND SYMPTOMS

- Abnormal behaviors and "personality changes"
- Seizures
- Headache
- Photophobia
- Focal neurologic findings
- Signs of peripheral disease:
    - Herpes—skin vesicles, rash
    - Rabies—animal bite
    - Arboviruses—bug bite

DIAGNOSIS

Primarily diagnosed via CSF culture or serology: Blood in the CSF is a non-specific clue but suggests herpes.

TREATMENT

- Mainly supportive
- Acyclovir for herpes

Brain Abscess

DEFINITION

A focal purulent cavity, covered by granulation tissue located in the brain.

CAUSES

Brain abscesses develop secondary to:
- Hematogenous spread
- Contiguous infections (sinuses, ears)
- Direct implantation via penetrating trauma or neurosurgery

SIGNS AND SYMPTOMS

- Headache
- Fever
- Focal neurologic findings
- Signs of primary infection
- History of trauma

Causes of encephalitis:
HEAR
Herpes
Epstein–Barr virus
Arboviruses
Rabies

DO NOT delay antibiotics for LP or head CT when meningitis is suspected.

DIAGNOSIS

CT scan of head with contrast (see Figure 5-6).

TREATMENT

Antibiotics tailored to suspected source of primary infection.

Guillain–Barré Syndrome

DEFINITION

- Ascending peripheral neuropathy
- Can affect all ages
- History of viral illness

CAUSES

Idiopathic

SIGNS AND SYMPTOMS

- Loss of deep tendon reflexes.
- Distal weakness greater than proximal (legs greater than arms).
- Weakness is symmetrical.
- Numbness or tingling of the extremities.
- Risk of respiratory failure.
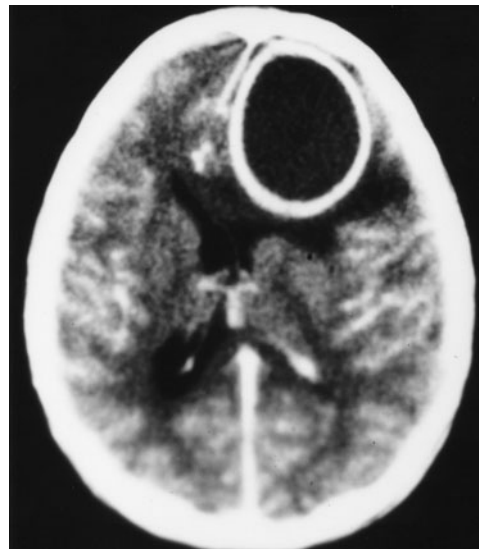
FIGURE 5-6. CT scan demonstrating brain abscess.

(Reproduced, with permission, from Schwartz SI, ed. *Principles of Surgery*, 7th ed. New York: McGraw-Hill, 1999: 1902.)

DIAGNOSIS

LP reveals increased CSF protein with a normal glucose and cell count.

TREATMENT

- Plasmapheresis.
- IV immunoglobulin.
- Intubate if there is respiratory compromise.

Myasthenia Gravis

DEFINITION

- Autoimmune disease of the neuromuscular junction.
- Affects old males and young females.

PATHOPHYSIOLOGY

- Acetylcholine receptor antibodies bind acetylcholine receptors, preventing binding of acetylcholine and subsequent muscular stimulation.
- Failure of neuromuscular conduction causes weakness.
- Association with thymoma.

SIGNS AND SYMPTOMS

- Generalized weakness.
- Usually proximal weakness affected more than distal weakness.
- Weakness relieved with rest.
- Ptosis and diplopia usually present.
- Symptoms may fluctuate, but usually worsen as the day progresses.
- Overuse of specific muscle groups can cause specific weakness of those muscle groups.

DIAGNOSIS

- Edrophonium test: Edrophonium is an anticholinesterase that prevents the breakdown of acetylcholine. Increased level of acetylcholine overcomes the receptor blockage from autoantibodies. There is rapid return of muscle strength. Since the duration is short acting, this is only used as a diagnostic modality.
- Myasthenia gravis can be diagnosed by detection of acetylcholine receptor antibodies in the serum.
- Repetitive stimulation on nerve conduction studies, single-fiber electromyocardiogram (EMG) for increased jitter.

TREATMENT

- Anticholinesterase
- Plasma exchange
- Immunoglobulins
- Respiratory support (intubate as needed)
- Thymectomy (with or without thymoma or thymic hyperplasia)

A 37-year-old female presents with severe weakness of respiratory muscles, diplopia, ptosis, and proximal muscle weakness. *Think: Myasthenic crisis.*

DEFINITION

Abnormal electrical discharge of neurons causing a clinical episode of neurologic dysfunction.

CLASSIFICATION

See Table 5-5.

T A B L E 5 - 5 . Classification of Seizures

TYPE	DESCRIPTION
Generalized	All generalized seizures involve loss of consciousness.
Tonic–clonic (grand mal)	Loss of consciousness immediately followed by tonic (rigid) contraction of muscles, then clonic (jerking) contraction. Patients may be cyanotic or apneic. Urinary incontinence may occur. Postictal period: Confusion, fatigue, or hypersomnolence following seizure; tongue biting may occur.
Absence (petit mal) (usually less than 30 seconds)	Loss of consciousness without loss of postural tone; eye flutter common. Patients do not respond to verbal stimuli, nor do they lose continence. No postictal period.
Myoclonic	Loss of consciousness with brief muscular contractions.
Clonic	Loss of consciousness with repetitive clonic jerks.
Tonic	Loss of consciousness with sustained, prolonged contraction of body.
Atonic	Loss of consciousness with sudden loss of postural tone ("drop attacks").
Partial (focal)	Usually involve focal area of abnormal electrical discharge in cerebral cortex. Partial seizures may progress to generalized seizure ("secondary generalization").
Simple partial	Abnormal focal neurological discharge in which consciousness remains intact.
Complex partial (usually 1–2 minutes)	Frequently of temporal lobe origin. Consciousness is altered. Patient usually has an abrupt termination of ongoing motor activity ("staring spell"). Postictal period.
Aura	Simple partial seizure that becomes more obviously a seizure as it spreads; patients interpret it as a warning rather than a seizure.

MANAGEMENT

Position
- Patients having a seizure should be rolled to a semiprone position to allow gravity to pull the tongue and secretions out of the airway. The head should be aligned with the body, and nothing should be put in the mouth.

ABCs
- Airway: Maintain adequate airway with nasal trumpet.
- Breathing: Administer oxygen. If properly positioned, cyanosis and apnea are rare.
- Circulation: Obtain IV access.

History
- Important history can be obtained from bystanders or witnesses.
- Include **syncope** as part of your differential diagnosis.
- Seizures can cause loss of bladder control.
- Differentiate between partial and generalized seizure (ask patient if they can recall event).
- First seizure or known seizure history.
- Baseline seizure history (frequency and last seizure episode).
- Recent history of trauma.
- Consider factors that may lower seizure threshold (alcohol/drug withdrawal, illness, sleep deprivation).

SIGNS AND SYMPTOMS

Physical Exam
- Check for injuries caused as a result of seizure activity.
- Look for signs of infection, especially CNS infections.
- Assess and reassess mental status for signs of deterioration.

DIAGNOSIS

- Routine labs.
- Magnesium, calcium, toxicology screen, alcohol level, liver function tests.
- Consider LP.
- Consider CT scan of head.

TREATMENT

- Prevention of injury and adequate oxygenation in the actively seizing patient.
- Benzodiazepines are the mainstay of treatment in the seizing patient.
- Correct subtherapeutic levels of anticonvulsants.
- Treat underlying causes (meningitis, hypoglycemia, etc.).
- Most often, treatment is mainly supportive.
- IV fosphenytoin, valproic acid, or phenobarbital if benzodiazepines fail.

Factors that lower seizure threshold: **I AM H⁴IP**
Infection

Alcohol withdrawal, drugs
Medication (changes in dosing or compliance)

Head injury, **H**ypoxia, **H**ypoglycemia, **H**ypertension, **H**yponatremia (and other electrolyte abnormalities [Ca⁺ Mg])
Intracranial lesions
Pregnancy (eclampsia)

Seizures can cause posterior shoulder dislocations, as well as intraoral lacerations.

Todd's paralysis: Focal neurological deficit persisting from seizure, which usually resolves within 48 hours.

- Some due to genetic defect in channel proteins.
- 0.5 to 1% of population has disease.
- *Epilepsy:* Diagnosed after two or more unprovoked seizures.

► SECONDARY SEIZURE DISORDER

DEFINITION

Seizures that occur as a result of another disease condition.

CAUSES

Causes of secondary seizures: **MITTEN**
Metabolic
Infection
Trauma
Toxins
Eclampsia
Neurologic lesions

- Metabolic:
  - Hyper/hypoglycemia
  - Hyper/hyponatremia
  - Uremia
  - Hypocalcemia
- Infection:
  - Meningitis
  - Encephalitis
  - Intracerebral abscess
- Trauma:
  - Subdural hematoma
  - Epidural hematoma
  - Intracerebral hemorrhage
  - SAH
- Toxic:
  - Theophylline
  - Amphetamines
  - Cocaine
  - Tricyclic antidepressants
  - Alcohol withdrawal
  - CO
  - Cyanide
- Eclampsia
- Neurologic:
  - Cortical infarction
  - Intracranial hemorrhage
  - Hypoxia
  - Hypertensive encephalopathy
  - Congenital cerebral malformation or cortical dysplasia

Continuous seizures can cause significant CNS injury.

► STATUS EPILEPTICUS

DEFINITION

Seizures occurring continuously for at least 30 minutes, or two or more seizures occurring without full recovery of consciousness between attacks.

TREATMENT

- Treat with IV benzodiazepines, fosphenytoin, valproic acid, or phenobarbital.
- General anesthetics are last-line agents.

► ECLAMPSIA

- Usually occurs in patients > 20 weeks' gestation.
- Present with hypertension, edema, proteinuria, headache, vision changes, confusion, and seizure.
- Magnesium sulfate can be used to treat eclampsia.

► DELIRIUM TREMENS

- Seizures can occur in alcohol withdrawal.
- Associated with autonomic hyperactivity.
- Seizures can occur within 6 hours after last drink.
- Treated with benzodiazepines and supportive care.

Patients may still have CNS neuronal discharge despite neuromuscular blockade. Electroencephalogram will be required to detect seizure in this circumstance.

Head and Neck Emergencies

DEFINITION

Thrombosis (infectious or otherwise) in the dural sinus causing occlusion.

ETIOLOGY

- Bacterial infection of the central nervous system (CNS), usually secondary to a staphylococcal infection of the face or sinuses.
- Thrombosis may occur without antecedent infection.

PATHOGENESIS

- An infection in the face or sinuses spreads to the CNS causing inflammation of the linings of the dural sinuses.
- The inflammation predisposes to thrombosis with resultant occlusion of the cavernous sinus.

SIGNS AND SYMPTOMS

- Fever.
- Headache.
- Nausea.
- Vomiting.
- Occasionally seizures.
- Proptosis of the ipsilateral eye with chemosis and ophthalmoplegia.
- Sensory loss in the first division of the trigeminal nerve.
- Papilledema—late and ominous finding.

DIAGNOSIS

Diagnosis can be made with magnetic resonance imaging or digital venous angiogram.

TREATMENT

- Infective sinus thrombosis is usually successfully treated with appropriate intravenous (IV) antibiotics (*Staphylococcus* should be covered).
- Sometimes, despite appropriate treatment, the clot propagates to cause cerebral infarction and death.
- Heparin/warfarin therapy may reduce mortality and prevent permanent neurological damage.

The neurologic signs result from the third, fourth, and sixth cranial nerves passing through the cavernous sinus.

▶ **ACUTE SINUSITIS**

Inflammation of the paranasal sinuses of less than 3 weeks' duration: There are one sphenoidal, two maxillary, and two frontal sinuses, along with the ethmoid air cells, which compose the paranasal sinuses. Sinusitis can occur when there is drainage obstruction of the sinuses.

PATHOPHYSIOLOGY AND ETIOLOGY

- Edema causes obstruction of the drainage pathways followed by reabsorption of the air in the sinuses.

HIGH-YIELD FACTS

Head and Neck Emergencies

- The resultant negative pressure causes transudate collection within the sinuses.
- When bacteria is present, a suppurative infection can occur.

RISK FACTORS

- Viral upper respiratory infection (URI)
- Allergic rhinitis

ETIOLOGY

Streptococcus pneumoniae causes 37% of bacterial sinusitis, with *Haemophilus influenzae* causing 38%. Other common URI bacteria are also implicated.

SIGNS AND SYMPTOMS

- Pain over the sinuses.
- Decreased sense of smell.
- Fever.
- Purulent nasal discharge.
- Headache (may be aggravated by coughing, sneezing, and leaning forward).
- Tenderness to palpation or percussion over the affected area.
- Nasal canal may be inflamed, and purulent exudates may drain from the ostia.

DIAGNOSIS

- Usually clinical based on history and physical.
- There is no defined role for sinus plain radiography in diagnosis.
- CT can be employed when the diagnosis is uncertain, or if the patient is immunocompromised or appears toxic.

TREATMENT

- Antibiotics improve symptoms, prevent complications, and decrease duration of illness if there is good clinical evidence that suppuration is present.
- Amoxicillin or amoxicillin with clavulanate is first-line therapy.
- If allergic to penicillin, erythromycin plus a sulfonamide can be used.
- Over-the-counter decongestant nasal sprays can provide symptomatic relief.

COMPLICATIONS

- The infection can extend beyond the sinuses and, in the case of ethmoidal involvement, may enter the CNS.
- Bony destruction can also occur and may result in facial deformity.
- Direct extension from sinuses to the venous or lymphatic system can cause cavernous sinus thrombosis.

ADMISSION CRITERIA

Presence of complications, toxicity, fever with neurologic signs, or orbital or periorbital cellulitis all warrant admission.

Approximately 70% of human immunodeficiency virus patients will develop sinusitis, which may be caused by opportunistic bacteria, viruses, or fungi.

Only 38% of patients with sinusitis will have normal computed tomography (CT) scans. Sixteen percent of patients without sinusitis will have abnormal findings in the sinuses on CT scan.

DEFINITION

Nosebleed.

EPIDEMIOLOGY

- Anterior epistaxis is more common in younger patients.
- Posterior epistaxis is more common in the elderly population.

ANATOMY

- The nose humidifies and warms air and has a rich blood supply.
- The internal and external carotid arteries supply blood to the nasal mucosa through a number of smaller branches.

Anterior Epistaxis

- Comprise 90% of nose bleeds.
- Most commonly originates from Kiesselbach's plexus (a confluence of arteries on the posterior superior nasal septum).

Kiesselbach's plexus is located in the "picking zone."

ETIOLOGY

- Trauma to the nasal mucosa (usually self-induced)
- Foreign body
- Allergic rhinitis
- Nasal irritants (such as cocaine, decongestants)
- Pregnancy (due to engorgement of blood vessels)
- Infection (sinusitis, rhinitis)
- Osler–Weber–Rendu syndrome (telangiectasias)

In anterior epistaxis, the bleeding is unilateral and the patient denies a sensation of blood in the back of the throat.

DIAGNOSIS

- Labs are not routinely required if there are no comorbidities.
- Facial or nasal films may be considered in the setting of nasal trauma.

TREATMENT

- **Direct pressure:** Compress the elastic portions of the nose between the thumb and middle finger. Hold continuously for 10 to 15 minutes.
- **Vasoconstrictive agents:**
  - Phenylephrine or oxymetazoline can be instilled into the nasal cavity in conjunction with other treatment methods.
  - Cotton-tipped applicators can be used to apply vasoconstrictive agents if the bleed can be visualized.
- **Anterior nasal packing:**
  - Should be performed on any patient in which vasoconstrictive agents and direct pressure have failed.
  - Patients should receive antistaphylococcal prophylaxis.
  - Nasal packs should be removed at ear, nose, and throat (ENT) follow-up after 2 to 3 days.
- **Chemical cautery** with silver nitrate-tipped applicators.
- **Electrocautery** performed by an otolaryngologist.

Twenty-five percent of properly placed nasal packs fail to control bleeding. In this case, an emergent ENT consult is indicated.

Nasal tampons can be inserted along the floor of the nasal canal. They expand to several times their original size when instilled with saline or blood:

1. Use one-fourth-inch petroleum jelly–impregnated gauze. Grasp the gauze 3 cm from the end with bayonet forceps.
2. The first layer is placed on the floor of the nose through a nasal speculum. The forceps and the speculum are then withdrawn, and the gauze stays in place.
3. The nasal speculum is then reintroduced on top of the previous layer of gauze, and another layer of gauze is introduced using the forceps.
4. This process is repeated until several layers have been placed.
5. Remove the speculum and use the forceps to pack down the layers.

A successful anterior nasal pack is placed in an "accordion" fashion so that each layer lies anterior to the prior layer. This prevents the gauze from falling into the posterior nasal pharynx.

Posterior Epistaxis

DEFINITION

- Comprises approximately 10% of epistaxis in the emergency department (ED).
- More common in older patients and is thought to be secondary to atherosclerosis of the arteries supplying the posterior nasopharynx.

ETIOLOGY

- Hypertension
- Anticoagulation therapy
- Liver disease
- Blood dyscrasias
- Neoplasm
- Atherosclerosis of nasal vessels

CLINICAL FEATURES

- Blood may be seen effluxing from both nares or down the posterior oropharynx.
- Visualization of the bleeding usually requires use of a fiber-optic laryngoscope.
- Bleeding is often more severe than with an anterior bleed.

DIAGNOSIS

Routine labs (including complete blood count, prothrombin time, and activated partial thromboplastin time are drawn to look for possible coagulopathies.

TREATMENT

- **Posterior nasal packing:** Commercial nasal packs and specialized hemostatic balloon devices are available and are more efficacious than the traditional methods of posterior nasal packing. The procedure for inserting a commercial nasal hemostatic balloon is described below:

  1. Prepare the nasal cavity with vasoconstrictors and anesthetic agents.
  2. Insufflate 25 cc of saline into the anterior balloon to test for leakage. The posterior balloon is tested with 8 cc of saline.

Patients may experience reflux of blood through the nasal puncta after packing and should be warned in advance. No specific therapy is recommended.

Approximately 50% of patients presenting with posterior epistaxis have a systolic blood pressure ≥ 180 mm Hg or a diastolic pressure > 110 mm Hg.

3. Lubricate the device with 4% lidocaine jelly and insert it into the nasopharynx. Advance until the distal balloon tip is visible in the posterior oropharynx when the patient opens his mouth.
4. Fill the posterior balloon tip with 4 to 8 cc of saline and pull the device anteriorly such that it wedges in the posterior nasopharynx.
5. Fill the anterior balloon with 10 to 25 cc of saline while maintaining traction on the device.
6. It may be necessary to pack both nares to obtain adequate hemostasis.
7. Many patients require sedation following the procedure.

- **Embolization and ligation:** These treatments are indicated when the other treatments fail and should be performed by ENT specialists.
- All patients with posterior bleeds should have an emergent ENT consult and are usually admitted.

▶ **OTITIS EXTERNA**

DEFINITION

Infection of the external ear or external canal: Can be localized (furuncle) or can affect the entire canal.

ETIOLOGY

The most common causes are *Pseudomanas aeruginosa* and *Staphylococcus aureus*.

EPIDEMIOLOGY

It is more common in moist environments (summer, swimming pools, and the tropics).

SIGNS AND SYMPTOMS

- Sense of fullness in the ear
- White or green cheesy discharge
- Pain on retraction of pinna
- Itching
- Decreased hearing
- Fever
- A bulging or erythematous tympanic membrane

TREATMENT

- Cleanse ear canal thoroughly.
- Polymyxin–neomycin–hydrocortisone ear drops.

COMPLICATIONS

Malignant external otitis media:
- Seen in diabetics and immunocompromised hosts.
- Results in destruction of bone underlying the external ear canal.
- Characterized by excruciating pain, fever, friable granulation tissue in external ear canal, and facial palsies.
- Treated with anti-pseudomonal antibiotics.

Otitis externa is also known as "swimmer's ear."

A 16-year-old swim team captain presents with a greenish discharge from his ear and complains that his ear feels "full." He withdraws as you tug on his ear to examine it. *Think: Otitis externa.*

Patients with otitis externa should avoid getting water into their ears for 2 to 3 weeks after treatment.

DEFINITION

A bacterial or viral infection of the middle ear, usually secondary to a viral URI. Diagnosis of AOM requires:

1. History of acute onset of symptoms.
2. Presence of a middle ear effusion.
3. Signs and symptoms of middle ear inflammation.

ETIOLOGY

- *S. pneumoniae*, nontypeable *H. influenzae*, and *Moraxella catarrhalis* are the most common causes.
- Newborns can also get suppurative otitis with *Escherichia coli* and *S. aureus*.

EPIDEMIOLOGY

- More common in colder months.
- Most common age is ages 6 months to 3 years.

PATHOPHYSIOLOGY

Dysfunction of the eustachian tube leads to retention of secretions, which leads to bacterial colonization.

Secondhand smoke is a risk factor for otitis media in children.

SIGNS AND SYMPTOMS

- Ear pain and sense of fullness
- Perception of gurgling or rumbling sounds inside the ear
- Decreased hearing
- Dizziness
- Fever
- Poor feeding and irritability in infants

DIAGNOSIS

- Middle ear effusion:
  - Bulging of the typanic membrane (TM)
  - Limited or absent mobility of TM
  - Air–fluid level behind TM
  - Otorrhea
- Middle ear inflammation:
  - Distinct erythema of TM
  - Distinct otalgia

TREATMENT

- Acetaminophen or ibuprofen for analgesia.
- Topical anesthetic drops (e.g., benzocaine) can provide additional analgesic relief.
- First-line antibiotic choice is amoxicillin 80 to 90 mg/kg/day for 5 to 7 days for mild cases in children over age 2. For those under age 2, standard therapy is 10 days. For severe cases, amoxicillin–clavulanic acid can be used instead.

- Observation option (48 to 72 hours):
  - For healthy children ages 6 months to 2 years who have nonsevere symptoms at presentation and in whom diagnosis is uncertain.
  - For healthy children over age 2 with any (severe or nonsevere) symptoms *or* an uncertain diagnosis.
  - This option is further limited to those who have ready access to follow-up health care and a caregiver who agrees.
- Failure of observation: Start antibiotics.
- Failure of initial antibiotics: Change antibiotic.

COMPLICATIONS

- Serous otitis media—an effusion of the middle ear resulting from incomplete resolution of otitis media.
- Acute mastoiditis.

▶ **ACUTE MASTOIDITIS**

DEFINITION

Bacterial infection of the mastoid process resulting in coalescence of the mastoid air cells: It is usually a complication of acute otitis media in which the infection has spread into the mastoid antrum.

SIGNS AND SYMPTOMS

- Swelling, erythema, tenderness, and fluctuance over the mastoid process
- Displacement of pinna laterally and inferiorly
- Fever
- Earache
- Otorrhea
- Decreased hearing

DIAGNOSIS

CT scan of the mastoid air cells reveals cell partitions that are destroyed, resulting in coalescence.

TREATMENT

- Antibiotics should cover the common acute otitis media pathogens and be resistant to beta-lactamase.
- Third-generation cephalosporins are preferred because they offer good penetration into the CNS at the proper doses.
- This therapy is usually effective in preventing neurological sequelae.

COMPLICATIONS

Subperiosteal abscess requires mastoidectomy.

▶ **LUDWIG'S ANGINA**

DEFINITION

Cellulitis of bilateral submandibular spaces and the lingual space: This is a potentially life-threatening infection.

ETIOLOGY

- Usually a result of spread of a bacterial odontogenic infection into the facial tissue spaces.
- Most common bug is mouth anaerobe *Bacteroides*.

RISK FACTORS

- Oral trauma
- Dental work
- Salivary gland infection

SIGNS AND SYMPTOMS

- Fever
- Drooling
- Trismus
- Odynophagia
- Dysphonia
- Elevated tongue
- Swollen neck
- Labored breathing

DIAGNOSIS

Soft-tissue radiographs of the neck can be obtained, but they should not delay treatment or place the patient in an area where emergent airway management is difficult.

TREATMENT

- Secure airway.
- IV antibiotics (penicillin and metronidazole or clindamycin).
- Definitive treatment is incision and drainage, then excision of fascial planes in the operating room (OR).
- ENT or oral and maxillofacial surgery consult.

▶ **PHARYNGITIS**

DEFINITION

Infection of the pharynx and tonsils that rarely occurs in infants and is uncommon under 2 years of age.

EPIDEMIOLOGY

Peak incidence is between 4 and 7 years old but occurs throughout adult life.

ETIOLOGY

Viruses
- Most common cause of all pharyngeal infection.
- Rhinoviruses and adenoviruses are the most common viral causes.
- Epstein–Barr virus, herpes simplex virus, influenza, parainfluenza, and coronaviruses also contribute.

Bacteria
- *Streptococcus pyogenes* (group A, beta-hemolytic strep) is the most common cause of bacterial pharyngitis.
- *Mycoplasma*, *Chlamydia*, and *Corynebacterium* also occur.

Fungal and Parasitic
- Can also occur in the immunocompromised host.

SIGNS AND SYMPTOMS

Symptoms
- Incubation period is 2 to 5 days, after which patients develop sore throat, dysphagia, chills, and fever.
- Headache, nausea, vomiting, and abdominal pain can also occur.

Signs
- Erythematous tonsils
- Tonsillar exudates
- Enlarged and tender anterior cervical lymph nodes
- Palatal petechiae

DIAGNOSIS

- Throat culture: Still the most effective means of diagnosis. There is a delay in obtaining results while the culture grows, and a good sample must be obtained.
- Rapid antigen detection tests (RADTs): Are available to detect streptococcus; > 95% specific. A negative RADT should be confirmed with a throat culture.
- See Figure 6-1.
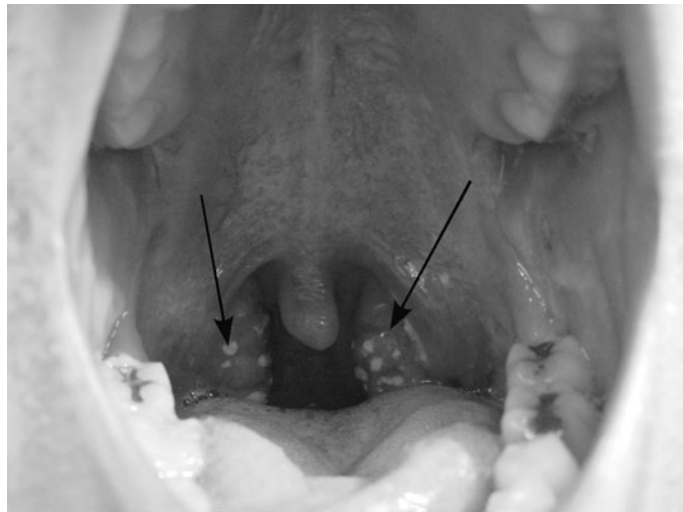
F I G U R E 6 - 1 . Streptococcal pharyngitis.

Note white exudates (arrows) on top of erythematous swollen tonsils. (Reproduced with permission from Nimishikavi S, Stead LG. *Streptococcal pharyngitis.* N Engl J Med 2005 Mar 17;352(11):e10. Copyright © 2005 Massachusetts Medical Society. All rights reserved.)

Signs and symptoms are the same for viral and bacterial pharyngitis. One cannot differentiate between the two causes without microbiology testing.

Treatment for Streptococcal Pharyngitis

A one-time intramuscular injection of penicillin (benzathine penicillin 1.2 million units) or a 10-day course of oral penicillin is the treatment of choice. Erythromycin is an alternative for penicillin-allergic patients.

Complications

- Post-streptococcal glomerulonephritis (PSGN)
- Rheumatic fever (RF)
- Cervical lymphadenitis
- Peritonsillar abscess
- Retropharyngeal abscess
- Sinusitis
- Otitis media

▶ EPIGLOTTITIS

Definition

A life-threatening inflammatory condition (usually infectious) of the epiglottis and the aryepiglottic folds and periglottic folds.

Etiology

- *H. influenzae* type B (Hib) is the most common cause.
- *Streptococcus* is the next most common cause.
- Can also be caused by other bacteria and rarely viruses and fungi.

Epidemiology

- The incidence in children has decreased dramatically after the introduction of the Hib vaccine.
- Most cases are now in adults and unimmunized children.

Signs and Symptoms

- Prodromal period of 1 to 2 days.
- High fever.
- Dysphagia.
- Stridor.
- Drooling.
- Secretion pooling.
- Dyspnea.
- Erect or tripod position.
- Pain on movement of the thyroid cartilage is an indicator of supraglottic inflammation.

Diagnosis

- High clinical suspicion is necessary.
- Radiographs of the neck soft tissue can aid in diagnosis (Figure 6-2), as can fiberoptic laryngoscopy.
- Direct laryngoscopy is contraindicated because it may induce fatal laryngospasm.

Treatment algorithm for strep pharyngitis:
- RADT (+): Treat
- RADT (−): Culture and treat until culture results are available.
- If culture is negative, discontinue antibiotics.
- No RADT available: Culture and treat until culture results are known. If negative, discontinue antibiotics.

Group-A, beta-hemolytic strep is associated with sequelae of RF and PSGN. Treating strep pharyngitis prevents RF, but not PSGN.

"Thumbprint sign" (Figure 6-2) is seen on lateral neck radiograph and demonstrates a swollen epiglottis obliterating the vallecula.

FIGURE 6-2. Epiglottitis.

Arrow indicates classic "thumbprint" sign of enlarged, inflamed epiglottis.

- Do not examine the oropharynx unless surgical airway capability is available at the bedside.

TREATMENT

- Intubation as needed to protect airway
- Ceftriaxone
- Intensive care unit admission

COMPLICATIONS

Airway obstruction and resultant respiratory arrest.

DIFFERENTIAL DIAGNOSIS: CROUP

- Croup caused by parainfluenza virus also presents with sore throat and stridor.
- Anteroposterior soft-tissue neck radiograph may show the "steeple sign," which is indicative of subglottic narrowing (Figure 6-3).
- Treat with humidified oxygen, bronchodilators, and racemic epinephrine.
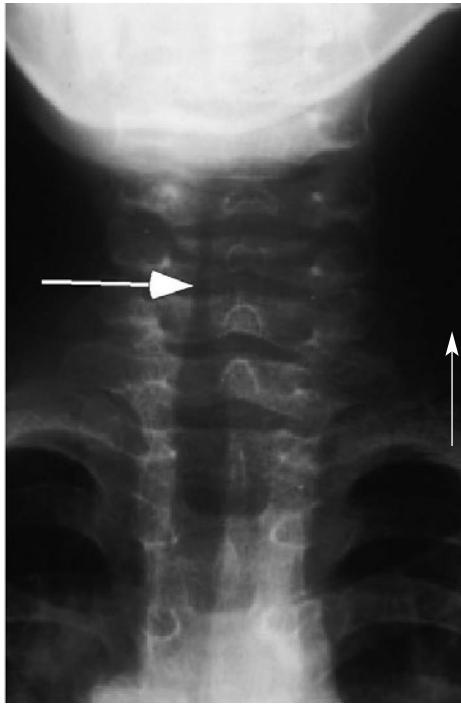
FIGURE 6-3. Radiograph demonstrating steeple sign of croup.

Note narrowing of airway (arrow).

► PERITONSILLAR ABSCESS

SIGNS AND SYMPTOMS

- Sore throat
- Muffled voice
- Decreased oral intake
- Tilting of head to affected side
- Trismus
- Deviation of uvula to affected side
- Swollen erythematous tonsils
- Fluctuant soft palate mass
- Cervical lymphadenopathy

DIAGNOSIS

By physical exam.

TREATMENT

- Secure the airway; these abscesses are in a very precarious space and can cause complete upper airway obstruction.
- Antibiotics against gram-positive oral flora (including anaerobes).
- Consider steroids to decrease inflammation.
- ENT consult.
- Incision and drainage of abscess may need to be done in the OR, depending on the size and degree of airway compromise.

A 29-year-old male who had been treated for strep throat the previous week presents with progressive difficulty swallowing. Physical exam reveals a fluctuant mass on the right side of the soft palate and deviation of the uvula to the right. *Think: Peritonsillar abscess.*

Patients with epiglottitis prefer to sit leaning forward with the neck slightly flexed. Patients with retropharyngeal abscess prefer recumbency and hyperextension of the neck.

DEFINITION

Abscess in the pharyngeal spaces.

SIGNS AND SYMPTOMS

- Difficulty breathing
- Fever, chills
- Severe throat pain
- Toxic appearance
- Hyperextension of neck
- Stridor
- Drooling
- Swollen, erythematous pharynx
- Tender cervical lymph nodes

DIAGNOSIS

Radiograph of the soft tissues of the neck (see Figure 6-4): Exaggerated swelling in the pharyngeal spaces is indicative of abscess.

TREATMENT

Same as for peritonsillar abscess, except that all parapharyngeal and retropharyngeal abscesses are drained in the OR.

FIGURE 6-4. Retropharyngeal abscess.

Lateral radiograph of the soft tissue of the neck. Note the large amount of prevertebral edema (solid arrow), and the collection of air (dashed arrow). (Photo courtesy of Dr. Gregory J. Schears.)

HIGH-YIELD FACTS IN

Respiratory Emergencies

EPIDEMIOLOGY

- Most common cause of death from infectious disease in the United States; sixth most common cause of death in the United States overall.
- Community-acquired pneumonia is an acute infection in patients not hospitalized or residing in a care facility for 14 days prior to onset of symptoms.

ETIOLOGY

Most common etiologies:
- Bacterial:
  - *Streptococcus pneumoniae*
  - *Haemophilus influenzae*
  - *Mycoplasma pneumoniae*
  - *Legionella pneumophila*
  - *Moraxella catarrhalis*
  - *Chlamydia pneumoniae*
  - *Staphylococcus aureus*
  - Gram-negative bacilli *(Pseudomonas)*
- Viral:
  - Influenza
  - Parainfluenza
  - Adenovirus
- *Mycobacterium tuberculosis* and endemic fungi are rare causes of community-acquired pneumonia.

SIGNS AND SYMPTOMS

- Signs:
  - Tachypnea
  - Tachycardia
  - Rales
  - Diaphoresis
- Symptoms:
  - Dyspnea
  - Chest pain
  - Cough
  - Hemoptysis
- Physical findings associated with increased risk:
  - Respiratory rate > 30/min
  - Heart rate > 140 bpm
  - Blood pressure, systolic < 90 or diastolic < 60 mm Hg
  - Temperature > 101°F
  - Change in mental status
  - Extrapulmonary infection
  - Blood cultures should be obtained in this high-risk group

DIAGNOSIS

Hemoglobin < 9 g/dL or hematocrit < 30%.

A 27-year-old patient presents with pneumonia, bullous myringitis, and a chest film that looks worse than expected. *Think:* Mycoplasma pneumonia.

A patient with human immunodeficiency virus (HIV) has a CD4 count of 52, does not take antiretroviral medications or trimethoprim–sulfamethoxazole, is hypoxic on room air, has an elevated lactic dehydrogenase (LDH), and has diffuse bilateral infiltrates on chest x-ray (CXR). *Think:* Pneumocystis carinii *pneumonia.*

An elderly man presents with pneumonia, diarrhea, bradycardia, and hyponatremia. *Think:* Legionella.

If you see *currant jelly sputum* on physical exam or *bulging fissure* on CXR, think *Klebsiella.*

TABLE 7-1. Pneumonia Severity Index ("PORT Score")

Characteristic	Points Assigned
Demographic factor	
Age (men)	Age (yrs)
Age (women)	Age (yrs) − 10
Nursing-home resident	+10
Coexisting illnesses	
Neoplastic disease	+30
Liver disease	+20
Congestive heart failure	+10
Cerebrovascular disease	+10
Renal disease	+10
Physical examination findings	
Altered mental status	+20
Respiratory rate ≥ 30 breaths/min	+20
Systolic blood pressure < 90 mm Hg	+20
Temperature < 35°C (95°F) or 40°C (104°F)	+15
Pulse ≥ 125 beats/min	+10
Laboratory and radiographic findings (if study performed)	
Arterial blood pH < 7.35	+30
Blood urea nitrogen level ≥ 30 mg/dL	+20
Sodium level < 130 mmol/L	+20
Glucose level ≥ 250 mg/dL	+10
Hematocrit < 30%	+10
Partial pressure of arterial O2 < 60 mm Hg or O_2 sat < 90%	+10
Pleural effusion	+10

Class	Points	Mortality*
I	< 51	0.1%
II	51–70	0.6%
III	71–90	0.9%
IV	91–130	9.5%
V	> 130	26.7%
Patients in classes I–III can usually be managed as outpatients.		

Adapted, with permission, from Fine MJ et al. A prediction rule to identify low-risk patients with community acquired pneumonia. *N Engl J Med* 1997;336:4:247. Copyright © 1997 Massachusetts Medical Society. All rights reserved.

TREATMENT AND DISPOSITION

- Treatment of viral pneumonia is supportive.
- Antibiotic selection for bacterial pneumonia depends on the organism involved. Empiric therapy and hospital admission are based on the patient's age, comorbidities, severity of symptoms, and particular risk factors.
- The Pneumonia Patient Outcomes Research Team (PORT) has proposed a severity index (see Table 7-1) that can be used to guide the decision of outpatient versus inpatient therapy.

Antibiotics should be administered for suspected bacterial pneumonia within 4 hours of presentation.

▶ ASPIRATION PNEUMONIA

DEFINITION

- Pathogens can enter the lung by inhalation of aerosols, by hematogenous spread, or by aspiration of oropharyngeal contents.
- Up to half of normal adults aspirate oropharyngeal contents during sleep. Individuals with swallowing disorders, impaired level of con-

sciousness, or an impaired gag reflex are more likely to aspirate material into the lungs. The chances of developing a pneumonia depend on the volume aspirated and the virulence of the material.

- Different organisms can be present in the aspirate depending on the individual.

PATHOPHYSIOLOGY

- Anaerobic pulmonary pathogens colonize dental plaque and gingiva; aspiration can cause pneumonia or lung abscess.
- Common pulmonary pathogens can colonize the nasopharynx of normal individuals.
- Aerobic gram-negative bacilli can colonize the stomach and reach the oropharynx in vomit or by spreading colonization in debilitated individuals.
- Mucociliary dysfunction, common in smokers, and alveolar macrophage dysfunction will reduce the clearing of aspirate and increase the chances of infection.
- Presence of foreign bodies in the aspirate will also increase the chances of infection.

TREATMENT

- Supportive
  - Oxygen
  - Suctioning
- Bronchoscopy for large foreign body aspirates
- No role for prophylactic steroids or antibiotics

▶ PULMONARY EDEMA

DEFINITION

Pulmonary edema is the accumulation of fluid in the interstitial space of the lung. The most common cause of pulmonary edema is cardiogenic in nature, which results from increased pulmonary capillary pressure.

CARDIOGENIC CAUSES

- Left ventricular failure (myocardial infarction [MI], ischemia, cardiomyopathy).
- Increased pulmonary venous pressure without failure (valvular disease).
- Increased pulmonary arterial pressure.

NONCARDIOGENIC CAUSES

- Hypoalbuminemia (decreased oncotic pressure).
- Altered membrane permeability (adult respiratory distress syndrome).
- Lymphatic insufficiency.
- High-altitude pulmonary edema.
- Opiate overdose.
- Neurogenic pulmonary edema.

CONGESTIVE HEART FAILURE (CHF)

- Acute pulmonary edema (APE) secondary to left ventricular failure is commonly known as CHF.

TREATMENT

- Diuretics (furosemide).
- Oxygen.
- Nitroglycerin to promote preload reduction and venodilation.
- Aspirin: Antiplatelet agent, protective against MI.
- Morphine to decrease preload and relieve anxiety.

▶ PULMONARY EMBOLUS (PE)

RISK FACTORS

- Genetic predisposition
- Age
- Obesity
- Cigarette smoking
- Hypertension
- Oral contraceptives
- Hormone replacement therapy
- Neoplasm
- Immobilization
- Pregnancy and postpartum period
- Surgery and trauma
- Hypercoagulable state

SIGNS AND SYMPTOMS

Signs
- Tachypnea
- Tachycardia
- Hypoxia
- Rales
- Diaphoresis
- Bulging neck veins
- Heart murmur

Symptoms
- Dyspnea (seen up to 90% of the time)
- Chest pain (seen up to 66% of the time)
- Apprehension
- Cough
- Hemoptysis

DIAGNOSIS

- ECG pattern of $S_1Q_3T_3$ (Figure 7-1) is seen in less than one fourth of patients. Most common rhythm is sinus tachycardia.
- Arterial blood oxygen levels are not useful in predicting the absence of PE, and a-A gradient is not considered a suitable screening test.
- Lower extremity venography studies are of value when positive but do not exclude PE when negative.
- The gold standard for the diagnosis of PE is pulmonary angiography. However, it is invasive, takes the patient away from the department, and has an up to 5% mortality and morbidity rate.

Treatment of APE: **NOMAD**
Nitroglycerin
Oxygen
Morphine
Aspirin
Diuretics

APE may be a presentation of acute MI.

There are no signs, symptoms, laboratory values, CXR, or electrocardiographic (ECG) findings that are diagnostic of PE or are consistently present. The absence of any of these should not be used to rule out PE. Diagnosis requires a high index of suspicion.

A 53-year-old female smoker who returned from a week-long road trip yesterday presents with dyspnea and tachycardia and appears extremely anxious. *Think: PE.*

Remember, PE is one of the causes of pulseless electrical activity.
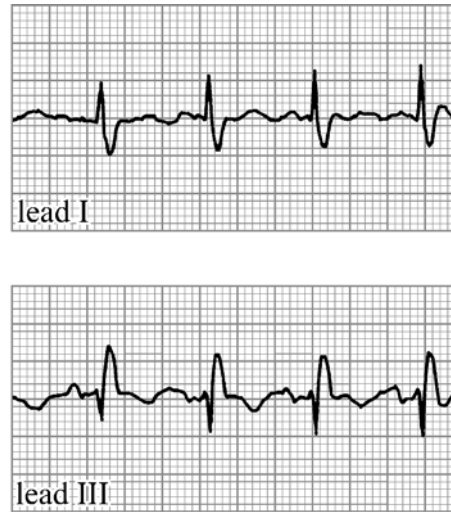
Figure 7-1. Classic S₁Q₃T₃ pattern of pulmonary embolism.

- Ventilation–perfusion scan can be a useful test in patients who cannot undergo spiral computed tomography (CT) (e.g., renal insufficiency, pregnancy). However, 15% of patients with low-probability ventilation–perfusion scans have had angiographically proven PE.
- Spiral CT is being used more often as the diagnostic test of choice. One great advantage of spiral CT is its ability to identify alternative diagnoses.
- Enzyme-linked immunosorbent assay (ELISA) D-dimer tests are 90% sensitive but nonspecific. False positives occur with malignancy, pregnancy, trauma, infection, recent surgery, many inflammatory states, and advanced age. For this reason, it is most often restricted to use in young patients with low pretest probability of PE.

TREATMENT

> *Possible treatments of pulmonary embolism:*
> **FATSO**
> **F**ilter
> **A**nticoagulation
> **T**hrombolytics
> **S**urgery
> **O**xygen

- All patients should get oxygen.
- Anticoagulation is the mainstay of therapy and may consist of heparin (inpatient) and coumadin or a low-molecular-weight heparin such as enoxaparin.
- Thrombolytics should be considered for hemodynamically unstable patients.
- Surgical options include an inferior vena cava filter (good for patients with contraindications to anticoagulation) and embolectomy (poor prognosis).

▶ **PLEURAL EFFUSION**

> *Most common causes of pleural effusion:*
> - CHF
> - Bacterial pneumonia
> - Malignancy

DEFINITION

- The pleural space lies between the chest wall and the lung and is defined by the parietal and visceral pleuras. A very small amount of fluid is normally present, allowing the two pleural membranes to slide over each other during respiration. An abnormal amount of fluid (> 15 cc) in the pleural space is known as a pleural effusion.

- Pleural effusions are divided into transudates and exudates. Transudative effusions happen when there is either an increase in capillary hydrostatic pressure or a decrease in colloid osmotic pressure. Both of these conditions will cause a net movement of fluid out of capillaries into the pleural space.

CAUSES OF TRANSUDATIVE EFFUSIONS

- CHF
- Hypoalbuminemia
- Cirrhosis
- PE
- Myxedema
- Nephrotic syndrome
- Superior vena cava obstruction
- Peritoneal dialysis

CAUSES OF EXUDATIVE EFFUSIONS

- Infection (pneumonia, tuberculosis [TB], fungi, parasites)
- Connective tissue diseases
- Neoplasm
- PE
- Uremia
- Pancreatitis
- Esophageal rupture
- Intra-abdominal abscess
- Post surgery or trauma
- Drug induced

DIAGNOSIS

Pleural Fluid Analysis
- Transudate:
  - LDH < 200 U
  - Fluid-to-blood LDH ratio < 0.6
  - Fluid-to-blood protein ratio < 0.5
- Exudate:
  - Glucose < 60 mg/dL in infection, neoplasm, rheumatoid arthritis, pleuritis.
  - Amylase: Elevated in esophageal rupture, pancreatitis, pancreatic pseudocyst, and some neoplasms.
  - Cell count.
  - Gram stain and culture.
  - Cytology.

TREATMENT

- Treat underlying cause.
- A thoracocentesis can be both diagnostic and therapeutic.

► **ASTHMA**

DEFINITION

Asthma is a disease in which the tracheobronchial tree is hyperreactive to stimuli, resulting in variable, reversible airway obstruction.

Steroids are given in asthma to decrease the late inflammatory response.

Drugs most commonly associated with acute asthma exacerbations are aspirin and beta blockers.

Early asthmatic response lasts a few hours. Late asthmatic response hyperresponsiveness can persist for weeks to months.

ED treatment of asthma:
BIOMES
- **B**eta agonists
- **I**pratropium
- **O**xygen
- **M**agnesium sulfate
- **E**pinephrine
- **S**teroids

EPIDEMIOLOGY

- Incidence greater in men than women.
- Incidence greater in African Americans than whites.
- Half of cases develop by age 17 and two thirds by age 40.

EXTRINSIC ASTHMA

- Sensitivity to inhaled allergens.
- Immunoglobulin E response.
- Causally related in a third of asthma cases and a contributing factor in another third.
- Frequently seasonal.
- Early asthmatic response is mast cell dependent and results in acute bronchoconstriction.
- Late asthmatic response is an inflammatory reaction that leads to prolonged airway responsiveness.

NONIMMUNOLOGIC PRECIPITANTS OF ASTHMA

- Exercise
- Infections
- Pharmacologic stimuli
- Environmental pollution
- Occupational stimuli
- Emotional stress
- Diet

SIGNS AND SYMPTOMS

- Dyspnea.
- Wheezing.
- Cough.
- Chest tightness.
- Severe exacerbation: Use of accessory muscles, tripod position, hypoxia, tachypnea, impending respiratory failure.

EMERGENCY TREATMENT

- The mainstay of therapy is oxygen and beta agonist nebulizers.
- Corticosteroids are added for moderate asthma.
- Severe asthma may require epinephrine or magnesium sulfate.
- Impending respiratory failure will require intubation.

OUTPATIENT MANAGEMENT OPTIONS

- Leukotriene inhibitors (e.g., zafirlukast, montelukast).
- Mast cell stabilizing agents (e.g., cromolyn sodium).
- Methylxanthines (e.g., theophylline, aminophylline).

► **CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)**

DEFINITIONS

- Three separate disease entities are part of the classification of COPD. These are asthma, bronchitis, and emphysema. Asthma is discussed above.

- Chronic bronchitis is defined as a condition in which excessive mucus is produced. The mucus production is enough to cause productive cough for a minimum of 3 months out of the year in at least 2 consecutive years.
- Emphysema is a disease in which there is distention of the air spaces distal to the terminal bronchioles and destruction of alveolar septa. Alveolar septa are important for providing support of the bronchial walls. Their destruction leads to airway collapse especially during expiration.

EPIDEMIOLOGY

Third most common cause of hospitalization and fourth most common cause of death (after stroke) in the United States.

RISK FACTORS

- Smoking
- Air pollution
- Occupational exposure
- Infection
- Genetic (e.g., alpha$_1$-antitrypsin deficiency)

SIGNS AND SYMPTOMS OF CHRONIC BRONCHITIS

- History of cough and sputum production
- History of smoking
- Commonly overweight
- Cyanosis
- Right ventricular failure
- Normal total lung capacity
- Increased residual volume
- Normal to slightly decreased vital capacity

SIGNS AND SYMPTOMS OF EMPHYSEMA

- Exertional dyspnea
- Thin to cachectic
- Tachypnea
- Prolonged expiratory phase often with pursed lips
- Use of accessory muscles of respiration
- Increased total lung capacity and residual volume
- Decreased vital capacity

TREATMENT

Acute
- Oxygen (correct to at least > 90%)
- Nebulized beta agonist
- Intravenous or oral corticosteroids
- Antibiotics if underlying pneumonia is suspected

Chronic
- Smoking cessation
- Optimize nutrition
- Regular exercise
- Home O$_2$ if needed
- Control of respiratory infections

Patients with chronic bronchitis are sometimes referred to as "blue bloaters."

Patients with emphysema are sometimes referred to as "pink puffers."

TB infection and subsequent scarring of the fallopian tubes used to be a common cause of infertility.

Smoking cessation is the most important treatment for COPD.

DEFINITION

Mycobacterium tuberculosis is an intracellular, aerobic, acid-fast bacillus that infects humans. It is primarily spread through the respiratory route.

PATHOPHYSIOLOGY

- Bacillus normally enters through the lungs.
- Macrophage phagocytoses bacillus.
- Bacillus multiplies intracellularly in the macrophage until it lyses the macrophage and repeats the process.
- Cell-mediated immunity, through T-helper cells, activates macrophages, destroying infected cells.
- Epithelioid cell granulomas are produced that wall off the primary lesion; remaining bacilli can survive within granulomas for years.
- Months to years later, bacilli can reactivate.

SIGNS AND SYMPTOMS

- Primary TB is usually asymptomatic.
- Reactivation pulmonary TB is the syndrome most commonly seen:
  - Cough
  - Fever
  - Night sweats
  - Weight loss
  - Hemoptysis
  - Fatigue
  - Anorexia
- Extrapulmonary TB occurs in 15% of cases and can be seen in the pericardium, skeletal system, gastrointestinal/genitourinary systems, adenoids, peritoneum, adrenal glands, and skin.

DIAGNOSIS

Purified Protein Derivative Testing
- > 5-mm induration considered positive in:
  - Persons with HIV or risk factors and unknown HIV status.
  - Close household contacts of person with active TB.
  - Persons with evidence of primary TB on x-ray.
- > 10 mm positive in high-risk groups:
  - Intravenous drug users who are HIV negative.
  - Persons from high-prevalence areas.
  - Persons with other medical conditions that render them debilitated or immunocompromised.
- > 15 mm is considered positive in all other individuals.

CXR
- Most useful test in the emergency department (ED).
- Active TB usually presents with parenchymal infiltrates. Most common locations for TB in the lung include apices and posterior segments of upper lobes (areas of highest blood flow and oxygen tension).
- Miliary TB presents as small diffuse nodules (millet seed appearance).
- Pulmonary lesions can be calcified or cavitated. Cavitation implies higher infectivity.

Tuberculous adenitis is known as *scrofula.*
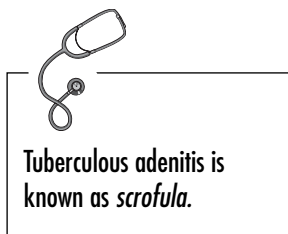
TABLE 7-2. Potential Adverse Effects of First-Line TB Medications

DRUG	POTENTIAL ADVERSE EFFECTS
Isoniazid	Hepatitis, peripheral neuropathy, drug interaction with phenytoin and carbamazepine
Rifampin	Orange body fluids, hepatitis, GI intolerance, thrombocytopenia, cholestatatic jaundice, significant drug interactions
Pyrazinamide	Hepatitis, hyperuricemia, rash, GI intolerance, thrombocytopenia, cholestatic jaundice, significant drug interactions
Ethambutol	Retrobulbar optic neuritis, color blindness, headache
Streptomycin (IM only)	Ototoxicity, nephrotoxicity

- Sputum and blood can be cultured for M. *tuberculosis* via acid-fast staining (results not available for a few weeks).
- Mantoux/PPD testing result also not available immediately; usually read 48 to 72 hours after placement. A positive test does not necessarily imply active TB; rather, it detects latent, prior, and active TB, so it is not that useful in the ED. Note that immunocompromised patients may have false-negative Mantoux tests; in this case an "anergy panel" is applied.

A Ghon complex is a calcified lesion of *primary* pulmonary TB.

TREATMENT

- Patient with suspected TB should be masked and placed in respiratory isolation (negative pressure) room.
- Health care workers should wear respiration masks when feasible.
- Initial therapy consists of four drugs, usually isoniazid, rifampin, pyrazinamide, and ethambutol or streptomycin for a 2-month period. (See Table 7-2 for adverse effects.)
- Patients with active multidrug-resistant TB should be admitted to the hospital.
- Patients with reliable follow-up who are clinically stable with adequate socioeconomic situation may be discharged home. Patients at risk for noncompliance may require directly observed treatment (DOT) wherein a health care worker physically administers the medication to the patient daily.
- Bacille Calmette–Guérin (BCG) vaccine: Given frequently in countries outside the United States. Interferes with the Mantoux test (false positive). In the United States, should be considered only for select persons in consultatin with a TB expert.

Miliary TB results from hematogenous spread.

Tuberculosis of the spine is known as Pott's disease.

▶ **HEMOPTYSIS**

DEFINITION

Coughing up of blood due to bleeding from the lower respiratory tract. Minor hemoptysis is < 5 mL blood in a 24-hour period. Massive hemoptysis is > 600 mL of bleeding in a 24-hour period, or bleeding that results in clinical impairment of respiratory function.

Patients die of massive hemoptysis secondary to *asphyxiation,* rather than *exsanguination.*

Pulmonary	
Airways diseases	▪ Bronchitis ▪ Bronchiectasis ▪ Cystic fibrosis
Neoplasms	▪ Bronchogenic carcinoma ▪ Bronchial carcinoid
Infections	▪ Tuberculosis ▪ Pneumonia ▪ Lung abscess ▪ Aspergilloma
Pulmonary vascular diseases	▪ Pulmonary thromboembolism ▪ Pulmonary vasculitis ▪ Arteriovenous malformations ▪ Bronchovascular fistula
Pulmonary renal syndromes	▪ Wegener's granulomatosis ▪ Goodpasture's syndrome ▪ Systemic lupus erythematosus
Cardiovascular	▪ Mitral stenosis ▪ Congestive heart failure
Miscellaneous	▪ Use of anticoagulants or fibrinolytics ▪ Bleeding diathesis ▪ Factitious hemoptysis (bleeding from nasopharynx or gastrointestinal tract) ▪ Pulmonary contusion/trauma ▪ Iatrogenic (pulmonary artery rupture secondary to pulmonary arterial catheterization) ▪ Idiopathic pulmonary hemosiderosis

Bronchiectasis is persistent and progressive dilation of bronchi or bronchioles as a consequence of chronic infections, tumor, or cystic fibrosis. Coughing, fetid breath, and expectoration of mucopurulent matter are hallmarks.

ETIOLOGY

See Table 7-3.

TREATMENT

- Supplemental oxygen.
- Have patient sit up with head of the bed elevated.
- Place patient with radiographically abnormal lung (presumably bleeding side) down when recumbent.
- Codeine to control coughing.
- Massive hemoptysis may require surgical intervention and/or invasive measures such as endobronchial cold saline or epinephrine, bronchial artery embolization, and mainstem bronchus intubation.

TYPES OF VENTILATORS

- Pressure-cycled ventilation delivers volume until a preset peak inspiratory pressure is reached.
- Volume-cycled ventilation delivers a preset tidal volume.
- Time-cycled ventilation delivers volume until a preset time is reached.

VENTILATOR SETTINGS

- Ventilatory rate
- Tidal volume
- Inspired oxygen concentration
- Positive end-expiratory pressure (PEEP)
- Inspiratory–expiratory ratio

VENTILATOR MODES

- Controlled mechanical ventilation (CMV): The patient is ventilated at a preset rate; the patient cannot breathe between the delivered breaths.
- Assist–control ventilation: A minimum rate is set, but if the patient attempts to take additional breaths, the machine will deliver a breath with a preset tidal volume.
- Intermittent mandatory ventilation and synchronized intermittent mandatory ventilation (SIMV): The machine delivers a preset number of breaths at the preset tidal volume; additional breaths initiated by the patient will have a tidal volume dependent on the patient's effort. In SIMV, the machine breaths are synchronized so as not to interfere with spontaneous breaths.
- Pressure support: Patient determines respiratory rate, and tidal volume depends on both the patient's pulmonary compliance and the preset inspiratory pressure.

Ventilatory rate is initially set at 12 to 14 breaths per minute.

Tidal volume is initially set at 6 to 10 cc/kg.

PEEP improves oxygenation by keeping alveoli open during expiration.

CMV is useful in patients with no spontaneous respirations, heavily sedated patients, and paralyzed patients.

HIGH-YIELD FACTS

Repiratory Emergencies

Cardiovascular Emergencies

Chest pain (CP) is one of the most common emergency department (ED) complaints. The differential is broad (see Figure 8-1), but the seven most emergent causes include:

1. Acute coronary syndrome (ACS)
2. Aortic dissection
3. Pericarditis/myocarditis
4. Tension pneumothorax (see Trauma chapter)
5. Pulmonary embolus (see Respiratory Emergencies chapter)
6. Esophageal rupture (see Gastrointestinal Emergencies chapter)
7. Perforated peptic ulcer (see Gastrointestinal Emergencies chapter)

ED evaluation of chest pain consists of rapid determination as to whether the CP represents a potentially life-threatening cause. The history and physical can provide helpful clues.

Features of history that increase likelihood that CP is due to ACS (in order of decreasing probability):

- Substernal location of CP.
- Pain lasts from 30 minutes to 3 hours, of gradual onset.
- Pain is described as crushing, squeezing, tightness or pressure ("like an elephant sitting on my chest"), rather than knifelike, sharp, stabbing, or pins and needles.
- Pain is accompanied by diaphoresis or "impending sense of doom."
- Pain is similar to previous anginal pain.
- Family history of first-degree male relative with ACS at age 50 or younger.

Levine sign: A patient puts a clenched fist to his chest to describe the pain of myocardial ischemia.

Cardiac	Pulmonary
Myocardial ischemia/acute coronary syndrome	Pulmonary embolism
Coronary vasospasm	Pleuritis
Dissecting aortic aneurysm	Mediastinitis
Pericarditis	Spontaneous pneumothorax
Myocarditis	Pneumonia
Aortic stenosis	Neoplasm
Hypertrophic cardiomyopathy	Bronchitis
Mitral valve prolapse	
	Musculoskeletal
GI	Herpes zoster
Esophageal rupture (Boerhaave's)	Costochondritis
Esophageal tear (Mallory–Weiss)	Rib fracture
Esophageal spasm	Myalgia
Reflux esophagitis	Chest wall pain
Cholecystitis/biliary colic	
Pancreatitis	

F I G U R E   8 - 1 .   Differential diagnosis of chest pain.

Features of physical exam that increase likelihood that CP is due to ACS (in order of decreasing probability):

- Pain radiating to left arm
- Pain radiating to both arms
- Pain radiating to right arm
- Diaphoresis
- Nausea/vomiting
- New S_3 sound
- Hypotension (systolic blood pressure < 80 mm Hg)
- Lung crackles

Women, the **elderly,** and **diabetics** tend to present with *atypical* symptoms, including:

- Dyspnea alone
- Nausea/vomiting
- Palpitations
- Syncope
- Cardiac arrest

Chest pain may or may not be present in these populations.

Contraindications to NTG:
- Patients who have taken phosphodiesterase inhibitors such as sildenafil (Viagra), vardenafil (Levitra), or tadalafil (Cialis) in the preceding 24 hours
- Patients with inferior wall MI
Severe hypotension can result in these cases.

Contraindications to beta blockers:
- Heart rate < 60 bpm
- Systolic BP < 100 mm Hg
- Second- or third-degree heart block
- Moderate to severe left ventricular dysfunction
- Severe chronic obstructive pulmonary disease (COPD) or asthma
- Signs of peripheral hypoperfusion
- PR interval > 0.24 msec
- Acute MI due to cocaine

▶ EVALUATION OF ACUTE CORONARY SYNDROME (ACS)

Acute coronary syndromes can be classified as ST segment elevation myocardial infarction (STEMI), non–ST segment elevation myocardial infarction (NSTEMI), or unstable angina (UA). NSTEMI and UA are thought to represent the ends of the same disease process spectrum.

Angina is considered "unstable" if:

1. It lasts longer than 20 minutes or frequency is increased from baseline.
2. It is new onset and markedly limits physical activity.
3. Onset occurs at rest.

Unstable angina with elevated serum cardiac biomarkers is NSTEMI.

T A B L E   8 - 1 .   **Cardiac Biomarkers**

ENZYME/CARDIAC BIOMARKER	RISE (HRS POST CP)	PEAK	RETURN TO BASELINE
Myoglobin	1–2 hrs	4–6 hrs	24 hrs
Troponin	3–6 hrs	12–24 hrs	7–10 days
Creatine kinase, total and MB fraction	4–6 hrs	12–36 hrs	3–4 days
Lactate dehydrogenase	6–12 hrs	24–48 hrs	6–8 days
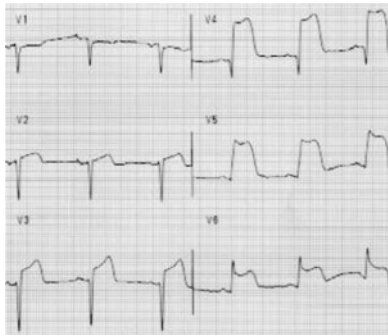
F I G U R E   8 - 2 .   Anterolateral STEMI.

Management of ACS

1. Airway, breathing, circulation (ABCs) assessed.
2. Intravenous (IV) O_2 monitor.
3. 12-lead electrocardiogram (ECG) obtained. If evidence of STEMI, see section on reperfusion therapy below.
4. Laboratory analyses obtained: cardiac biomarkers (see Table 8-1), complete blood count (CBC), electrolytes, coagulation studies, and drug levels (e.g., digoxin) if relevant.
5. Chewable aspirin 162 to 325 mg unless patient is allergic or hat it prior to arrival.
6. Sublingual (SL) nitroglycerin (NTG) provides analgesia and coronary dilatation; 0.4 mg should be given SL q 5 minutes up to total of three doses. If patient transiently responds to NTG but pain returns, IV NTG can be started.
7. Beta blocker (e.g., metoprolol 5 mg q 5 minutes up to three doses) to maintain heart rate around 60 bpm (minimizes myocardial demand).
8. IV morphine 4 mg should be administered for analgesia with titrated increments of 2 to 10 mg repeated in 5- to 15-minute intervals. Reduces excess catecholamine release.

Sgarbossa criteria for impending MI in LBBB:
1. ST elevation > 1 mm in leads with dominant R waves (concordant with QRS complex)
2. ST elevation > 5 mm in leads with dominant S waves (discordant with QRS complex)
3. ST depression > 1 mm in V_1, V_2, or V_3

Age > 65
Presence of three or more cardiac risk factors:
    Male gender
    MI in male relatives < 50 years of age
    Cigarette smoking
    Hypertension
    Diabetes mellitus
    Hypercholesterolemia
Aspirin use within last 7 days
Two or more anginal events within last 24 hours
ST segment elevation on initial ECG
Elevated cardiac biomarkers
Prior coronary artery stenosis > 50%

F I G U R E   8 - 3 .   TIMI Risk Score for UA/NSTEMI (1 point for each).

(Data from Antman EM, Cohen M, Bernink PJ, et al. The TIMI Risk Score for unstable angina/non-ST elevation MI: A method for prognostication and therapeutic decision making. *JAMA* 2000;284:835–842.)

Contraindications to thrombolytics for STEMI:

- History of intracranial hemorrhage
- Suspected aortic dissection
- Active bleeding (except menses)
- Significant closed head or facial trauma within the preceding 3 months
- Malignant brain neoplasm
- History of ischemic stroke > 3 hours but < 3 months
- Known structural brain lesion (e.g., arteriovenous malformation)
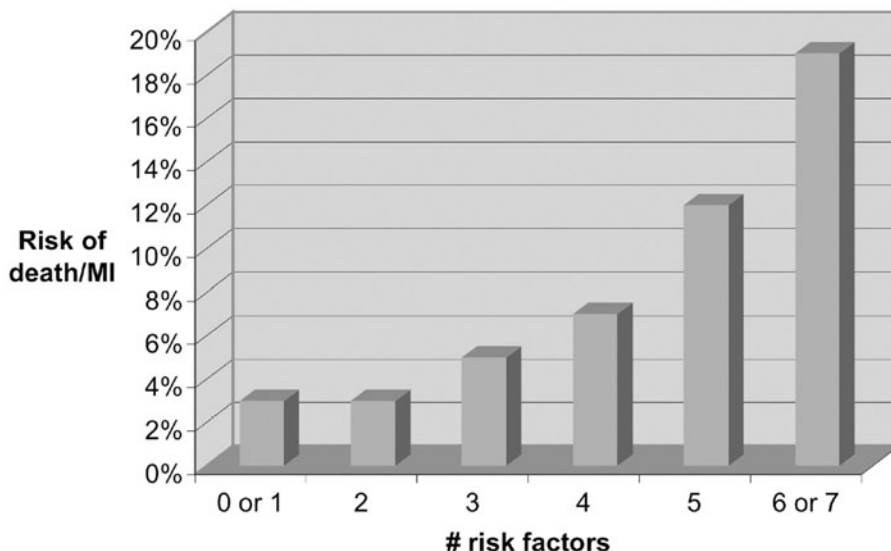
FIGURE 8-4. Risk of death or MI at 14 days based on ED TIMI score for UA/NSTEMI.

DIAGNOSIS OF STEMI

- Elevation of > 1 mm in ST segments of two or more contiguous segments (Figure 8-2)
- New left bundle branch block (LBBB)

REPERFUSION THERAPY FOR STEMI

Reperfusion therapy is given for STEMI within 12 hours of onset. There are two options: pharmacologic thrombolytics (e.g., alteplase, reteplase, tenecteplase, streptokinase, urokinase) and percutaneous coronary intervention (PCI or "cardiac catheterization").

The American College of Cardiology (ACC)/American Heart Association (AHA) class I recommendation for reperfusion therapy is as follows: primary

Factor	# Points
Age ≥ 75	3
Age 65–74	2
Diabetes mellitus or hypertension	1
Systolic blood pressure < 100 mm Hg	3
Heart rate > 100 bpm	2
Killip Class II or higher	2
Weight < 67 kg	1
Anterior ST elevation or LBBB	1
Time to Rx (PCI or thrombolytics)	1

FIGURE 8-5. TIMI Risk Score for STEMI.

(Data from Morrow DA, Antman EM, Charlesworth A, et al. TIMI Risk Score for ST-elevation myocardial infarction: A convenient, bedside, clinical score for risk assessment at presentation. *Circulation* 2000;102:2031–2037.)
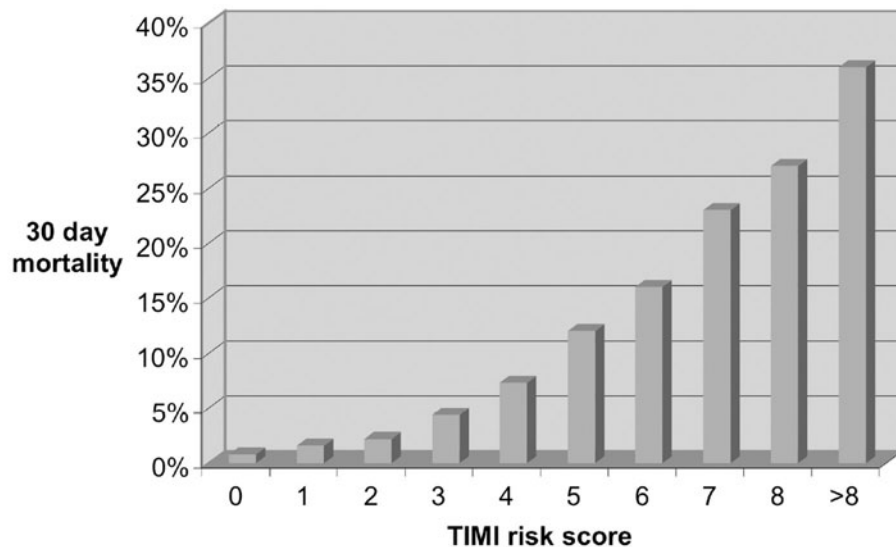
F I G U R E 8 - 6 . Risk of death at 30 days for STEMI based on ED TIMI score.

PCI is preferred to thrombolytics when it can be performed within 90 minutes of presentation and if the patient is in severe congestive heart failure (CHF) or cardiogenic shock. If primary PCI is more than 90 minutes away, thrombolytics should be administered unless any contraindications exist (see box).

Risk Stratification in ACS

The TIMI Risk Score is a stratification tool developed by the Thrombolysis in Myocardial Infarction (TIMI) group that can be helpful to categorize patients into high- and low-risk groups. This score has been prospectively validated in several studies. Separate scores have been developed for UA/NSTEMI and STEMI. For UA/NSTEMI, the TIMI Risk Score may be helpful for disposition decisions (hospital admission vs. observation unit vs discharge home). A score of 5 or more is considered high risk, 3–4 intermediate risk, and 0–2 low risk (see Figures 8-3 through 8-6).

▶ **PACEMAKERS**

DEFINITION

- Most pacers use a three-letter code; advanced functions are described by a fourth/fifth letter:
  1. Pacing chamber: V (ventricular), A (atrial), or D (dual)
  2. Sensing chamber: V, A, or D
  3. I (inhibited), T (triggered), D (dual)
  4. P (programmable rate), C (communications stored), R (rate responsive)
  5. P (pacing), S (shock), D (dual), 0 (neither)
- Transcutaneous pacing: Indicated in refractory bradycardia with hypotension when a transvenous pacemaker cannot be placed quickly.

- Transvenous pacing: Pacemaker wire is passed through central venous access line into heart for pacing.

EMERGENT PACEMAKER PLACEMENT INDICATIONS

Symptoms of cardiovascular compromise associated with:
- New bi-/trifascicular block with acute ischemia:
  - Bi = RBBB + LAFB, RBBB + LPFB, LBBB
  - Tri = RBBB + LAFB + first-degree AV block, or RBBB + LPFB + first-degree AV block, or LBBB + first-degree AV block or alternating LBBB + RBBB
- Mobitz type II AV block
- Third-degree or complete AV block
- Significant bradycardia (< 40 beats per minute)

MOST COMMON PROBLEMS

- Failure to capture
- Failure to sense
  - Undersensing
  - Oversensing

► CONDUCTION ABNORMALITIES

First-Degree AV Block

- First degree (prolonged PR interval—see Figure 8-7):
  - Prolonged conduction of atrial impulses without loss of any impulses.
  - PR interval > 0.20 second.
  - Benign and asymptomatic.
  - Doesn't warrant further ED workup or treatment.

Second-Degree AV Block, Type I

- Second degree, Mobitz type I (Wenckebach—see Figure 8-8):
  - Progressive prolongation of PR interval with each successive beat until there is a loss of AV conduction and hence a dropped beat or failure of ventricles to depolarize (P wave but no QRS).

Second-Degree AV Block, Type II

- Second degree, Mobitz type II (see Figure 8-9):
  - Random loss of conduction and beat below the AV node without change in PR interval (His–Purkinje system) (P, no QRS).

FIGURE 8-7. First-degree AV block.
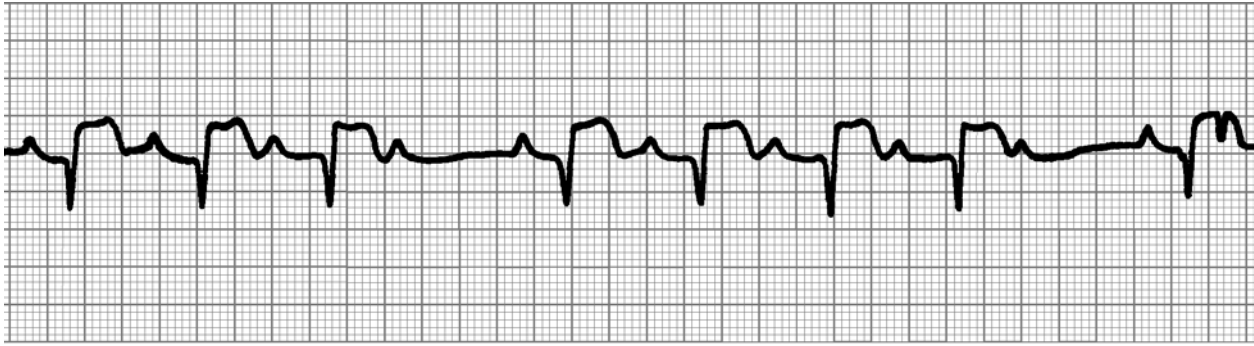
FIGURE 8-8. Mobitz I (Wenckebach) second-degree AV block.

- Potentially serious pathology present.
- Can be seen with anterior wall MI.
- Often progresses to complete AV block (third degree).

Third-Degree AV Block

- Third degree (complete AV dissociation [see Figure 8-10]):
  - No conduction of atrial signal and P wave through to the ventricle, and hence independent atrial and ventricular rhythms.
  - Either congenital (with associated anatomic anomalies) or acquired (many causes—idiopathic fibrosis most common).
  - Patients present with tachypnea, dyspnea on exertion, cyanosis, or syncope.
  - ECG shows no correlation between atrial (faster) and ventricular (slower) rhythms; P waves "march through" the rhythm strip ignoring the QRS complexes.

TREATMENT

- ABCs, IV access, O$_2$, monitor.
- Immediate transcutaneous or transvenous pacemaker.
- Eventual implantable pacemaker if no reversible cause found.
- Treat underlying cause if possible.

Type II second-degree block has a worse prognosis than type I.

Causes of AV Blocks
- Age
- Ischemia
- Cardiomyopathies
- Myocarditis
- Congenital
- Surgery
- Valvular disease

FIGURE 8-9. Mobitz II second-degree AV block.
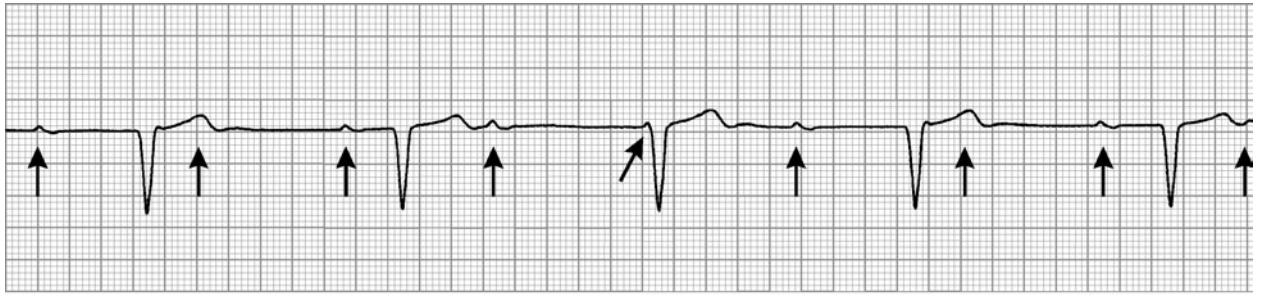
FIGURE 8-10. Third-degree (complete) AV block.

Note the P waves (arrows) "marching through."

Left Anterior Fascicular Block (LAFB)

- ECG:
  - Left axis deviation (LAD).
  - QRS duration 0.10 to 0.12 second.
  - Peak of terminal R in aVL precedes peak of terminal R in aVR.
  - Deep S waves in II, III, and aVF.
  - Lead I R wave > leads II or III.

Left Posterior Fascicular Block (LPFB)

- ECG:
  - R axis deviation
  - QRS duration 0.10 to 0.12 second
  - Small R and deep S in lead I
  - Lead III R wave > lead II
  - Small Q in II, III, and aVF

Left Bundle Branch Block (LBBB)

- Conduction blocked before anterior and posterior fascicles split (Figure 8-11).
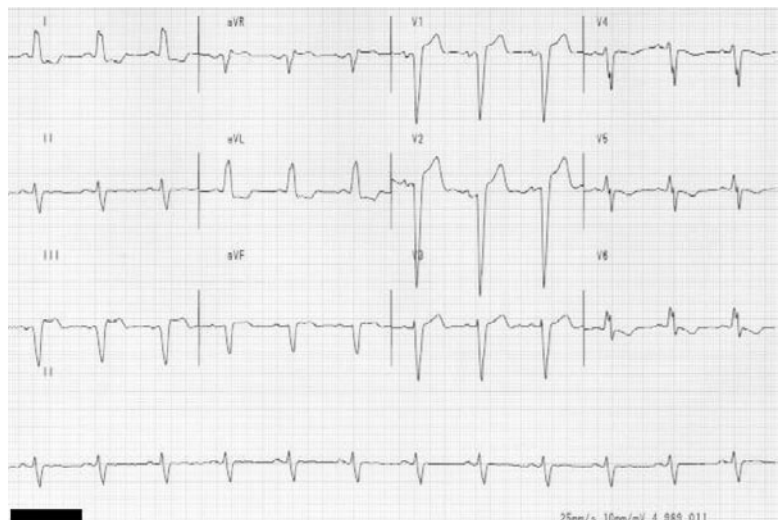
FIGURE 8-11. Left bundle branch block.

Presence of new LBBB may indicate AMI.

154
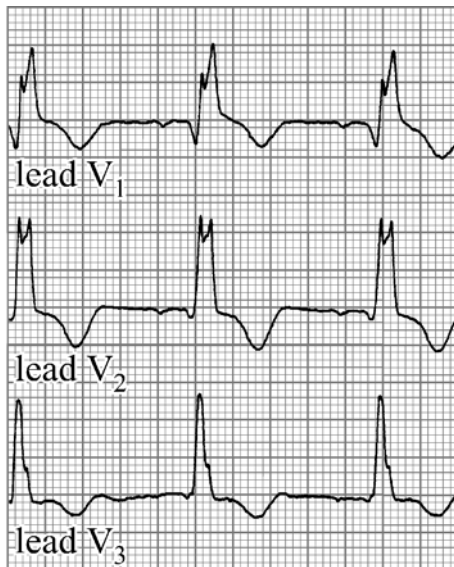
F I G U R E   8 - 1 2 .   Right bundle branch block.

Note the M-shaped QRS complex (R,R').

- Ischemia can be masked with LBBB. Use of Sgarbossa criteria can be helpful.
- Point system for determining acute ischemic change in the presence of LBBB (the more points, the more likely is ischemia):
  - ST segment elevation ≥ 1 mm concordant (in the same direction) with its QRS axis = 5 points.
  - ST segment elevation ≥ 1 mm in V_1–V_3 = 3 points.
  - ST segment elevation ≥ 5 mm, discordant with QRS = 2 points.
- ECG:
  - LAD.
  - QRS duration > 0.12 second.
  - ST and T waves directed opposite to terminal 0.04-second QRS.
  - No Q waves in I, aVF, V_5, V_6.
  - Large wide R waves in I, aVL, V_5, V_6.

Right Bundle Branch Block (RBBB)

- Ischemia is not masked with RBBB (see Figure 8-12), except in leads V_1–V_3.
- ECG:
  - QRS duration > 0.12 second
  - Triphasic QRS complexes
  - RSR´ described as "rabbit ears" in morphology in V_1, V_2
  - Wide S waves in I, aVL, V_5, V_6

▶ DYSRHYTHMIAS

Prolonged QT Syndrome

DEFINITION

- QT_c > 430 msec in men
- QT_c > 450 msec in women

Causes of prolonged QT:
QT WIDTH
QT: Prolonged QT syndromes, including Romano–Ward and Jervell and Lange–Nielsen
Wolff–Parkinson–White (WPW) syndrome
Infarction
Drugs
Torsades
Hypocalcemia, hypokalemia, hypomagnesemia

Prolonged QT and hypertrophic cardiomyopathy are causes of sudden death in young people.

SIGNS AND SYMPTOMS

- Syncopal episodes.
- Can predispose to paroxysmal episodes of ventricular tachycardia and torsade de pointes by "R-on-T phenomenon." This is when a premature ventricular complex–QRS fires at the same time as the peak of the T wave or "vulnerable period" in ventricular repolarization (when some but not all myocardial tissue is ready for the signal) inducing ventricular tachycardia or ventricular fibrillation via a ventricular reentry pathway.

DIAGNOSIS

- QT_I (QT interval) = 0.34 to 0.42 second or 40% of RR interval
- QT_c = HR corrected QT = $QT_I \div \sqrt{RR}$

See Figure 3-1 for ECG of prolonged QT.

TREATMENT

- ABCs, monitor.
- Correct electrolytes.
- Discontinue offending medications (Table 8-2).
- If inherited, beta blockers to decrease sympathetic stimulus and implantable overdrive pacemaker/defibrillator.
- Magnesium sulfate IV for torsade de pointes.

T A B L E 8 - 2 . **Drugs That Prolong the QT$_c$ Interval**

Antiarrhythmics	Antidepressants
■ Amiodarone	■ Fluoxetine (Prozac)
■ Flecainide	■ Quetiapine (Seroquel)
■ Quinidine (Quinaglute, Cardioquin)	■ Sertraline (Zoloft)
■ Procainamide (Pronestyl, Procan)	
■ Sotalol (Betapace)	**Antipsychotics**
■ Bepridil (Vasocor)	■ Chlorpromazine (Thorazine)
■ Disopyramide (Norpace)	■ Haloperidol (Haldol)
■ Dofetilide (Tikosyn)	■ Mesoridazine (Serentil)
■ Ibutilide (Corvert)	■ Pimozide (Orap)
	■ Thioridazine (Mellaril)
Antimicrobials	
■ Sparfloxacin (Zagam)	**Other**
■ Pentamidine (Pentam)	■ Cisapride (Propulsid)
■ Quinolone antibiotics (e.g., levofloxacin)	■ Salmeterol (Serevent)
■ Macrolide antibiotics (e.g., erythromycin, clarithromycin)	■ Arsenic trioxide (Trisenox)
■ Fluconazole (Diflucan)	
■ Chloroquine (Arelan)	

Sinus Bradycardia

DEFINITION

- HR < 60 and regular:
  - P wave prior to every QRS complex
  - Upright Ps in I and aVF
  - Narrow (< 0.12 second) QRS complexes

ETIOLOGY

- Can be normal, especially in athletes.
- Hypoxemia.
- Hypothyroidism.
- Excessive vagal tone.
- Hypothermia.
- Medication side effects: Beta blockers, digoxin, Ca^{2+} channel blockers, cholinergic toxins.

TREATMENT

- Correct underlying problem.
- In code situation, give atropine 0.5 to 1.0 mg IV followed by epinephrine and transcutaneous pacing.
- May require transvenous pacemaker if severe and there is evidence of cardiac compromise.

Sinus Tachycardia

DEFINITION

- HR > 100 and regular:
  - Usually < 150 bpm
  - P wave prior to every QRS complex
  - Upright Ps in I and aVF
  - Narrow QRS complexes

ETIOLOGY

- Anemia
- Dehydration/hypovolemia
- Fever
- Sepsis
- Drug overdose
- Anxiety
- Hypermetabolic state

TREATMENT

Correct underlying problem.

Atrial Ectopy

- **Premature atrial complexes (PACs):** Abnormal electrical focus triggers an atrial contraction before the sinus node fires, thus triggering a QRS and ventricular contraction. There is a compensatory pause (longer RR interval) before the next sinus beat. Benign and asymptomatic.

If the RR distance is at least one inch, consider:
One INCH
Overmedication

Inferior wall MI, increased intracranial pressure
Normal variant
Carotid sinus hypersensitivity
Hypothyroidism

Sinus bradycardia is commonly seen in inferior wall MI, but usually resolves within 1 to 2 days.

- **Wandering atrial pacemaker:** ≥ 3 different P wave morphologies/foci in a normal 12-lead ECG rhythm strip with an HR between 60 and 100 bpm. QRS follows each P wave. Usually asymptomatic; patient may complain of palpitations or anxiety.
- **Multifocal atrial tachycardia (MAT):** Wandering atrial pacemaker with a rate > 100 bpm. Patient is usually symptomatic (dyspnea, diaphoresis, ± angina). Associated with chronic obstructive pulmonary disease (COPD) and theophylline overdose. Rate is often difficult to control.

Atrial Flutter

- Rapid atrial depolarization (240 to 350 bpm) from an abnormal focus within the atria and variable ventricular conduction described as block (i.e., 2:1–4:1 flutter [see Figure 8-13]).
- Can be considered a transitional dysrhythmia between normal sinus and atrial fibrillation.
- Causes are same as for atrial fibrillation.

F I G U R E 8 - 1 3 . Atrial flutter.

Atrial Fibrillation

DEFINITION

Very rapid atrial depolarization (350 to 600 bpm) from many ectopic atrial foci, usually with ineffective conduction to ventricles.

ETIOLOGY

- Hypertensive heart disease
- Ischemic heart disease
- Valvular heart disease
- Alcohol
- Thyrotoxicosis
- Lung disease
- Fever

EPIDEMIOLOGY

- Most common sustained cardiac dysrhythmia, very frequently seen in the ED.
- Lifetime risk for developing atrial fibrillation is 25% for men and women over age 40 (Framingham Heart Study).

PATHOPHYSIOLOGY

- Rapid ventricular response gives ineffective systole (from poor filling) and subsequent heart failure/pulmonary edema, palpitations, or angina.
- Presence of atrial fibrillation predisposes to atrial blood stasis and subsequent clotting, which can embolize and cause stroke.
- Ineffective atrial contraction results in complete loss of atrial kick and its contribution to cardiac output, most relevant to those with heart failure.

DIAGNOSIS

ECG (see Figure 8-14):
- Irregularly irregular ventricular rhythm.
- Narrow QRS complexes (unless coexistent bundle branch block or aberrant ventricular conduction).
- Ventricular rate can be rapid (uncontrolled) or controlled (with medications).
- Echocardiography should be done to identify structural heart disease and rule out atrial thrombi prior to electrical cardioversion (transesophageal).
- Labs:
  - Check complete blood count (CBC), cardiac enzymes, thyroid function tests (TFTs), and ethanol level to look for underlying cause.

TREATMENT

- ABCs, IV, O_2, monitor.
- Control ventricular response rate to between 60 and 100 with AV nodal blocking drug (Ca^{2+} channel blocker or beta blocker acutely, and digoxin long term).
- Consider elective cardioversion (see Procedures chapter) if < 48 hours duration. If > 48 hours, anticoagulate for 4 weeks prior to cardioversion (or until transesophageal echo that demonstrates no atrial clot). Emergency cardioversion if severe compromise refractory to medications, regardless of duration (rare).
- Anticoagulation in new-onset atrial fibrillation is mandatory because of the significant risk for embolization, which can result in stroke, ischemic bowel, and deep venous thrombosis.
- Treat for ACS as appropriate.

> Causes of atrial fibrillation:
> **PIRATES**
> **P**ulmonary disease
> **I**schemia
> **R**heumatic heart disease
> **A**nemia, atrial myxoma
> **T**hyrotoxicosis
> **E**thanol
> **S**epsis

> *Atrial fibrillation and stroke:* Atrial fibrillation increases the risk of acute ischemic stroke (AIS) fivefold. It is responsible for 15 to 20% of all AIS.
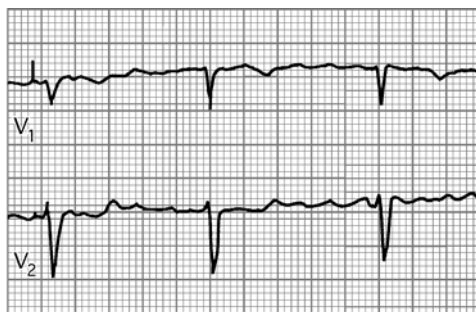
F I G U R E   8 - 1 4 .   Atrial fibrillation.

Note lack of distinct P waves.

SVT is the most common pediatric dysrhythmia.

Supraventricular Tachycardia (SVT)

DEFINITION

Narrow QRS complex tachycardia with regular RR intervals at a rate of 150 to 250 bpm.

ETIOLOGY

- Due to either increased atrial automaticity or reentry phenomenon.
- Etiologies similar to atrial fibrillation.

SIGNS AND SYMPTOMS

- Dyspnea.
- Palpitations.
- Angina.
- Diaphoresis.
- Varying degrees of hemodynamic stability.
- Weak or nonpalpable pulses.
- CHF.
- Shock.

DIAGNOSIS

- Chest x-ray (CXR): Most often normal
- ECG:
  - Narrow QRS complex.
  - Tachycardia at a rate > 150 bpm.
  - Typically **regular,** P waves may or may not be visible.

TREATMENT

- ABCs, IV, O_2, monitor.
- Immediate synchronized cardioversion (50 J) if hemodynamically unstable, in CHF or ACS.
- Vagal maneuvers or adenosine (6-mg rapid IV push followed by 20-cc flush, repeat as needed × 2 with 12 mg each time) to block AV nodal conduction.
- Diltiazem (0.25 mg/kg IV over 2 minutes) or verapamil (0.15 mg/kg IV over 1 minute) or beta blocker to control rate: Watch out for hypotension.

Vagal maneuvers:
- Carotid massage (check carotids for bruits prior to massage; never massage both sides simultaneously)
- Diving reflex (cold water immersion of head)
- Valsalva maneuver

Wolff–Parkinson–White (WPW) Syndrome

DEFINITION

- A syndrome in which there is an accessory electrical pathway that causes SVT in older children and adults.
- Heart beats too fast for adequate filling and may lead to shock.

SIGNS AND SYMPTOMS

- Palpitations
- Diaphoresis
- Tachypnea
- Variable degrees of hemodynamic stability
- Weak to no pulses

Features of WPW:
- Short PR interval
- Widened QRS interval
- Delta wave slurring QRS upswing

- CHF
- Shock

DIAGNOSIS

- ECG (see Figure 8-15):
  - Narrow QRS complex.
  - HR > 200.
  - P waves present.
  - Slurred upstroke of QRS (delta wave)—may not be evident during tachyarrhythmia.

TREATMENT

- ABCs, monitor, IV access, O_2.
- Patients with WPW, rapid atrial fibrillation, and rapid ventricular response require emergent cardioversion.
- Stable patients with WPW and atrial fibrillation are treated with amiodarone, flecainide, procainamide, propafenone, or sotalol.
- Adenosine, beta blockers, calcium channel blockers, and digoxin are *contraindicated* because they preferentially block conduction at the AV node, allowing unopposed conduction down the accessory bypass tract.

> Don't give ABCD (adenosine, beta blockers, calcium channel blockers, or digoxin) to someone with WPW.
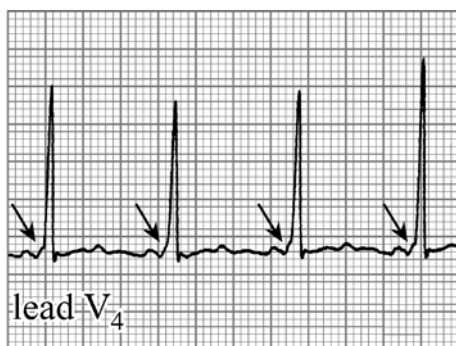
F I G U R E   8 - 1 5 .   WPW syndrome.

Arrows indicate pathognomonic delta waves.

Sick Sinus Syndrome (SSS)

DEFINITION

- Sinus arrest: Also known as "pause" of sinoatrial node signal that usually results in "ectopic" or "escape" beats and rhythms that take over as source for ventricular impulse.
- Also called tachy–brady syndrome: Any combination of intermittent fast and slow rhythms with associated AV block and inadequate escape rhythm:
  - Fast: A-fib, atrial flutter, SVT, junctional tachycardia
  - Slow: Sinus brady, varying sinus pauses, escapes

SIGNS AND SYMPTOMS

- Reflect fast or slow HR:
  - Palpitations
  - Syncope

- Dyspnea
- Angina
- Embolic events

DIAGNOSIS

ECG: Any of above rhythms (see definition).

TREATMENT

- ABCs, IV, O_2, monitor
- Follow ACLS protocols
- Pacemaker

> ► **ESCAPE RHYTHMS**

Junctional Ectopy

- Ectopic beats that originate from the junction of atria and ventricle.
- Normal ventricular depolarization and repolarization.
- Narrow QRS complexes.
- Absent or late, retrograde P waves coming on or after the QRS.

Ventricular Ectopy

DEFINITION

Ectopic beats that originate from below the AV node.

ETIOLOGY

- Normal
- Ischemia
- Electrolyte abnormality
- Medications (digoxin, beta blockers, Ca^{2+} channel blockers)
- Caffeine, alcohol

DIAGNOSIS

- ECG (see Figure 8-16):
  - Wide QRS
  - No preceding P wave
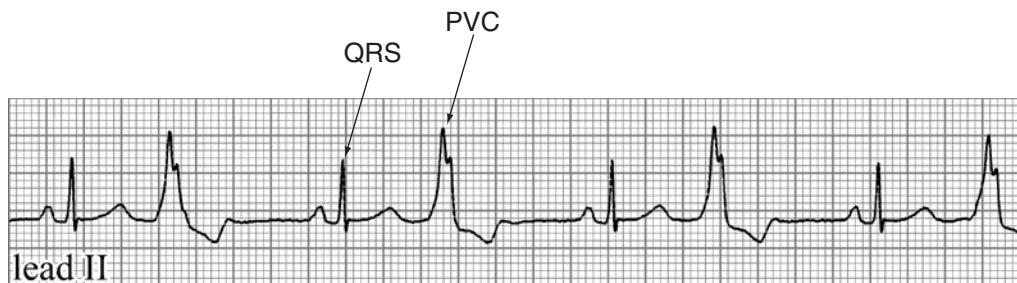
F I G U R E 8 - 1 6 . Ventricular bigeminy.

Note the premature ventricular complex (PVC) that regularly follows the QRS complex.
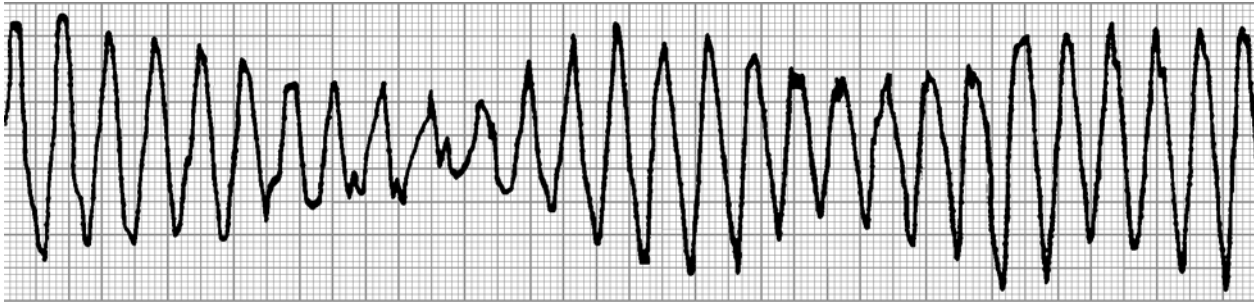
FIGURE 8-17. Torsade de pointes.

TREATMENT

None; treat underlying cause.

Torsade de Pointes

- French: "Twisting of the points."
- Refers to a ventricular tachycardia variation in which QRS axis swings from positive to a negative in a single lead (see Figure 8-17).
- Can be caused by R-on-T phenomenon.
- Treatment: IV magnesium sulfate, cardioversion if unstable.

Ventricular Tachycardia

- ≥ 3 ectopic ventricular beats in a row.
- See the Resuscitation chapter.

Ventricular Fibrillation

See the Resuscitation chapter.

Causes of torsade:
POINTES
Phenothiazines
Other medications (tricyclic antidepressants)
Intracranial bleed
No known cause (idiopathic)
Type I antidysrhythmics
Electrolyte abnormalities
Syndrome of prolonged QT

▶ **CARDIOMYOPATHIES**

Types

There are three types of cardiomyopathies: dilated, restrictive, and hypertrophic. The end stages of dilated and restrictive cardiomyopathies ultimately result in heart failure, and thus ED management of these conditions is the same as for decompensated heart failure from any other cause. More details on these cardiomyopathies are not immediately relevant to emergency medicine. Hypertrophic cardiomyopathy, however, does have special relevance to emergency medicine and is thus discussed in detail here.

A 25-year-old man becomes severely dyspneic and collapses while running laps. His father had died suddenly at an early age. *Think: Hypertrophic cardiomyopathy.*

Hypertrophic cardiomyopathy: Symptoms prior to 30 years correlates with increased risk of sudden death, but severity of symptoms (whenever they occur) does not.

Hypertrophic cardiomyopathy: Very few murmurs *decrease* with squatting (this one does).

Causes of paradoxical splitting of S_2:
- Hypertrophic cardiomyopathy
- Aortic stenosis
- LBBB

Hypertrophic Cardiomyopathy

DEFINITION

- Hypertrophied, nondilated, often asymmetric left ventricle (septum > free wall) with 2° atrial dilation.
- Also known as idiopathic hypertrophic subaortic stenosis, or IHSS.

PATHOPHYSIOLOGY

Results in:
- Systolic dysfunction (end-stage dilation)
- Diastolic dysfunction (poor filling and relaxation)
- Myocardial ischemia (increased O_2 demand because of increased myocardial mass)

ETIOLOGY

- Idiopathic or inherited (50%)
- Hypertension (HTN)
- Aortic or pulmonic stenosis

SIGNS AND SYMPTOMS

- Angina:
  - Not well understood in terms of known pathophysiology.
  - Occurs at rest and during exercise.
  - Frequently unresponsive to nitroglycerin.
  - May respond to recumbent position (pathognomonic but rare).
- Syncope:
  - Most often occurs following exercise—*decreased afterload* due to peripheral vasodilation resulting in peripheral pooling since muscular contractions no longer enhance return to heart causing *decreased preload*.
  - Arrhythmias: atrial fibrillation, ventricular tachycardia.
- Palpitations due to arrhythmias.
- Signs of CHF.
- Pulsus bisferiens (rapid biphasic carotid pulse).
- S_4 gallop.
- Systolic ejection murmur heard best along the left sternal border, decreases with increased LV blood volume (squatting), increases with increased blood velocities (exercise), and decreased LV end-diastolic volume (Valsalva).
- Paradoxical splitting of S_2.
- Sudden death is usually due to an arrhythmia rather than obstruction.

DIAGNOSIS

- ECG: LVH, PVCs, atrial fibrillation, septal Q waves, nonspecific ST segment and T wave abnormalities.
- Echocardiography: Septal hypertrophy, LVH but small LV size, atrial dilatation, mitral regurgitation with systolic anterior motion of mitral valve leaflets.

TREATMENT

- Beta blockers to reduce heart rate, increasing LV filling time and decreasing inotropy are first line; calcium channel blockers considered second line.

- Anticoagulation for A-fib or signs of peripheral embolization.
- Septal myomectomy or transcatheter alcohol septal ablation for severely symptomatic patients.
- Permanent pacemaker to change pattern of ventricular contraction, reducing obstruction.
- Implanted automatic defibrillator should be considered.
- Bacterial endocarditis prophylaxis for dental, gastrointestinal, and genitourinary procedures.
- Vigorous exercise should be discouraged.

Hypertrophic cardiomyopathy: Digitalis and other positive inotropic agents are contraindicated even if presenting symptoms are those of CHF (because they increase outflow obstruction).

▶ ACUTE BACTERIAL ENDOCARDITIS (ABE)

DEFINITION

Bacterial infection of endocardium.

ETIOLOGY

Duke Criteria
1. Positive blood culture for infective endocarditis (IE):
   - Isolation of typical organisms from two positive cultures of blood samples drawn > 12 hours apart, or all of three or a majority of four separate cultures of blood (with first and last sample drawn 1 hour apart).
   - Typical microorganisms consistent with IE include viridans streptococci, *Streptococcus bovis*, HACEK (*Haemophilus aphrophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens,* and *Kingella kingae*) group, or community-acquired *Staphylococcus aureus* or enterococci, in the absence of a primary focus.
2. Evidence of endocardial involvement as evidenced by positive echocardiogram for IE defined as:
   - Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation, or
   - Abscess, or
   - New partial dehiscence of prosthetic valve

RISK FACTORS

- Congenital heart disease
- Valvular heart disease
- Prosthetic valve
- IV drug abuse
- Indwelling venous catheters
- Dialysis
- Previous history of IE

Frequency of valves affected in ABE: aortic > mitral >> tricuspid

A 28-year-old female with a prosthetic valve presents with fever. Physical exam reveals a systolic ejection murmur and retinal hemorrhages. *Think: Bacterial endocarditis.*

CLINICAL PRESENTATION

- Fever: temperature > 38.0°C (100.4°F)
- Arthralgias
- Pleuritic chest pain
- Vascular phenomena: Major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, splinter hemorrhages, and **Janeway lesions** (nontender palmar plaques)

■ Immunologic phenomena: Glomerulonephritis, **Osler's nodes** (tender fingertip nodules), **Roth spots** (retinal hemorrhages)

TREATMENT (PER AHA 2005 GUIDELINES)

- ■ Therapy for native valve endocarditis:
  - ■ First line (class IA recommendation): **Penicillin G** 12–18 million U/24 h IV either continuously or in four or six equally divided doses. Preferred in most patients > 65 years old or patients with impairment of eighth cranial nerve function or renal function; or **ceftriaxone** 2 g/24 h IV/IM in one dose. Therapy given for 4 weeks.
  - ■ Alternative for allergic patients: Vancomycin.
- ■ Therapy for prosthetic valve endocarditis:
  - ■ Class IB recommendation: **Penicillin G** 24 million U/24 h IV either continuously or in four to six equally divided doses; or **ceftriaxone** 2 g/24 h IV/IM in one dose with or without **gentamicin** 3 mg/kg/24 h IV/IM in one dose. Therapy given for 6 weeks.
  - ■ Alternative therapy for penicillin- and cephalosporin-allergic patients: Vancomycin.

Echo for ABE:
TEE: 90 to 100% sensitive
TTE: 28 to 63% sensitive

> ▶ **HEART FAILURE**

DEFINITION

Condition characterized by inadequate systemic perfusion to meet body's metabolic demands due to "failure" of the heart's pump function. Also known as congestive heart failure (CHF).
- ■ Systolic failure: Reduced cardiac contractility.
- ■ Diastolic failure: Impaired cardiac relaxation and abnormal ventricular filling.

EPIDEMIOLOGY

United States
- ■ Prevalence is 4.7 million (~ 1.5% of the population).
- ■ There are 1 million hospital admissions per year in which CHF is the primary diagnosis, and another 2 million hospitalizations with heart failure as a secondary diagnosis.
- ■ One-third of these patients are readmitted within 90 days for recurrent decompensation (see next section).

Worldwide
- ■ It is estimated that there are 15 million new cases of CHF per year.
- ■ Aging population contributes to the increasing incidence.

PATHOPHYSIOLOGY

Systolic Dysfunction
1. Cardiac output is low, pulmonary pressures are high.
2. This leads to pulmonary congestion and systemic hypoperfusion.
3. Neurohormonal pathways activated to increase circulating blood volume.

4. Sympathetic nervous system increases heart rate and contractility, leading to increased cardiac output.

5. Catecholamine release also aggravates ischemia; can potentiate arrhythmias; promotes cardiac remodeling (left ventricular [LV] dilation and hypertrophy); and stimulates renin–angiotensin system, which exacerbates arteriolar vasoconstriction, causing retention of sodium and water.

6. Clinical symptoms of CHF appear when these mechanisms can no longer compensate.

Diastolic Dysfunction

1. Main problem is impaired LV relaxation. This causes high diastolic pressures and poor filling of the ventricles.

2. In order to increase diastolic filling, left atrial pressure increases until it exceeds the hydrostatic and oncotic pressures in the pulmonary capillaries.

3. Pulmonary edema results.

4. Patients are therefore symptomatic with exertion when increased heart rate reduces LV filling time.

5. Circulating catecholamines worsen diastolic dysfunction.

6. Clinical symptoms of CHF appear when these mechanisms can no longer compensate.

ETIOLOGY

- LV **systolic** dysfunction:
  - Most common cause (~ 60%)
  - History of MIs and chronic underperfusion
  - Valvular heart disease
  - Hypertension
  - Dilated cardiomyopathy
    - Toxins (e.g., alcohol, doxorubicin, lithium, cocaine)
    - Heavy metals (lead, arsenic, mercury)
    - Viral (e.g., coxsackie)
    - Familial predisposition/congenital disease
    - Neuromuscular disease
- Right ventricular (RV) **systolic** dysfunction:
  - LV systolic dysfunction
  - RV infarction
  - Pulmonary HTN
  - Chronic severe tricuspid regurgitation
  - Arrhythmogenic RV dysplasia
- LV **diastolic** dysfunction:
  - Restrictive cardiomyopathy
    - Hemochromatosis
    - Sarcoidosis
    - Carcinoid heart disease (e.g., Gaucher, Hurler, glycogen storage diseases)
  - Hypertrophic cardiomyopathy
  - Infiltrative cardiomyopathy (e.g., amyloidosis)
  - Pericardial constriction
  - Cardiac tamponade
  - High-output states: Pregnancy, thyrotoxicosis, wet beriberi, arteriovenous fistulae, Paget's disease, severe chronic anemia

Right ventricular failure manifests as peripheral vascular congestion (pedal edema, JVD, hepatomegaly).

LV failure manifests as pulmonary edema or shock (dyspnea, orthopnea, hypotension).

SIGNS AND SYMPTOMS

Left Heart Failure
Orthopnea
Paroxysmal nocturnal dyspnea
Dyspnea on exertion
Rales
Cough (sometimes frothy pink); hemoptysis; wheezing ("cardiac asthma")
S_3 and/or S_4 heart sound
Tachycardia
Diaphoresis

Right Heart Failure
RUQ pain (due to hepatic congestion
Hepatomegaly with mild jaundice
Hepatojugular reflex
Jugular venous distension (JVD)
Ascites
Peripheral cyanosis
Peripheral edema (pitting)
Nausea
Wasting
Oliguria and nocturia

DIAGNOSIS

- ECG:
    - Pattern may reveal normal sinus, sinus tachycardia, or atrial fibrillation.
    - Findings commonly seen in CHF include LV hypertrophy, LBBB, intraventricular conduction delay, and nonspecific ST segment and T wave changes. Presence of Q waves suggest old MI.
- CXR: Often reveals cardiomegaly, pulmonary vascular redistribution, pulmonary venous congestion, Kerley B lines, alveolar edema, and pleural effusions.
- Echocardiogram:
    - Most useful to distinguish between systolic and diastolic dysfunction.
    - Can discern regional wall motion abnormalities, LV aneurysm, ejection fraction, diastolic function, valvular problems.
- Plasma brain natriuretic peptide (BNP): BNP is available as a rapid bedside test and is elevated in decompensated CHF, and can be useful in new diagnoses, before compensated state occurs.

CLASSIFICATION

See Table 8-3.

TABLE 8-3. **New York Heart Association (NYHA) Heart Failure Symptom Classification System**

NYHA CLASS	LEVEL OF IMPAIRMENT
I	No symptom limitation with ordinary physical activity
II	Ordinary physical activity somewhat limited by dyspnea (i.e., long-distance walking, climbing two flights of stairs)
III	Exercise limited by dyspnea at mild workloads (i.e., short-distance walking, climbing one flight of stairs)
IV	Dyspnea at rest or with very little exertion

- Patients are maintained on a variety of cardiac drugs to optimize cardiac output; scope is beyond this book.
- Ultimately, patients with end-stage heart failure will require heart transplantation.
- Circulatory assist devices can provide a bridge to heart transplantation.

PROGNOSIS

- Poor prognosis is associated with ventricular arrhythmias, NYHA Class III or IV, lower LV ejection fraction, marked LV dilatation, high catecholamine and BNP levels, low serum sodium, and hypocholesterolemia.
- Patients with combined systolic and diastolic LV dysfunction also have a worse prognosis than patients with either in isolation.

Circulatory Assist Devices

Circulatory assist devices are mechanical devices that allow an increase in myocardial oxygen supply and decrease the workload of the left ventricle, thereby increasing cardiac output and perfusion to vital organs. There are three main types of circulatory assist devices:

- Counterpulsation devices such as intra-aortic balloon pump (IABP)
  - Most commonly used.
  - Percutaneous insertion (most often through femoral artery).
  - Balloon is placed within the descending aorta.
  - Device is designed to inflate during diastole and deflate during systole, synchronized with native heartbeat.
- Cardiopulmonary assist devices
- LV assist devices
  - Centrifugal pumps: Limits ambulation
  - Extracorporeal pumps: Require heparinization

Indications
- Cardiogenic shock
- Intractable angina
- Weaning from cardiopulmonary bypass
- As adjunctive therapy after thrombolysis in patients at high risk for stenosis
- As prophylactic therapy in patients with severe left main coronary arterial stenosis or critical aortic stenosis in whom surgery is pending

Contraindications
- Aortic valve regurgitation
- Aortic aneurysm
- Severe peripheral vascular disease
- Coagulopathy
- Uncontrolled sepsis
- Aortic stenosis or prosthetic aortic valve (LV assist device only)

HIGH-YIELD FACTS

Cardiovascular Emergencies

**Acute Decompensated Heart Failure
(Cardiogenic Pulmonary Edema)**

DEFINITION

Also known as acute cardiogenic pulmonary edema.

ETIOLOGY

- Most common reason is noncompliance with medications
- Dietary indiscretion (excess salt intake)
- Acute MI
- Sepsis

DIAGNOSIS

- CXR: Cardiomegaly, bilateral interstitial infiltrates ("hazy" appearance).
- ECG: Often sinus tachycardia or atrial fibrillation.
- Laboratory studies: Obtain CBC, electrolytes, cardiac biomarkers (elevated in acute MI) and BNP (elevated in acute decompensated CHF).

TREATMENT

- First line:
  - Oxygen: 100% O_2 by face mask to maintain O_2 saturation above 90%. In patients for whom this is inadequate, bilevel positive airway pressure (BiPAP) is an excellent option. Often, use of BiPAP will obviate the need for endotracheal intubation, and is considered first line where available.
  - Aspirin: Given as part of protocol to rule out acute coronary syndrome, 160 to 352 mg PO.
  - Morphine: Decreases work of breathing and reduces anxiety. Overall, this causes decrease in sympathetic discharge and leads to arterioloar and venous dilatation and decreased cardiac filling pressures. Given IV at 4 mg initially, followed by 2- to 4-mg titrated increments.
  - Nitroglycerin: Reduces preload and thus pulmonary capillary wedge pressure. Can be given in the transdermal (nitropaste 1 to 2 inches), SL (0.4 mg q 5 minutes) or IV form (5 µg/min, titrated to effect).
  - Diuretics: Loop diuretics (e.g., furosemide, bumetanide) are used to produce venodilation, which reduces pulmonary capillary pressure by causing diuresis. With furosemide (40 to 80 mg IV), peak onset of diuresis occurs in about 30 minutes and lasts about 6 hours.
- Second line: If the above modalities do not reslve the pulmonary edema, then the following agents can be used to boost inotropic support.
  - Milrinone: A phosphodiesterase inhibitor that inhibits degradation of cyclic adenosine monophosphate (cAMP). Causes reduced systemic vascular resistance, and improved LV diastolic compliance. Loading dose is 50 µg/kg over 10 minutes, followed by in fusion of 0.375 to 0.750 µg/kg/min.
  - Dobutamine: Increases stroke volume and cardiac output by mainly working on β_1 receptors with minimal effect on α_1 receptors. Initial dose is 2.5 µg/min, gradually increased to 7 to 20 µg/kg/min.
  - Dopamine: Reserved for hypotensive patients in whom arterial pressure increase is required, due to stimulation of α_1-adrenergic receptors

in addition to β_1 (cardiac) receptors. "Cardiac dosing" is 3 to 10 μg/min.

- Other pharmacologic therapy: Nesiritide is recombinant human B-type (brain) natriuretic peptide. It produces vasodilatory, natriuretic, and diuretic effects, primarily mediated via natriuretic peptide receptor A on vascular smooth muscle, endothelium, kidneys, and adrenals. It has no direct inotropic effect. Its use is limited to those who are hospitalized for severe CHF, who are not in cardiogenic shock, in whom first-line therapy in inadequate.

DEFINITION

Inflammatory damage of myocardium.

PATHOPHYSIOLOGY

- Myocyte necrosis/degeneration and correlating inflammatory infiltrate due to infectious and inflammatory etiologies.
- Some infectious agents cause an autoimmune response to cardiac myocytes by molecular mimicry.
- Some cases spontaneously resolve.
- Some cases progress to end-stage dilated cardiomyopathy.

ETIOLOGIES

- Viral (coxsackie B4, adenovirus, influenza A and B, varicella-zoster virus, HIV, cytomegalovirus, hepatitis A and B, Epstein–Barr virus)
- Vaccine related
- Bacterial *(Mycoplasma, Streptococcus, Chlamydia)*
- Lyme disease *(Borrelia burgdorferi)*
- Chagas' disease *(Trypanosoma cruzi)*
- Kawasaki disease
- Steroid abuse

CLINICAL FINDINGS

- Fever
- Chest pain
- Tachycardia out of proportion to fever
- Syncope
- Dyspnea
- Fatigue
- Palpitations
- Soft S_1
- S_3 or S_4 gallop
- Mitral or tricuspid regurgitation murmur

A 29-year-old male presents with fever and retrosternal chest pain. He had "the flu bug" 2 weeks ago. He is tachycardic. *Think: Myocarditis.*

DIAGNOSIS

- CXR: ± Cardiomegaly/pulmonary edema.
- ECG: Sinus tachycardia, low voltage, long QT/PR/QRS, AV blocks, increased or decreased STs, decreased Ts.

- Labs: Increased ESR, increased WBC count, increased CK-MB, increased troponin.
- Echocardiography: Multichamber dysfunction, decreased left ventricular ejection fraction, global hypokinesis, focal wall motion abnormality.

TREATMENT

- Intensive care unit admission.
- Bed rest, supportive care, vital signs.
- Antibiotics for bacterial and parasitic causes.
- ASA and gamma globulin for Kawasaki disease.
- ACE inhibitors for CHF associated with myocarditis.

In new diagnosis of refractory asthma in a young adult, consider myocarditis.

▶ PERICARDITIS

Constrictive

- Fibrous reparative thickening of pericardial layers (sometimes calcified) that restricts diastolic ventricular filling.
- Caused by trauma, uremia, tuberculosis (TB), radiation.

Acute Inflammatory

Inflammation of pericardial tissue resulting in pain and effusion. Causes include:
- Trauma
- Uremia
- Infectious (viral > bacterial > parasitic > fungal)
- Post irradiation
- Post MI
- Aortic dissection
- Tumors

A 31-year-old female presents with pleuritic chest pain that improves with leaning forward. Cardiac auscultation reveals a pericardial friction rub. ECG demonstrates diffuse PR depression. *Think: Pericarditis.*

CLINICAL FINDINGS

- Fever
- Pleuritic and positional chest pain
- Tachycardia
- Myalgias
- Shallow breathing
- Anxiety
- Pericardial friction rub
- Distant heart sounds

DIAGNOSIS

- CXR: May see cardiomegaly if effusion present.
- ECG (see Figure 8-18):
  - Stage 1 (first few hours/days): Diffuse ST elevations with PR depression
  - Stage 2: Normalization of STs and PRs
  - Stage 3: Diffuse T wave inversions
  - Stage 4: Normalization of T waves
- Labs: Increased ESR and WBC counts, + CK and troponin if concomitant myocarditis or endocarditis; check BUN/Cr, blood cultures.

FIGURE 8-18. ECG in pericarditis.

Note diffuse ST elevations with PR depression, early stage of pericarditis. ECG will change and normalize as disease progresses and resolves.

- Echocardiography: Normal global cardiac function unless an effusion is present.

TREATMENT

- Rule out ACS.
- NSAIDs for viral, post MI, and idiopathic causes.
- Antimicrobials for bacterial, fungal, TB, and parasitic causes.
- Surgical pericardiectomy for purulent pericarditis.
- Dialysis for uremic pericarditis.

▶ PERICARDIAL EFFUSIONS

DEFINITIONS

- Pericardial effusion: Excessive fluid in pericardial space.
- Cardiac tamponade: Pericardial effusion that restricts ventricular filling and eventually stroke volume, leading to systemic hypotension, shock, PEA, and death.

Always rule out ACS in a patient presenting with classical signs of pericarditis.

Cardiac tamponade is one of the causes of pulseless electrical activity (PEA).

Beck's triad:
- Hypotension
- JVD
- Muffled heart sounds

ETIOLOGIES

Same as pericarditis.

CLINICAL FINDINGS

- Same as pericarditis, but alterations in vital signs may be more pronounced and shock state may exist.
- Often asymptomatic when small.
- Beck's triad for cardiac tamponade.

DIAGNOSIS

- CXR: Cardiomegaly (see Figure 8-19).
- ECG: Differing QRS amplitudes ("alternans," Figure 8-20) and axes caused by ventricle swaying within fluid-filled pericardial sac with each beat.
- Echocardiography: Effusion, decreased systolic and diastolic function, collapse of right ventricular/right atrial free walls in diastole.

TREATMENT

- ABCs, IV, O$_2$, monitor.
- Pericardiocentesis immediately if hemodynamically unstable or pulseless (see Procedures chapter).
- If more stable, a pericardial window can be created in the operating room (OR) to prevent reaccumulation of effusion.

F I G U R E   8 - 1 9 .   CXR demonstrating cardiomegaly secondary to pericardial effusion.

FIGURE 8-20. ECG demonstrating electrical alternans. Note alternating heights in the R (arrow) in the QRS complexes.

Aortic Stenosis

DEFINITION

Valve hardening obstructs blood flow from left ventricle. Results in progressive LVH, decreased cardiac output, hypertrophic and later dilated cardiomyopathy. Predisposes to endocarditis.

ETIOLOGY

- Congenital
- Rheumatic fever
- Degenerative calcification

SIGNS AND SYMPTOMS

Dyspnea on exertion, angina, syncope on exertion, sudden death, low-pitched crescendo–decrescendo murmur at the base radiating to carotids, carotid pulse weak (parvus) and slow-rising (tardus), S_3, S_4.

DIAGNOSIS

- CXR: Cardiomegaly ± pulmonary edema.
- ECG: LVH ± ischemic change.
- Echocardiography: Can estimate severity of obstruction and LV systolic function, and may identify cause (e.g., calcified bicuspid valve).

TREATMENT

- Definitive treatment is valve replacement.
- Acute presentation warrants ruling out ACS, CHF, and other etiologies.
- ABCs, IV, O_2, monitor.
- Gentle hydration if hypotensive.

> Prognosis: *Mean survival for patients with AS and:*
> Angina = 5 years
> Syncope = 2 to 3 years
> Heart failure = 1 to 2 years

> Any change in preload *or* afterload can cause acute decompensation in AS.

Aortic Insufficiency

DEFINITION

Regurgitation of blood flow back into the ventricle, leading to dilated cardiomyopathy and failure.

ETIOLOGY

Acute Causes
- Infective endocarditis
- Aortic root dissection

Chronic Causes
- Rheumatic fever
- Congenital
- Ankylosing spondylitis
- Syphilis
- Carcinoid
- Reiter's syndrome
- Fen-Phen (fenfluramine and phentermine) use

In patients with AS presenting with angina, rule out ACS.

SIGNS AND SYMPTOMS

- Dyspnea.
- Angina.
- Presence of S_3 heart sound.
- High-pitched blowing diastolic murmur at base, ± systolic flow murmur.
- "Water-hammer" pulse: Peripheral pulse with quick upstroke and then collapse.
- Wide pulse pressure.
- Bounding "Corrigan" pulse, "pistol shot" femorals, pulsus bisferiens (dicrotic pulse with two palpable waves in systole).
- Duroziez 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 mm Hg higher than in the arms.
- Quincke's sign: Visible capillary pulse in nails.
- De Musset's sign: Bobbing of head with heartbeat.

A 70-year-old male presents with angina. Physical exam reveals bounding pulses and there is an SBP difference of 25 mm Hg between the upper and lower extremities. *Think: Aortic insufficiency.*

DIAGNOSIS

- CXR:
  - Chronic—cardiomegaly ± pulmonary edema.
  - Acute—pulmonary edema without cardiomegaly.
- ECG:
  - Chronic—LVH ± strain pattern.
  - Acute—± ischemic change (especially inferior leads), low voltage, if dissection—tachycardia.
- Echocardiography: Will diagnose disease by visualizing regurgitant flow, may identify cause (e.g., vegetations), and facilitates assessment of LV systolic function and chamber size.

TREATMENT

- ABCs, IV, O_2, monitor.
- In acute and chronic cases of pulmonary edema, reduce afterload with nitrates and diuretics.
- Treat endocarditis as indicated.
- Dissection treated with surgical repair.
- Valve replacement is indicated once LV becomes enlarged or systolic function is impaired.

Mitral Stenosis

DEFINITION

Decrease in cross-sectional area for blood flow from left atrium to left ventricle, resulting in atrial dilatation, atrial fibrillation, left heart failure, progressive pulmonary HTN, pulmonic and tricuspid valve regurgitation, and right heart failure.

ETIOLOGY

- Rheumatic fever
- Atrial myxomas
- Congenital
- Degenerative calcification

SIGNS AND SYMPTOMS

- Dyspnea on exertion
- Orthopnea
- Early diastolic opening snap followed by diastolic rumble at the apex

DIAGNOSIS

- CXR: Can be normal; severe disease can show a straightening of the LV border ± pulmonary edema. It may reveal double density sign, straightening and lifting of carina.
- ECG: Left atrial enlargement ± atrial fibrillation, right axis deviation.
- Echocardiography: Will diagnose disease by showing thickened valve leaflets, decreased valve movement, and commissural fusion.

TREATMENT

- ABCs, IV, O_2, monitor.
- Acute atrial fibrillation: See above.
- Pulmonary edema: Nitrates, diuretics, oxygen, and morphine.
- Surgical valvulotomy or valve replacement is indicated when significant symptoms develop despite medical treatment or if pulmonary hypertension develops.

Mitral Regurgitation

DEFINITION

Regurgitation from LV to LA during systole, results in increased LV stroke volume with eventual LV dilatation and dysfunction.

ETIOLOGY

Acute Causes
- MI with ischemic necrosis and subsequent rupture of papillary muscle or chordae tendineae usually from right coronary infarct.
- Infective endocarditis.
- Trauma.

Chronic Causes
- Rheumatic fever heart damage.
- Appetite suppressant drugs (Fen-Phen).

- Mitral valve prolapse.
- Carcinoid tumor syndrome.
- Marfan's syndrome.
- Any cause of LV dilatation can cause *secondary* MR.

SIGNS AND SYMPTOMS

- Dyspnea.
- Tachycardia and tachypnea.
- Angina.
- Presence of S_3 and S_4 heart sounds.
- Loud crescendo–decrescendo murmur between S_1 and S_2 at the apex radiating to axilla.
- Rales.
- Rapidly rising and poorly sustained carotid pulse.

DIAGNOSIS

- CXR:
  - Chronic: Cardiomegaly ± pulmonary edema.
  - Acute: Pulmonary edema without cardiomegaly.
- ECG:
  - Chronic: LVH, atrial enlargement, A-fib.
  - Acute: ± Ischemic change, tachycardia without chronic changes.
- Echocardiography: Can diagnose acute and chronic cases by visualizing chordae tendineae, vegetations, wall motion abnormality, and estimating severity of disease (volume of MR jet, LV size and function).
- Cardiac catheterization: Indicated in acute cases to evaluate and treat ACS.

TREATMENT

- ABCs, IV, O_2, monitor.
- Nitrates, morphine, and diuretics for pulmonary edema, reducing afterload and regurgitant flow.
- Antibiotics for infectious endocarditis (IE).
- Catheterization and emergency mitral valve reconstruction for ischemic rupture.
- ACE inhibitors, long-acting nitrates, and salt restriction for chronic disease.

Artificial Valves

- Mechanical: Bileaflet hinged disk, tilting disk, or caged-ball prostheses:
  - Patients require lifelong anticoagulation (warfarin).
  - Monitor INR (usual range 2 to 3).
  - "Mechanical murmur" systolic with loud, closing machine-like sound.
  - Complications: Chronic low-grade hemolysis from turbulent flow and subsequent anemia, valve failure, thrombosis, systemic emboli, bleeding from high INR, risk for IE (contamination and bacteremic).
- Bioprosthetic: Bovine or porcine:
  - ASA only anticoagulation.
  - Mitral bioprosthesis has diastolic rumble.

Loss of the click of the mechanical valve may indicate valvular dysfunction, infection, dehiscence, or abscess formation.

- Complications: 30 to 70% failure rate at 10 years, risk for IE (contamination and bacteremic), valve failure, thrombosis, systemic emboli.

▶ HEART TRANSPLANT

- Denervated hearts have no native sympathetic and parasympathetic tone, responding only to circulating catecholamines and medications, so don't try any vagal maneuvers or atropine (which inhibits vagal tone).
- All transplant patients are immunocompromised. If they present with fever, diagnose and treat aggressively with broad-spectrum antibiotics.
- With piggyback heart transplant, you may see two separate independent P waves in the ECG, one from the old heart and one from the new heart.
- Before prescribing anything for the transplant patient as an outpatient, find out if it will interact with his or her immunosuppressant medication.
- Dysrhythmias, atypical fatigability, and exertion intolerance should be treated as acute rejection until proven otherwise. If hemodynamically unstable because of the dysrhythmias, acute rejection regimen should be instituted (methylprednisolone 1 g IV), and modified ACLS protocols should be followed.

Abdominal aneurysms *rupture;* thoracic aneurysms *dissect.*

▶ AORTIC ANEURYSM

Abdominal Aortic Aneurysm (AAA)

PATHOPHYSIOLOGY

- Atherosclerotic, thinned tunica media has decreased elastin fibers and forms aneurysm from HTN.
- The larger the aneurysm, the weaker the wall, and therefore gradual enlargement of AAA.

A 73-year-old male who is a 2-pack-per-day smoker with HTN and peripheral vascular disease presents with severe midabdominal and left flank pain. He states he had this same pain 1 week ago, and that it got so bad he passed out. Physical exam reveals bruits over the abdominal aorta and a tender pulsatile mass. *Think: AAA.*

RISK FACTORS

- Age > 60
- Male gender
- HTN
- Cigarette smoking
- Coronary artery disease
- Peripheral vascular disease
- Family history of AAA in first-degree relative

SIGNS AND SYMPTOMS

- Abdominal pain (77%)
- Pulsatile abdominal mass (70%)
- Back/flank pain (60%)
- Tender abdomen (41%)

AAA is most frequently misdiagnosed as renal colic.

- Nausea/vomiting (25%) with blood (5%)
- Syncope (18%)
- Nonpalpable distal pulses (6%)
- Known history of AAA (5%)

The mortality is 50% for those who rupture and get to the OR.

DIAGNOSIS

- CXR: Can be normal.
- Abdominal x-ray: May see calcified outline of AAA.
- ECG: Tachycardia ± ischemic changes.
- Ultrasound: Ideal for the unstable patient because of machine portability; however, bowel gas and obesity may obscure visualization.
- Computed tomography (CT) scan: Only for hemodynamically stable patients. Contrast allows full evaluation for both aneurysmal size and possible dissection.
- Magnetic resonance imaging (MRI)/magnetic resonance angiography (MRA): Best for the asymptomatic patient. Extremely useful in evaluation of patients in whom IV contrast is contraindicated.

TREATMENT

- ABCs, IV access (two large bore), O$_2$, monitor.
- IV fluid if in a shock state.
- In unstable patients, rapid transport to OR with vascular surgeon.

Aortic Dissection

DEFINITION

A tearing of the aorta due to hypertensive "shearing forces" on an atherosclerotic vessel that infiltrate through the intima and track or "dissect" between the intima and adventitial layers. Dissection can occur proximally or distally and can involve other vessels (carotids, renals, iliacs, pericardium).

CLASSIFICATION

- DeBakey classification (anatomic):
  - Type I: Ascending and descending
  - Type II: Ascending only
  - Type III: Descending only
- Stanford classification (more widely used clinically):
  - Type A: Ascending aorta
  - Type B: Descending aorta

RISK FACTORS

- HTN
- Connective tissue disease (Marfan's, Ehlers–Danlos)
- Male gender (three times more affected than women)
- Congenital heart disease
- Third-trimester pregnancy
- Turner's syndrome
- Cocaine use

SIGNS AND SYMPTOMS

- Abrupt onset of pain that is maximal at onset and migrates:
    - Type I: Pain begins in anterior chest and radiates to jaw, neck, or arms.
    - Type II: Pain begins between the scapulae and radiates to the abdomen and lumbar area.
- Elevated BP.
- Tachycardia.
- Shock.
- Focal neurological deficits:
    - Stroke-like syndrome if carotid involvement.
    - Hoarse voice if there is compression of recurrent laryngeal nerve.
    - Horner's syndrome if superior cervical sympathetic ganglion is compressed.
- ≥ 20 mm Hg BP difference between upper and lower extremities.
- Aortic insufficiency murmur.
- May present with cold pulseless extremity.

DIAGNOSIS

- ECG: LVH with strain pattern, ± ischemic change if dissection into coronaries or if MI, low voltage if effusion, electrical alternans if tamponade.
- CXR: Mediastinal widening (75%) (see Figure 8-21), calcification of aortic arch, displacement of trachea and/or nasogastric tube to one side.
- TEE: Diagnostic study of choice with almost 100% sensitivity and specificity. Can differentiate between true and false lumens. Does not require IV contrast. May not however, be readily available. Other limitations include difficult positioning and poor and incomplete visualization in certain patients.
- MRI/MRA: Also close to 100% sensitivity and specificity, but is time

A 67-year-old male with history of HTN presents with a sudden-onset excruciating chest pain radiating to the left jaw. Physical exam reveals an aortic insufficiency murmur, a BP of 217/110, and cool clammy skin. *Think: Thoracic dissection (type I).*

Thoracic dissections are most frequently misdiagnosed as AMIs.

Always get at least a chest film when you suspect MI: Some of these patients will have aortic dissection, and thrombolysis may kill them.

FIGURE 8-21. **CT chest demonstrates aortic dissection involving ascending and descending aorta (Stanford A).**

consuming, allows limited access to patient during scan. Noninvasive, no contrast dye needed.

- CT scan: Rapid dynamic scans of multiple levels of chest immediately following IV bolus of contrast (67 to 85% sensitivity). May not always readily detect communication between true and false lumen.
- Aortography: Used to be gold standard. Invasive, requires contrast dye. About 90% sensitive and specific. Can miss thrombosed false lumens. Time consuming, takes patient out of ED suite.

TREATMENT

- ABCs, IV access (two large bore), O_2, monitor.
- Antihypertensive medications (decrease shearing force), labetalol IV 0.25 mg/kg (or 20 mg) over 2 minutes and then nitroprusside 0.3 to 10 μg/kg/min.
- Immediate surgical consultation: Go to OR for repair if patient is unstable or hypotensive.
- Ascending involvement = repair.
- Descending involvement unless medical management.

> ▶ **PERIPHERAL ARTERIAL OCCLUSION**

PATHOPHYSIOLOGY

- A blockage in arterial flow compromises tissue distally, resulting in irreversible cell death within 4 to 6 hours.
- Without rapid aggressive treatment, it can lead to gangrene, limb amputation, and death.
- Embolic sources (e.g., thrombus of cardiac origin breaks off and travels distally) and nonembolic sources (e.g., atherosclerosis and plaque rupture with thrombus occlusion, vasospasm, and/or arteritis).

RISK FACTORS

- HTN
- Smoking
- High cholesterol
- Diabetes
- Recent MI or atrial fibrillation
- Aortic aneurysm

CLINICAL FINDINGS

- Abrupt onset of pain in leg which may be known to have poor circulation.
- The 6 Ps may not all be present.
- Use handheld Doppler to try finding nonpalpable pedal pulse.

DIAGNOSIS

- ECG: Atrial fibrillation or atrial flutter, or sinus rhythm, LVH.
- CXR: ± Cardiomegaly.
- Lower limb vascular imaging (femoral angiogram, MR angiography).

The most specific CXR sign for thoracic dissection is extension of the aortic shadow by more than 5 mm beyond its calcified aortic wall.

The 6 Ps of peripheral arterial occlusion:
- **Pain**
- **Pallor**
- **Polar (coldness)**
- **Pulselessness**
- **Paresthesias**
- **Paralysis**

TREATMENT

- ABCs.
- Immediate vascular surgery consult for consideration of thrombectomy.
- IV heparinization if no contraindications.

Hypertensive Urgency

- SBP ≥ 180 mm Hg, DBP ≥ 110 mm Hg without evidence of end-organ damage.
- Most common cause is noncompliance with medications.
- Treatment: Oral agents.

Hypertensive Emergency

DEFINITION

HTN that causes end-organ damage.

SIGNS AND SYMPTOMS

Signs of end-organ damage:
- Brain:
  - Hypertensive encephalopathy—loss of integrity of the blood–brain barrier and resulting cerebral edema.
  - Intracerebral hemorrhage—a result of long-standing HTN, vascular disease, or aneurysm rupture.
- Eye: Hypertensive retinopathy—"cotton-wool" spots (focal ischemia), hemorrhage, and papilledema (optic disc edema from hypoxia).
- Heart: LV failure and pulmonary edema due to increased afterload.
- Kidney: Acute renal failure—causes and is caused by HTN:
  - Pregnancy: Eclampsia (see obstetrics section).
  - Vascular: Aortic dissection (see above).

DIAGNOSIS

- Labs: Elevated BUN and Cr, urinalysis (for red blood cells [RBCs], protein, and casts), cardiac enzymes if chest pain or pulmonary edema, CBC, electrolytes.
- CXR: ± Pulmonary edema, ± cardiomegaly.
- ECG: LVH, ischemic change.
- CT head: ± Bleed/edema.

TREATMENT

- Reduce the mean arterial pressure by no more than 25% within minutes to 1 hour.
- For stable patients, initial reduction should be followed by further reduction toward a goal of 160/110 mm Hg within 2 to 6 hours.
- Parenteral therapy is preferred because of rapidity of action and ease of titration. Also, the treatment can be stopped if the patient becomes hypotensive.

The *mean arterial pressure* is: (2DBP + SBP)/3

Nitroprusside can cause cyanide toxicity.

A 47-year-old female presents with BP of 200/130. She is a known hypertensive but admits to being noncompliant with her medications. Physical examination is unremarkable. *Think: Hypertensive urgency.*

- Common intravenous agents: Vasodilators (nitroprusside, nicardipine, hydralazine, enalapril, fenoldopam); adrenergic inhibitors (labetalol, esmolol, phentolamine).
  - Nitroprusside: A powerful direct vasodilator. Prolonged use may cause cyanide toxicity.
  - Nicardipine: Antihypertensive response is comparable to nitroprusside, with much fewer side effects.
  - Labetalol: A combined alpha and beta blocker—an excellent agent of choice. The main drawback is prolonged duration of action.
- Remember, hypertensive emergency is a state of volume depletion, so diuretics should be avoided unless specifically indicated.

▶ VENOUS INSUFFICIENCY

PATHOPHYSIOLOGY

- Incompetent valves in peripheral venous system (~90% lower extremities) cause "venous stasis" of peripheral blood, microextravasation of RBCs, and fluid causing pigment (hemosiderin) deposition in local tissues (stasis dermatitis) and pitting edema.
- Stasis in turn can lead to poor wound healing and intravascular thrombosis (see below).

TREATMENT

- Advise avoidance of prolonged periods of standing/working on feet.
- Elevate legs when resting.
- Wear gradient compression hose.
- A mild diuretic in a low dose may be helpful.

▶ THROMBOPHLEBITIS

DEFINITIONS

- DVT: Involves the deep venous system, typically calf, popliteal, femoral, common femoral, and iliac.
- Superficial thrombophlebitis can be present at the same time as DVT and can occur in any superficial vein. Varicose veins are a predisposing factor.

PATHOPHYSIOLOGY

- Intravascular (intravenous) spontaneous clot (thrombosis) and concurrent surrounding inflammatory response to that clot.
- Clot can dissolve, propagate, or embolize to a distal site (creating pulmonary embolus or a paradoxic cerebrovascular accident [CVA] through ASD/VSD).

RISK FACTORS

- Prior DVT
- Pregnancy or postpartum state
- Malignancy

A 39-year-old female who arrived home from her 18-hour car ride the previous evening presents with right calf swelling and pain. Physical exam reveals the right calf to be 4 cm larger than the left, and it is warm to the touch. *Think: DVT.*

- Prolonged immobility
- IV drug abuse
- Recent trauma or burns
- Coronary artery disease
- Polycythemia vera
- Thrombocytosis
- Antithrombin III, protein C or S deficiency
- Aquired immune deficiency syndrome
- Autoimmune disease (e.g., systemic lupus erythematosus)
- Indwelling catheter

CLINICAL FINDINGS

- Unilateral swelling and pitting edema of a lower extremity.
- Redness, pain, heat (very similar to cellulitis in appearance).
- Palpable pulses in extremity.
- Palpable cord if superficial enough.

DEFINITION

- Labs: PT and PTT should be normal. D-Dimer may be elevated but is nonspecific.
- Ultrasound of affected lower extremity: Looking at venous system and its compressibility is the most readily available imaging study, while venography is the gold standard.

TREATMENT

- Anticoagulation with heparin if DVT or PE present: 80 U/kg IV bolus followed by 18 U/kg/hr infusion. LMWH can be used for DVT without PE.
- Inferior vena caval filter (Greenfield) for patients with malignancy, already on oral anticoagulation, or who have a contraindication to anticoagulation (frequent falls in elderly).
- Consider thrombolytics for massive iliofemoral thrombosis.

Gastrointestinal Emergencies

Abdominal Pain

VISCERAL PAIN

- Vague, dull, and poorly localized pain.
- Midline location due to bilateral innervation of organs based on their embryological origin.
- Associated with stretching, inflammation, or ischemia, involving bowel walls or organ capsules.

PARIETAL PAIN

- Sharp, well-localized pain; peritonitis associated with rebound and involuntary guarding.
- Pain location correlates with associated dermatomes:
  - Occurs commonly with inflammation, frank pus, blood, or bile in or adjacent to the peritoneum.

Peritonitis is associated with rebound tenderness and involuntary guarding.

REFERRED PAIN

- Pain stimuli generated at an afflicted location are perceived as originating from a site in which there is no current pathology.
- These sites are usually related by a common embryological origin.
- The pain can sometimes be perceived in both locations.

CAUSES OF ABDOMINAL PAIN (BY QUADRANTS)

Right Upper Quadrant

Gastric ulcer
Peptic ulcer
Biliary disease
Hepatitis
Pancreatitis
Retrocecal appendicitis
Renal stone
Pyelonephritis
MI
Pulmonary embolus
Pneumonia

Left Upper Quadrant

Gastric ulcer
Gastritis
Pancreatitis
Splenic injury
Renal stone
Pyelonephritis
MI
Pulmonary embolus
Pneumonia

Right Lower Quadrant

Appendicitis
Ovarian cyst
Mittelschmerz
Pregnancy (ectopic or normal)
Tubo-ovarian abscess
Pelvic inflammatory disease
Ovarian torsion
Cystitis
Prostatitis
Ureteral stone

Left Lower Quadrant

Diverticulitis
Ovarian cyst
Mittelschmerz
Pregnancy (ectopic or normal)
Tubo-ovarian abscess
Pelvic inflammatory disease
Ovarian torsion
Cystitis
Prostatitis
Ureteral stone

V is for Vague.
Example: Early appendicitis; initially dull, periumbilical pain.

P is for Pinpoint.
Example: Late appendicitis; local inflammation leads to tenderness in the RLQ.

Example: Ureteral obstruction can produce pain in the ipsilateral testicle.

A 26-year-old woman complains of severe LLQ pain, vaginal bleeding, weakness, and light-headedness. Last menstrual period was 6 weeks ago. *Think: Ectopic pregnancy.*

HIGH-YIELD FACTS

Gastrointestinal Emergencies

Testicular torsion   Testicular torsion
Epididymitis   Epididymitis
Diverticulitis   Diverticulitis
Abdominal aortic aneurysm   Abdominal aortic aneurysm

Note: *All premenopausal women with abdominal pain must have a pregnancy test, even if they say they are not sexually active.*

OTHER CAUSES OF ABDOMINAL PAIN

Abdominal Wall
- Hernia
- Rectus sheath hematoma

Metabolic
- Diabetic ketoacidosis
- Acute intermittent porphyria
- Hypercalcemia

Infectious
- Herpes zoster
- Mononucleosis
- Human immunodeficiency virus (HIV)

Drugs/Toxins
- Heavy metal poisoning
- Black widow spider envenomation

Other
- Sickle cell anemia
- Mesenteric ischemia

ABDOMINAL PAIN IN THE ELDERLY

Elderly patients who present with abdominal pain must be treated with particular caution. Common problems include:
- Difficulty communicating.
- Comorbid disease.
- Inability to tolerate intravascular volume loss.
- Unusual presentation of common disease.
- May not mount a WBC count or a fever.
- Complaint often incommensurate with severity of disease.

Note: Up to 10% of elderly patients with an MI will present with abdominal pain.

> **ESOPHAGUS**

Varices

DEFINITION

Dilated submucosal veins.

A 28-year-old woman presents with diffuse abdominal pain, nausea, and confusion. She is not pregnant. She currently takes a stained-glass class. *Think: Lead poisoning.*

In elderly patients with abdominal pain, always consider vascular causes, including:
- Abdominal aortic aneurysm
- Mesenteric ischemia
- MI

Gastrointestinal Emergencies

- Found in 25 to 40% of patients with cirrhosis.
- Usually develop due to portal hypertension.
- Thirty percent of patients with varices develop upper gastrointestinal (GI) bleeds.

CLINICAL FINDINGS

- Asymptomatic until rupture.
- Bleeding is usually massive.
- Present with spontaneous emesis of either bright red blood or coffee ground material.

TREATMENT

- Volume replacement with normal saline (NS) and packed red blood cells.
- Nasogastric (NG) suction.
- Intravenous (IV) vasopressin or octreotide to control bleeding; also IV proton pump inhibitor.
- Emergent endoscopy to localize bleeding for possible sclerotherapy or rubber band ligation of varices.
- Consider tamponade with Sengstaken–Blakemore tube for persistent bleeding.
- GI consult.
- Hospital admission for all unstable cases.

Esophageal Foreign Body (FB) Ingestion

SITES OF IMPACTION

- The most common site in children is at the cricopharyngeus muscle (C5).
- The most common site in adults is at the lower esophageal sphincter (LES) (T10).

EPIDEMIOLOGY

- Eighty percent of obstructions occur in children and are due to coins, marbles, buttons, etc.
- Twenty percent of obstructions occur in adults and are due to meat impaction.

SIGNS AND SYMPTOMS

- Dysphagia
- Gagging
- Throat pain
- FB sensation
- Vomiting
- Anorexia
- Anxiety

DIAGNOSIS

- CXR and soft-tissue films of the neck to look for:
  - The flat surface of a coin or other such FB will be seen when it is lodged in the esophagus (see Figure 9-1).
  - The edge will be seen when located in the trachea.

Higher morbidity and mortality rate than any other source of upper GI bleed.

Many patients with varices have coagulopathy due to underlying cirrhosis.

Beta-blocker therapy may reduce rebleeds by decreasing portal hypertension.

Adults with esophageal meat impactions almost always have underlying pathology such as carcinoma or strictures.

Endoscopy offers the advantage of being able to visualize and *remove* FB.

HIGH-YIELD FACTS

Gastrointestinal Emergencies
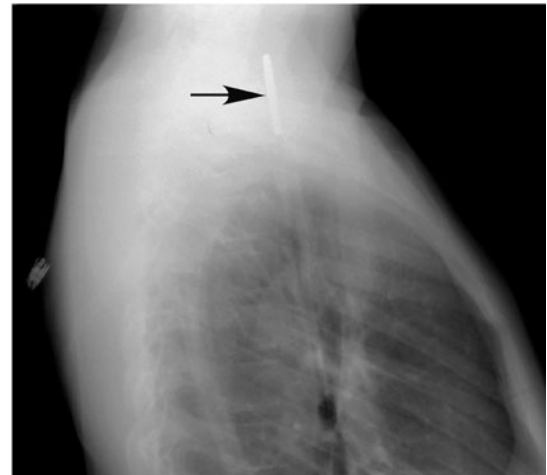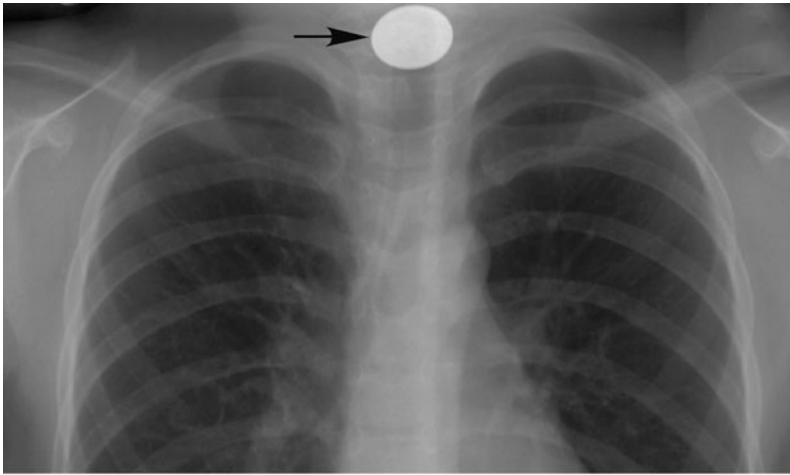
FIGURE 9-1. AP and lateral views demonstrating a coin in the esophagus.

A coin in the trachea would present in the opposite manner—the coin would be seen on edge on the AP view, and flat on the lateral view.

- If radiographs do not demonstrate FB, then consider esophagogram with contrast or endoscopy.

TREATMENT

- Eighty percent of ingested FBs pass spontaneously; no treatment is required in these cases, except observatin of stool for 3 to 5 days.
- If FB is in the upper third of the esophagus (cervical esophagus, top 5 cm), it can be removed with a Magill forceps and a laryngoscope.
- For meat impactions in the distal (last 3 cm) esophagus:
  - IV glucagon or sublingual nitroglycerin can be used to relax smooth muscle and decrease LES tone.
  - Carbonated beverages and other gas-forming agents may be useful to push the meat impaction down into the stomach by raising the intraluminal pressure.
- Endoscopic removal is required for sharp objects and objects larger than 2 cm wide or 5 cm long.
- Removal of these objects before they pass the pylorus decreases chance of perforation.
- Approximately 1% of impacted FBs cannot be removed by direct visualization or do not pass into the stomach and must be removed surgically.

> Most alkaline batteries will pass into the stomach and can be managed expectantly. However, 10% of button batteries will lodge in the esophagus, and these must be removed because they are highly corrosive (alkali causes liquefaction necrosis).

Gastroesophageal Reflux Disease (GERD)

DEFINITION

Reflux of acidic gastric contents into the esophagus.

CAUSES

- Relaxed or incompetent LES
- Hiatal hernia
- Decreased esophageal motility
- Delayed gastric emptying
- Diabetes mellitus

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- Gastroparesis
- Gastric outlet obstruction
- Anticholinergic use
- Fatty foods

CAUSES OF LOWERED LES TONE

- Coffee
- Cigarettes
- Alcohol
- Chocolate
- Peppermint
- Anticholinergics
- Progesterone
- Estrogen
- Nitrates
- Calcium channel blockers

SIGNS AND SYMPTOMS

- Substernal burning pain
- Dysphagia
- Hypersalivation (water brash)
- Cough

DIAGNOSIS

Barium swallow, esophagoscopy, mucosal biopsy.

TREATMENT

- Elevate head of bed.
- Discontinue foods that decrease LES tone.
- Oral antacids.
- H_2 blocker or proton pump inhibitor.
- Patients with hiatal hernia may be candidates for Nissen fundoplication (the stomach is wrapped around the distal esophagus to create a "new sphincter").

COMPLICATIONS OF GERD

- **Esophagitis:** Esophageal damage, bleeding, and friability due to prolonged exposure to gastric contents.
- **Peptic stricture:** Occurs in about 10% of patients with GERD.
- **Barrett's esophagus:** Transformation of normal squamous epithelium to columnar epithelium, sometimes accompanied by an ulcer or stricture.
- **Esophageal cancer:** Upper two thirds squamous, lower one third adenocarcinoma.

A majority of patients with asthma have associated GERD.

Barrett's esophagus carries a 2 to 5% risk of development of esophageal adenocarcinoma, which carries a < 5% chance of 5-year survival.

► STOMACH

Boerhaave's Syndrome

- Spontaneous rupture of the esophagus that typically occurs after forceful emesis.
- Most lethal perforation of the GI tract, with mortality ~35%.

Boerhaave syndrome is a transmural perforation of the esophagus to be distinguished from Mallory–Weiss syndrome, a nontransmural esophageal tear also associated with vomiting.

RISK FACTORS

Alcohol ingestion.

CLINICAL FINDINGS

- The Mackler triad: Vomiting, lower thoracic pain, and subcutaneous emphysema.
- Pneumomediastinum.
- Crackling sound on chest auscultation ("Hamman's crunch").

DIAGNOSIS

- Chest x-ray (CXR) in 90% of patients shows abnormal findings after perforation.
- The most common findings on CXR are pleural effusion (usually left), pneumothorax, hydropneumothorax, pneumomediastinum, subcutaneous emphysema, or mediastinal widening.
- Water-soluble contrast esophagram helps confirm the diagnosis.

TREATMENT

- IV volume resuscitation.
- Administration of broad-spectrum antibiotics.

Mallory–Weiss Tear

DEFINITION

A **partial-thickness** tear at the gastroesophageal junction associated with hematemesis, usually self-limited.

RISK FACTORS

- Alcoholism
- Hiatal hernia
- Gastritis

CLINICAL FINDINGS

- Prior history of vomiting, retching, or straining.
- Endoscopy establishes diagnosis.

TREATMENT

Usually self-limited.

Acute Gastritis

DEFINITION

Inflammation of the stomach.

ETIOLOGIES

- Stress gastritis is due to severe medical or surgical illness including trauma, burns, hypotension, sepsis, central nervous system (CNS) injury (Cushing ulcer), mechanical respiration, and multiorgan failure.
- Corrosive gastritis is most commonly seen with alcohol.
- Often associated with *Helicobacter pylori* infection.

Things that cause gastritis:
SPIT BANDS
Shock states
Pancreatic juice
Infection with *Helicobacter pylori*
Tobacco

Bile
Alcohol
Nonsteroidal anti-inflammatory drugs (NSAIDs)
Drug-induced
Steroids, stress

CLINICAL FINDINGS

- Most asymptomatic unless ulcers or other complications develop.
- Symptomatic: Abdominal pain, nausea/vomiting, GI bleed.
- Typically diagnosed at endoscopy for complaints of dyspepsia or upper GI bleed.

TREATMENT

- Avoid alcohol, cigarettes, caffeine, and citrus and spicy foods for 6 weeks.
- Prophylaxis with H_2 blocker.

Upper GI Bleed

DEFINITION

Bleeding that is proximal to ligament of Treitz.

ETIOLOGY (IN ORDER OF DECREASING FREQUENCY)

- Peptic ulcer (accounts for > 50%)
- Gastric erosions
- Varices
- Mallory–Weiss tear
- Esophagitis
- Duodenitis

CLINICAL FINDINGS

- Most common presentation is hematemesis (bright red blood or "coffee grounds") or melena (dark tarry stool) with or without abdominal pain.
- Hematochezia (bright red bloody stool) usually indicates lower GI bleed, but may also be present in massive upper GI bleeds.
- Check for hypotension, tachycardia, weakness, pallor, syncope, and diaphoresis.
- Ear, nose, and throat exam should be done to rule out nosebleeds, which can mimic upper GI bleed (swallowed blood).

DIAGNOSIS

- Routine labs: Complete blood count (CBC), prothrombin time, partial thromboplastin time, type and crossmatch 4 to 6 units, electrolytes, liver function tests (LFTs).
- Abdominal radiograph (AXR): Usefulness very limited but may rule out free air.

TREATMENT

- Rapid assessment and management with ABCs (airway, breathing, and circulation) supporting airway, IV (NS or lactated Ringer's solution), O_2, and monitor.
- Blood products for continued active bleeding or failure to improve vitals. Consider use of IV vasopressin or octreotide.
- NG lavage looking for coffee grounds or fresh blood.
- GI consult for identification of bleeding sites with endoscopy.
- Surgical consultation and intervention is indicated if patient does not respond to medical or endoscopic treatment.
- Admit all unstable patients.

The most common cause of gastritis is chronic use of NSAIDs.

NSAID use, steroids, or dyspepsia: *Think: Peptic ulcer disease (PUD).*

Chronic liver disease: *Think: Esophageal varices or portal hypertension.*

Heavy alcohol ingestion or retching: *Think: Mallory–Weiss tear.*

Initial hematocrit is a poor indicator of the severity of acute bleeding because it takes 24 to 72 hours to equilibrate.

Peptic Ulcer Disease (PUD)

DEFINITION

Disruption of the mucosal defensive factors by acid and pepsin, which causes ulceration of the mucosa beyond the muscularis.

RISK FACTORS

- Infection with *H. pylori.*
- Cigarette use.
- Ethanol use.
- NSAIDs (prostaglandin depletion).
- Others: Steroids, hepatic cirrhosis, renal failure, familial predisposition.

CLINICAL FINDINGS

- Classically, the pain is described as burning, gnawing, dull, or hunger-like.
- Gastric ulcer (GU) pain begins shortly after eating.
- Duodenal ulcer (DU) pain occurs 2 to 3 hours after meal and is relieved by food or antacids.
- Ulcers are most commonly located along the lesser curvature of the stomach or the first portion of duodenum.
- Simple bleeding (most common cause of upper GI bleed):
  - Most stop spontaneously.
  - Posterior ulcer erodes into the gastroduodenal artery.

DIAGNOSIS

- Most peptic ulcer disease is not definitively diagnosed in the emergency department, but rather treated empirically.
- Endoscopy is 95% accurate for diagnosis.

TREATMENT

- Treatment is primarily outpatient unless complications occur.
- Advise patient to avoid substances that exacerbate ulcers.
- Eradicate *H. pylori* disease.
- Pain relief with antacids given 1 hour before and 3 hours after meal (poor compliance due to frequency of therapy).
- H_2 receptor antagonists (cimetidine, ranitidine, famotidine, or nizatidine) and proton pump inhibitors (e.g., omeprazole, lansoprazole) are mainstay of therapy in noninfected individuals.
- Patients who demonstrate any complication should be stabilized and admitted.

COMPLICATIONS OF PUD

Perforation

Signs and Symptoms
- Sudden onset of generalized abdominal pain associated with a rigid abdomen often radiating to back.
- Vomiting involved in 50%.

Crossmatched blood takes ~45 to 60 minutes. Use O negative if type-specific blood is not ready.

H. pylori is associated with 80% of GUs and 95% of DUs.

Pain is visceral in nature and is therefore vague and midline.

Adverse effects of cimetidine:
- Increases levels of drugs cleared via P450 system (e.g., warfarin, phenytoin, diazepam, propranolol, lidocaine, theophylline, tricyclic antidepressants)
- CNS dysfunction in the elderly
- Thrombocytopenia
- Painful gynecomastia

Diagnosis
- Upright CXR to look for free air: Useful for 70% of anterior perforations (most common type). Does not pick up posterior perforations because the posterior duodenum is retroperitoneal.
- Posterior perforations may lead to pancreatitis.

Treatment
IV fluids, NG drainage, antibiotics, immediate surgery.

Gastric Outlet Obstruction

Pathophysiology
Healing ulcer may scar and block the antral or pyloric outlet.

Signs and Symptoms
- History of vomiting undigested food shortly after eating.
- Succussion splash: Splashing sound made when abdomen is gently rocked.
- Early satiety.
- Weight loss.

Diagnosis
- Characteristic electrolyte abnormalities (hypokalemia, hypochloremia, and metabolic alkalosis).
- AXR will show dilated stomach with large air–fluid level.

Treatment
- NG suctioning
- Correction of electrolyte abnormalities
- Hospital admission

Inflammatory Bowel Disease (IBD)

Definition
A chronic, inflammatory disease affecting GI tract. Two major types are Crohn's disease (CD) and ulcerative colitis (UC).

Epidemiology
- More common in people of Caucasian and Jewish background.
- Peak incidence in ages 15 to 35.
- Occurs with familial clustering.
- Incidence: UC = 2 to 10/100,000; CD = 1 to 6/100,000.
- UC more common in men.
- CD more common in women.
- Associated risk of colon cancer is 10 to 30 times for UC and 3 times for CD.

CLINICAL PRESENTATION AND DIAGNOSIS

See Tables 9-1 and 9-2.

Treatment

Supportive Care

Antidiarrheals
- Decrease frequency of stool.
- Loperamide and diphenoxylate are used for patients with fatty acid–induced diarrhea.

Severe sudden abdominal pain with history of ulcer: *Think: Perforation* (common board case). Patients usually lie still and avoid movement.

Crohn's: Lower incidence, lower risk of cancer, more common in women.

Early institution of NG suction, IV fluids, and steroids to reduce inflammation is suspected bowel obstruction.

TABLE 9-1. Clinical Presentation and Diagnosis of Ulcerative Colitis (UC) and Crohn's Disease (CD)

	ULCERATIVE COLITIS	CROHN'S DISEASE
Signs and symptoms	▪ Bloody diarrhea ▪ Rectal pain	▪ Crampy abdominal pain, typically in RLQ ▪ Fatigue, malaise (due to chronic anemia)
Location	▪ Limited to rectum and colon	▪ Can affect any part of GI tract
Pathology	▪ Inflammation of mucosa only (exudates of pus, blood, and mucus from the "crypt abscess")	▪ Inflammation involves all bowel wall layers, which may lead to fistulas and abscesses ▪ Rectal sparing in 50%
Colonoscopy findings	▪ Continuous lesions ▪ Lead pipe colon apperance due to chronic scarring and subsequent retraction and loss of haustra	▪ Skip lesions ▪ Aphthous ulcers ▪ Cobblestone apperance from submucosal thickening interspersed with mucosal alteration
Complications	▪ Perforation ▪ Stricture ▪ Megacolon	▪ Abscesses ▪ Fistulas ▪ Obstruction ▪ Perianal lesions

- Cholestyramine is used for patients without fatty acid–induced diarrhea.
- Contraindicated in severe colitis due to risk of toxic megacolon.

Anticholinergics
- Reduce abdominal cramping, pain, and urgency.
- Opium–belladonna combination works well to control diarrhea and pain.

Specific Therapy

Sulfasalazine
- Consists of 5-acetylsalicylic acid (ASA) (active component) and sulfapyridine (toxic effects are due to this moiety).
- Side effects include GI distress in one third of patients (give enteric-coated preparation), decreased folic acid absorption, and male infertility (reversible).
- Drug appears safe in children and pregnant women.

Corticosteroids
- Early phase of action blocks vascular permeability, vasodilation, and infiltration of neutrophils.
- Late phase of action blocks vascular proliferation, fibroblast activation, and collagen deposition.
- May be given as enemas (decreases systemic absorption).

Antibiotics (used for CD)
- Three-week courses of metronidazole and ciprofloxacin have been used to induce disease remission with some success.

> Sulfasalazine is also used to treat rheumatoid arthritis, but in this case, it is the sulfapyridine component that is the active one.

> Drugs with only the 5-ASA component are not effective in IBD.

Eye involvement	■ Uveitis	CD > UC
	■ Episcleritis	Uveitis, erythema nodosum, and colitic arthritis are commonly seen together.
Dermatologic	■ Erythema nodosum	CD > UC, especially in children
		Parallels disease course (gets better as IBD improves)
	■ Pyoderma gangrenosum	UC > CD
		May or may not follow disease course
	■ Aphthous ulcers	CD
Arthritis	■ Colitic arthritis	CD > UC
		Parallels disease course
	■ Ankylosing spondylitis	30 times more common in UC
		Unrelated to disease course
Hematologic	■ Anemia	
	■ Thromboembolism	
Hepatobiliary	■ Fatty liver	
	■ Hepatitis	
	■ Cholelithiasis	
	■ Primary sclerosing cholangitis	UC > CD
Renal	■ Secondary amyloidosis leading to renal failure	CD
		Unrelated to disease course

■ Mechanism of action is unknown because other antibiotics with a similar antimicrobial spectrum have not been shown to be effective.

Immunomodulators
■ Used in refractory cases, especially in CD.
■ Include azathioprine, 6-mercaptopurine, and methotrexate.
■ Resistant cases may also benefit from anti–tumor necrosis factor-alpha (TNF-α) antibodies, recombinant anti-TNF cytokines.

Mesenteric Ischemia

DEFINITION

■ Lack of perfusion to bowel
■ High-mortality disease

RISK FACTORS

■ Age > 50.
■ Valvular or atherosclerotic heart disease.
■ Arrhythmias (especially atrial fibrillation).
■ Congestive heart failure.

- Recent myocardial infarction (MI).
- Critically ill patients with sepsis or hypotension.
- Use of diuretics or vasoconstrictive drugs.
- Hypercoagulable states.

CLINICAL FINDINGS

- Severe acute midabdominal **pain out of proportion to findings** (i.e., patient complains of severe pain but is not very tender on exam).
- Sudden onset suggests arterial vascular occlusion by emboli, consistent with acute ischemia.
- Insidious onset suggests venous thrombosis or nonocclusive infarction (intestinal angina), consistent with chronic ischemia.
- As infarct develops, peritoneal signs develop suggestive of necrotic bowel.

DIAGNOSIS

- Laboratory analyses reveal metabolic acidosis, elevated lactate, and elevated phosphorus.
- AXR may reveal dilated loops of bowel, air–fluid level, irregular thickening of the bowel wall (thumbprinting), and gas in the bowel wall or portal system.
- Computed tomography (CT) scan may demonstrate air in the bowel wall, mesenteric portal vein gas, and bowel wall thickening.
- Angiography is the gold standard and should not be delayed.
- Diagnosis can frequently be delayed without high index of suspicion.

TREATMENT

- IV fluid to correct fluid and electrolyte abnormalities.
- Supplemental O_2.
- NG tube to decompress bowel.
- Antibiotics to cover gut flora.
- Selective vasodilator infusion (e.g., papaverine) during angiography.
- Surgery to remove emboli or necrotic bowel.

Hernias

DEFINITIONS

- Protrusion of a structure through an opening that is either congenital or acquired.
- Reducible hernia: Protruding contents can be pushed back to their original location.
- Incarcerated hernia: An irreducible hernia may be acute and painful or chronic and asymptomatic.
- Strangulated hernia: Incarcerated hernia with vascular compromise.

RISK FACTORS

- Obesity
- Chronic cough
- Pregnancy
- Constipation
- Straining on urination

A diagnosis of mesenteric ischemia requires a high index of suspicion.

Gas in the bowel wall is known as *pneumatosis intestinalis.*

Abdominal pain and metabolic acidosis. *Think: Mesenteric ischemia* until proven otherwise.

- Ascites
- Previous hernia repair

CLINICAL FINDINGS OF INGUINAL HERNIAS

Direct
- Protrudes through floor of Hesselbach's triangle.
- Frequency increases with age.
- Rarely incarcerates.

Indirect
- Protrudes lateral to the inferior epigastric vessels.
- Most commonly occurring hernia.
- Frequently incarcerates.
- History of palpable, soft mass that increases with straining (patient bears down and coughs while you pass digit in external canal).
- Bowel sounds may be heard over hernia if it contains bowel.

DIAGNOSIS
- Can be made from physical exam.
- Abdominal radiograph to look for air–fluid levels (obstruction) or free air under the diaphragm (perforation).

TREATMENT
- May attempt reduction of incarcerated hernia with outpatient referral for surgery. Advise patient to refrain from straining.
- A strangulated hernia requires immediate surgery. Do not attempt to reduce dead bowel into abdomen!

Intussusception

DEFINITION

The telescoping of one segment of bowel into another, the most common being the ileocecal segment.

RISK FACTORS

Fifty percent have recent viral infection.

CLINICAL FINDINGS
- Classic triad:
  - Colicky abdominal pain
  - Vomiting
  - Currant jelly stool (late finding)
- Elongated mass may be palpable in right upper quadrant (RUQ).

DIAGNOSIS

Diagnosis by air or barium enema—"coiled spring" appearance of bowel (Figure 9-2).

TREATMENT

Air or barium enema leads to reduction in 60 to 70%. Remaining cases require surgery.

Incarcerated hernias are the second most common cause of SBO (after adhesions).

Bowel obstruction can be the first presenting sign of a hernia.

Intussusception is the most common cause of bowel obstruction in children ages 2 months to 5 years.

FIGURE 9-2. Intussusception.

Note the paucity of bowel gas in film A. Air enema partially reduces it in film B and then completely reduced it in film C.

Small Bowel Obstruction (SBO)

ETIOLOGIES

- Adhesions (most common)
- Hernia (second most common)
- Neoplasms
- Intussusception
- Gallstones
- Bezoars
- IBD
- Abscess

CLINICAL FINDINGS

- Intermittent crampy abdominal pain.
- Vomiting.
- Abdominal distention.
- Absence of bowel movements or flatulence for several days.
- Hyperactive high-pitched bowel sounds **(borborygmi)** (become hypoactive and eventually absent as the obstruction progresses).

DIAGNOSIS

AXR may demonstrate stepladder appearance of air–fluid levels (see Figure 9-3), thickening of small bowel wall, or loss of markings (plicae circulares).

TREATMENT

- IV fluids, NG suction, and early surgical consult.
- May consider antibiotics if infection present.
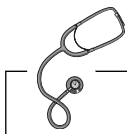
Inspect for scars and hernias and ask about past surgical history (risk for adhesions).
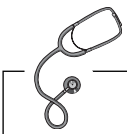
In proximal obstruction, bilious vomiting can occur early with minimal distention.
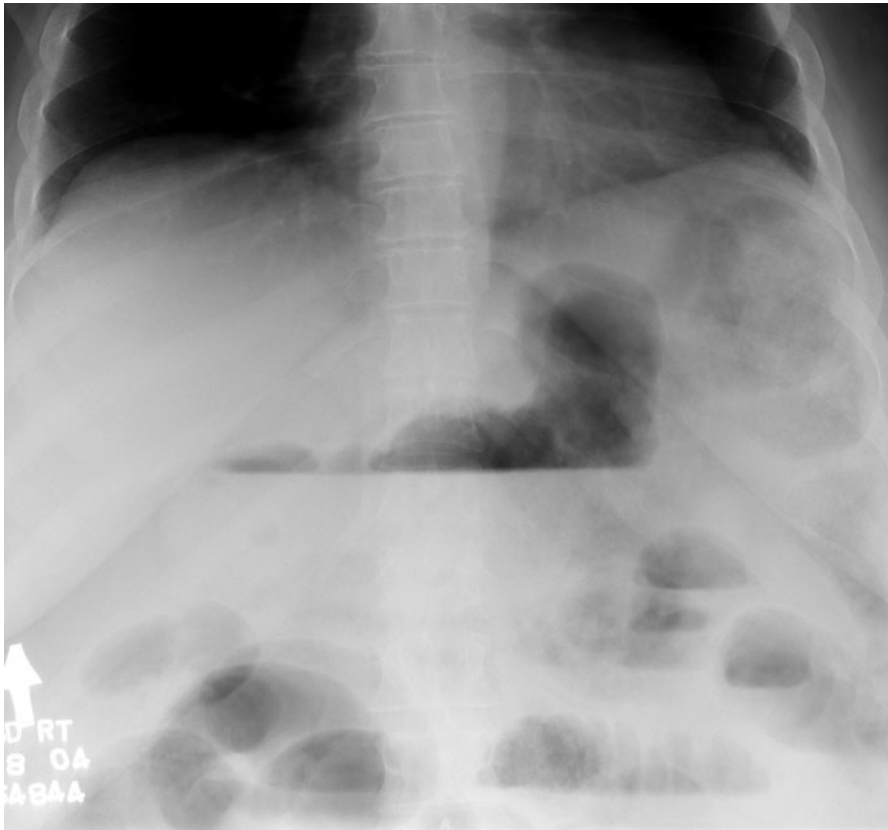
F I G U R E   9 - 3 .   SBO.

Radiograph of the abdomen demonstrates multiple distended small bowel loops with air–fluid levels throughout abdomen.

Hepatitis

DEFINITION

Inflammation of the liver secondary to a number of causes.

ETIOLOGIES

- Alcohol:
  - Most common precursor to cirrhosis.
  - May develop after several decades of alcohol abuse or within 1 year of heavy drinking.
- Autoimmune: Little is known at this time.
- Toxins: Acetaminophen, carbon tetrachloride, heavy metals, tetracyclines, valproic acid, isoniazid, amiodarone, phenytoin, halothane, methyldopa.
- Viruses:
  - Hepatitis A (HAV): Fecal–oral transmission via contaminated water or food, endemic areas; no carrier state; does not cause chronic liver disease.
  - Hepatitis B (HBV): Sexual and parenteral transmission; has a carrier state and causes chronic disease; effective vaccine available.

HBV and HCV are more contagious than human immunodeficiency virus (HIV). Always use universal precautions.

Concurrent HBV/HCV infection is common and exacerbates liver disease. Both carry increased risk of cirrhosis and hepatocellular carcinoma.

HCV is the most common blood-borne cause of viral hepatitis in the United States.

In alcoholic hepatitis, the SGOT is greater than SGPT by a factor of 2.

Admit hepatitis if:
- Encephalopathic
- Excessive bleeding
- INR > 3
- Intractable vomiting
- Immunosuppressed
- Due to alcohol
- Hypoglycemic
- Bilirubin > 25

- Hepatitis C (HCV): Sexual and parenteral transmission; has a carrier state; 85% go on to develop chronic liver disease; no vaccine available.
- Hepatitis D: Sexual and parenteral transmission; incomplete virus—requires coinfection with HBV.
- Hepatitis E (HEV): Similar to HAV but higher incidence of fulminant liver failure; no serologic marker.
- Cytomegalovirus.
- Herpes simplex virus.
- Parasites:
  - *Entamoeba histolytica* abscess presents with RUQ pain, fever, and diarrhea.
  - *Clonorchis sinensis* (liver fluke).

CLINICAL FINDINGS

- RUQ tenderness (due to distention of liver capsule).
- Alcoholic:
  - Can range from mild liver disease to acute liver failure.
  - May present with liver enlargement, weakness, anorexia, nausea, abdominal pain, and weight loss.
  - Dark urine, jaundice, and fever are frequent complaints.
  - Physical exam may reveal jaundice, pedal edema, gynecomastia, palmar erythema, and spider angiomata.
  - Complications: Ascites, portal hypertension, esophageal varices, spontaneous bacterial peritonitis (SBP), hepatic abscess, hepatorenal syndrome, hepatic encephalopathy.
- Viral:
  - Prodrome of anorexia, nausea, vomiting, malaise, and flulike symptoms.
  - History of travel to endemic area for HAV and HEV, IV drug users, homosexuals.
  - Serologic studies may be ordered in emergency department, but results are not immediately available.
- Parasites: History of travel to endemic area.

DIAGNOSIS

- Thrombocytopenia.
- Elevated bilirubin.
- Serum glutamate pyruvate transaminase (SGPT) > serum glutamic-oxaloacetic transaminase (SGOT) suggestive of viral hepatitis.
- PT usually normal.

TREATMENT

- Supportive care is the mainstay of therapy; treat complications.
- Alcohol:
  - Hospital admission for all but the mildest cases.
  - Correct electrolyte abnormalities.
  - Supplement thiamine and folate.
  - High-calorie/high-protein diet.
- HBV: Interferon-alpha, ribavirin.
- Acetaminophen poisoning: N-acetylcysteine (best if given within 24 hours of ingestion, but usually no hepatitis by then; can give up to 1 week after ingestion).

- Parasites:
  - Metronidazole/albendazole.
  - Occasionally needle aspiration and decompression or surgical decontamination.

Hepatic Encephalopathy

DEFINITION

A manifestation of hepatic failure, the final common pathway.

TREATMENT

- Correction of fluids and electrolyte abnormalities.
- Lactulose and neomycin to clear the gut of bacteria and nitrogen products.
- Liver transplant may be lifesaving.

See Table 9-3 for grading of hepatic encephalopathy.

Hepatorenal Syndrome

DEFINITION

Acquired renal failure in association with liver failure; cause unknown.

CLINICAL FINDINGS

- Hypotension
- Ascites:
  - Portal hypertension (high hydrostatic pressure)
  - Hypoalbuminemia (low oncotic pressure)

Reye's syndrome: Acute hepatic encephalopathy associated with ASA use in children.

TABLE 9-3. Grading of Hepatic Encephalopathy

GRADE	LEVEL OF CONSCIOUSNESS	PERSONALITY AND INTELLECT	NEUROLOGIC SIGNS
0	Normal	Normal	None
1	Day/night sleep reversal Restlessness	Forgetfulness Mild confusion Agitation Irritability	Tremor Apraxia Incoordination Impaired handwriting
2	Lethargy Slowed responses	Disorientation to time Loss of inhibition Inappropriate behavior	Asterixis Dysarthria Ataxia Hypoactive reflexes
3	Somnolence Confusion	Disorientation to place Aggressive behavior	Asterixis Muscular rigidity Babinski signs Hyperactive reflexes
4	Coma	None	Decerebration

DIAGNOSIS

- Azotemia, oliguria, hyponatremia, low urinary sodium.
- Sodium retention by kidneys from increase renin and angiotensin levels.
- Impaired liver clearance of aldosterone (all hormones).

TREATMENT

- Low-salt diet.
- Fluid restriction.
- Diuretics (spironolactone and furosemide or hydrochlorothiazide).
- Paracentesis: Therapeutic in massive ascites with respiratory compromise; low risk of bleeding, infection, or bowel perforation.

> Hepatorenal syndrome has a bad prognosis. Mortality is almost 100%.

Spontaneous Bacterial Peritonitis (SBP)

ETIOLOGY

- Bacterial breach of intestinal barrier to peritoneum
- *Escherichia coli*, pneumococci (anaerobes rare)

CLINICAL FINDINGS

- SBP should be suspected in cirrhotics with fever, abdominal pain, worsening ascites, and encephalopathy.
- Paracentesis:
  - Total white blood cell (WBC) count > 500 cells/mL
  - > 250 polymorphonuclear neutrophil/mL, very specific for SBP
  - Total protein > 1 g/dL
  - Glucose < 50 mg/dL
  - Cultures positive in 80 to 90%

> A high index of suspicion is necessary for diagnosis of SBP, as symptoms can be very mild.

TREATMENT

Hospital admission for IV antibiotics (third-generation cephalosporin).

Hepatic Abscess

ETIOLOGY

- Ascending cholangitis:
  - Most common cause of hepatic abscess.
  - Frequent organisms include *E. coli*, *Proteus vulgaris*, *Enterobacter aerogenes*, and anaerobes.
- Parasites (e.g., *E. histolytica*, *Echinococcus*): Travel history
- Idiopathic

> ▶ **GALLBLADDER**

Cholangitis

DEFINITION

Obstruction of the biliary tract and biliary stasis leading to bacterial overgrowth and infection.

> Cholangitis is a surgical emergency with high mortality.

ETIOLOGY

- Common duct stone is the most common cause.
- 1° sclerosing cholangitis.

CLINICAL FINDINGS

- **Charcot's triad:** RUQ pain, jaundice, fever/chills.
- **Reynolds' pentad:** Charcot's triad + shock and mental status change.
- Labs: Elevated WBC, bilirubin (direct > indirect), and alkaline phosphatase ultrasound (95% sensitivity) reveals ductal dilatation and gallstones.

TREATMENT

- ABCs.
- IV hydration.
- Correction of electrolytes.
- Antibiotics.
- Surgery consult.
- Endoscopic retrograde cholangiopancreatography (ERCP) may be effective in decompression (high morbidity).

Cholelithiasis and Cholecystitis

DEFINITIONS

- Cholelithiasis is a stone in the gallbladder.
- Choledocholithiasis is a stone in the common bile duct.
- Biliary colic: Transient gallstone obstruction of cystic duct causing intermittent RUQ pain lasting a few hours after a meal. No established infection.
- Acute cholecystitis is the obstruction of the cystic duct with pain lasting longer, fever, chills, nausea, and positive Murphy's sign.

DIAGNOSIS

- Labs: Alkaline phosphatase, bilirubin, LFTs, electrolytes, blood urea nitrogen (BUN), creatinine, amylase, lipase, CBC.
- Plain films may reveal radiopaque gallstones (10 to 15%).
- Ultrasound to look for: Presence of gallstones, thickened gallbladder wall, positive sonographic Murphy's sign, gallbladder distention, fluid collection:
  - Presence of gallstones, thickened gallbladder wall, and pericholecystic fluid has a positive predictive value of > 90% (Figure 9-4).
- Hepato-iminodiacetic acid *(the study of choice):* For this test, technetium-99m–labeled iminodiacetic acid is injected IV and is taken up by hepatocytes. In normals, the gallbladder is outlined within 1 hour.

TREATMENT

- Uncomplicated biliary colic may go home.
- Acute cholecystitis should be admitted to the surgical service.
- Poor surgical candidates may receive oral bile salts to promote dissolution of the stones: Second-line therapy.

Gas in biliary tree is strong supportive evidence of cholangitis.

Risk factors for cholelithiasis: **8 Fs**
Female
Fat
Fertile
Forty
Fibrosis, cystic
Familial
Fasting
F-Hgb (sickle cell disease)
Also:
Diabetes
Oral contraceptives

Murphy's sign: The arrest of inspiration while palpating the RUQ. This test is > 95% sensitive for acute cholecystitis, less sensitive in the elderly.
Sonographic Murphy's sign: The same symptom when the ultrasound probe is placed on the RUQ.

Gallstone composition:
- Cholesterol (70%): Radiolucent
- Pigment (20%): Radiodense
- Mixed (10%)

FIGURE 9-4. Sonogram demonstrating classic "headlight" appearance of cholelithiasis.

► **PANCREAS**

Acute Pancreatitis

DEFINITION

Inflammation and self-destruction of the pancreas by its digestive enzymes.

RISK FACTORS

- Gallstones and alcohol account for 85% of all cases.
- Pancreatic tumor (obstructing common duct).
- Hyperlipidemia.
- Hypercalcemia.
- Trauma.
- Iatrogenic (ERCP).
- Ischemia.
- Drugs (thiazide, diuretics, steroids).
- Familial.
- Viral (coxsackievirus, mumps).

CLINICAL FINDINGS

- Abrupt onset of deep epigastric pain with radiation to the back.
- Positional preference—leaning forward.
- Nausea, vomiting, anorexia, fever, tachycardia, and abdominal distention with diminished bowel sounds.
- Jaundice with obstructive etiology.

DIAGNOSIS

- Leukocytosis, elevated amylase and lipase (lipase more specific).

A 45-year-old obese woman complains of fever, RUQ pain, and nausea that is worse when she eats. *Think: Cholecystitis.*

*Hyper*calcemia can cause pancreatitis, and pancreatitis can cause *hypo*calcemia.

A sentinel loop is distention and/or air–fluid levels near a site of abdominal distention. In pancreatitis, it is secondary to pancreatitis-associated ileus.

- AXR: Sentinel loop, colon cutoff (distended colon to midtransverse colon with no air distally).
- Ultrasound: Good for pseudocyst, abscess, and gallstones.
- CT preferred diagnostic test (Figure 9-5).

PROGNOSIS

Ranson's criteria (see Table 9-4): Mortality rates correlate with the number of criteria present. Presence of more than three criteria equals a 1% mortality rate, while the presence of six or more criteria approaches a 100% mortality rate:
- At presentation:
  - Age > 55
  - WBC > 16,000
  - Glucose > 200
  - Lactic dehydrogenase > 350
  - SGOT > 250
- During initial 48 hours:
  - Hematocrit decrease > 10 points
  - BUN increase > 5
  - Serum Ca^{2+} < 8
  - Arterial PO_2 < 60
  - Base deficit > 4
  - Fluid sequestration > 6 L

TREATMENT

- Fluid resuscitation.
- Electrolyte correction.

A 50-year-old male alcoholic presents with midepigastric pain radiating to the back. He is leaning forward on his stretcher and vomiting. *Think: Pancreatitis.*

A 66-year-old female with hypertension and seizures for which she is on furosemide and valproic acid presents with abdominal pain, back pain, and fever. Her nonfasting glucose is noted to be 300. *Think: Pancreatitis.*
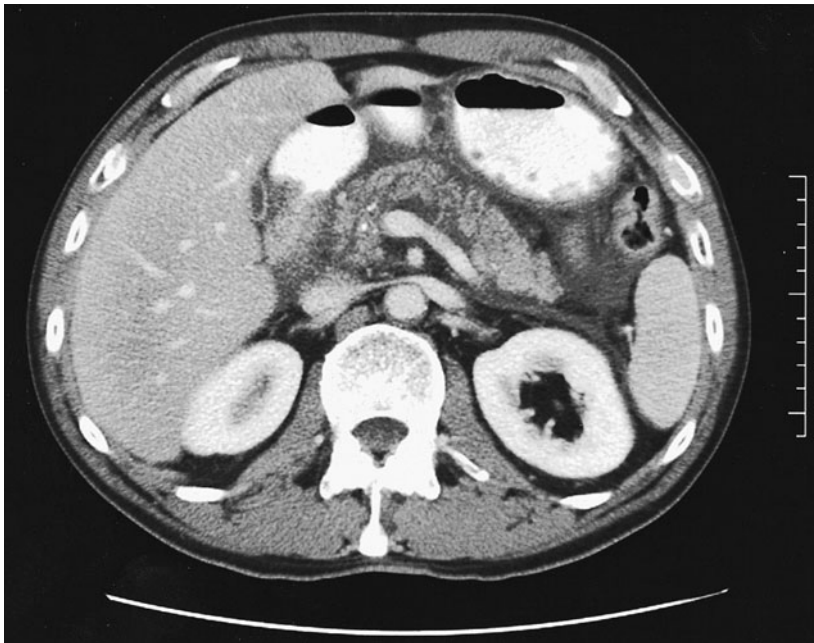
F I G U R E   9 - 5 .   Abdominal CT demonstrating stranding in the peripancreatic region, consistent with acute pancreatitis.

TABLE 9-4. **Mortality Rate Based on Number of Ranson's Criteria**

NUMBER OF CRITERIA	MORTALITY RATE
0–2	< 5%
3–4	15–20%
5–6	30–40%
> 6	Almost 100%

- Prevention of vomiting with antiemetics.
- Analgesia.
- Nothing by mouth (NPO) (pancreatic rest).
- NG suction as needed.

Pancreatic Pseudocyst

DEFINITION

- Encapsulated fluid collection with high enzyme content in a pseudocyst protruding from the pancreatic parenchyma.
- Most common complication of pancreatitis (2 to 10%).

CLINICAL FINDINGS

- Symptoms of pancreatitis.
- CT and ultrasonography (US) both have a sensitivity of 90%.

Suspect a pancreatic pseudocyst when patients with pancreatitis fail to resolve.

TREATMENT

- Surgical creation of fistula between cyst and stomach allowing for continuous decompression is most effective. Cyst eventually resolves without further intervention.
- Drain in 6 weeks when walls mature to reduce secondary infection, hemorrhage, or rupture.

Pancreatic Abscess

DEFINITION

Extensive necrosis of fat and mesentery with inflamed pancreas.

ETIOLOGY

Most commonly enteric organisms (50% polymicrobial).

CLINICAL FINDINGS

- Presents 1 to 4 weeks after acute pancreatitis.
- Fever, abdominal pain, tenderness, distention, paralytic ileus, and leukocytosis.
- CT is diagnostic study of choice.

TREATMENT

- Antibiotics
- Surgical debridement

Appendicitis

DEFINITION

Inflammation of the appendix.

PATHOPHYSIOLOGY

- The inciting event is obstruction of the lumen of the appendix.
- This leads to an increase in intraluminal pressure with vascular compromise of the wall of the appendix.
- The environment is now ripe for bacterial invasion.

ETIOLOGY

- Fecalith (most common cause)
- Lymphoid hyperplasia
- Worms
- Granulomatous disease
- Inspissated barium
- Tumors
- Adhesions
- Dietary matter such as seeds

CLINICAL FINDINGS

- Usually begins as vague periumbilical pain, then migrates to the RLQ where it becomes more intense and localized (McBurney's point).
- Retrocecal appendicitis can present as right flank pain.
- Anorexia.
- Nausea, vomiting.
- Low-grade fever.
- RLQ pain with rebound tenderness and guarding.
- **Rovsing's sign:** Pain in RLQ when palpation pressure is exerted in left lower quadrant (LLQ).
- **Iliopsoas sign:** Pelvic pain upon flexion of the thigh while the patient is supine.
- **Obturator sign:** Pelvic pain on internal and external rotation of the thigh with the knee flexed.

DIAGNOSIS

- Labs: Leukocytosis, hematuria, pyuria.
- If the diagnosis is clear-cut, no imaging studies are necessary.
- AXR may demonstrate fecalith (5% of time) or loss of psoas shadow.
- US will show noncompressible appendix.
- CT scan with contrast may demonstrate periappendiceal streaking. CT is 90 to 95% sensitive for appendicitis.

TREATMENT

- Prompt appendectomy
- NPO

Appendicitis is the most common surgical emergency.

Appendicitis in late pregnancy presents with RUQ pain due to displacement of appendix by gravid uterus.

Appendicitis:
Do not delay the diagnosis. Mortality of perforation is about 3%.

Conditions that mimic appendicitis: **CODE APPY CD**
CD
Ovarian cyst
Diverticulitis
Ectopic pregnancy

Adenitis of the mesentery
Pelvic inflammatory disease
Pyelonephritis
Yersinia gastroenteritis

- IV hydration
- Perioperative antibiotics

COMPLICATIONS

- Perforation
- Appendiceal abscess

Large Bowel Obstruction

ETIOLOGIES

- Tumor (most common)
- Diverticular disease
- Volvulus (sigmoid and cecal)
- Fecal impaction (especially elderly and mentally retarded)

CLINICAL FINDINGS

- Intermittent crampy abdominal pain, vomiting, abdominal distention.
- Absence of bowel movements or flatulence for several days.

DIAGNOSIS

AXR may demonstrate stepladder appearance of air–fluid levels, thickening of bowel wall, or loss of colonic markings (haustra).

TREATMENT

- IV fluids, NG suction, and early surgical consult.
- May consider antibiotics if infection present.
- Sigmoidoscopy may be done to decompress bowel.

Ogilvie's Syndrome

DEFINITION

Colonic pseudo-obstruction due to marked cecal dilatation.

RISK FACTORS

- Use/abuse of opiates, tricyclic antidepressants, anticholinergics.
- Prolonged bed rest.

DIAGNOSIS

AXR reveals cecal dilatation. A cecum diameter > 12 cm is a risk for perforation.

TREATMENT

- Decompression with enemas.
- If unsuccessful, colonoscopic decompression.

Due to its small radius, the cecum is normally the site of highest pressure in the GI tract.

Diverticular Disease

DEFINITIONS

- Diverticula are saclike herniations of colonic mucosa (most common at sigmoid) occurring at weak points in the bowel wall (insertions of arteries) with increased luminal pressures.
- Diverticulosis is the presence of diverticula, with massive painless lower GI bleeding.
- Diverticulitis (diverticula + inflammation) is the most common complication of diverticular disease. Fecal material lodges in diverticula, leading to inflammation and ischemia and mucosa erosion.

EPIDEMIOLOGY

- Prevalent in 35 to 50% of general population.
- Increased in the elderly and industrialized nations.

ETIOLOGIES

- Low-fiber diet
- Chronic constipation
- Family history

CLINICAL FINDINGS

- Diverticulosis:
  - Rectal bleeding
  - Anemia
  - Hematochezia
- Diverticulitis:
  - Constant severe LLQ pain with guarding
  - Abdominal distention
  - Fever
  - Diarrhea
  - Anorexia
  - Nausea

DIAGNOSIS

- AXR: Ileus, air–fluid levels, free air if perforation.
- CT: Study of choice.
- Colonoscopy and barium enema are relatively contraindicated in acute diverticulitis due to risk of perforation.

TREATMENT

- ABCs.
- Treat any hemodynamic compromise associated with massive GI bleeding.
- Diverticular disease: High-fiber diet and stool softeners to decrease luminal pressure and prevent constipation.
- Diverticulitis: IV fluids, NPO, NG suction (for ileus), and broad-spectrum antibiotics. Admit with surgical consult if severe.
- Diverticulosis: Address the bleeding.

COMPLICATIONS

Abscesses, obstruction, fistula, stricture, and perforation.

Diverticulosis is the most common cause of painless lower GI bleeding in older patients.

Diverticulitis is a common cause of large bowel obstruction.

A 70-year-old male presents with LLQ pain, diarrhea, fever, and guaiac-positive stool. AXR shows an ileus. *Think: Diverticulitis.*

Barium enema and colonoscopy should be avoided in acute cases of diverticulitis due to risk of perforation.

Lower GI Bleed

DEFINITION

Bleeding distal to the ligament of Treitz (small intestine or colon).

ETIOLOGIES

- Diverticulosis (70%)
- Angiodysplasia
- Colon cancer
- Hemorrhoids
- Trauma
- IBD
- Ischemic colitis
- Inappropriate anticoagulation
- Irradiation injury

CLINICAL FINDINGS

- Hematochezia
- Abdominal pain
- Weakness
- Anorexia
- Melena
- Syncope
- Shortness of breath

DIAGNOSIS

- NG lavage to rule out upper source (if blood is not seen and bile is aspirated, an upper source is unlikely).
- CBC (note acute blood loss will not be reflected in hematocrit).
- Colonoscopy to localize and possibly limit bleeding.
- If colonoscopy fails to reveal source, consider angiography or nuclear bleeding scan.

TREATMENT

- ABCs.
- Treat any hemodynamic compromise associated with massive GI bleeding similar to upper GI bleeding (stabilize first).
- Anoscopy and sigmoidoscopy for evidence of anorectal disease.
- Consider early GI/surgery consultation for large bleeds.
- Surgery if unstable or refractory to medical therapy.

> **RECTUM/ANUS**

Anal Fissure

DEFINITION

Linear tear of the anal squamous epithelium.

ETIOLOGIES

- Most benign fissures occur in posterior or anterior line.
- Fissures in other location or multiple sites are associated with CD, infection, and malignancy.

Angiodysplasia is the most common cause of lower GI bleeding in younger patients.

Upper GI bleed is the most common cause of apparent lower GI bleed.

Bright red blood that drips into the toilet or streaks stool suggests anorectal source.

Clinical Findings

- Perianal pain during or after defecation with blood-streaked toilet paper (subsides between bowel movements).
- Diagnosis made by visual inspection.

Treatment

Sitz baths, stool softener, high-fiber diet, hygiene, and analgesics.

Hemorrhoids

Definition

Dilated veins of the hemorrhoidal plexus (see Figure 9-6):
- Internal—arise above the dentate line and usually insensitive.
- External—below the dentate line, well innervated, painful!

Clinical Findings

External hemorrhoids present with painful thrombosis.

Treatment

If pain is severe, then excision of clot under local anesthesia followed by sitz baths and analgesics. Otherwise, manage expectantly with hydrocortisone cream, local anesthetic ointment, and sitz baths.

Perirectal Abscess

Definition

An abscess in any of the potential spaces near the anus or rectum (perianal, ischiorectal, submucosal, supralevator, and intersphincteric); begins with infection of the anal gland as it drains into the anal canal.

Clinical Findings

Extreme pain and mass on rectal exam.
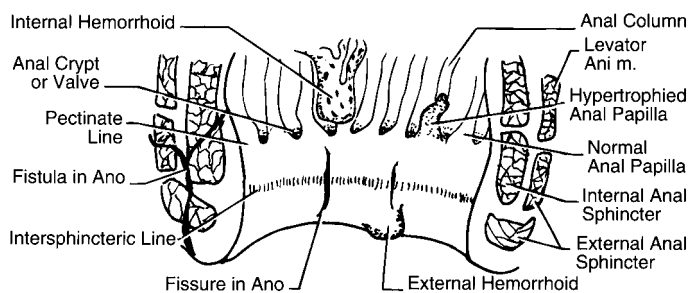
F I G U R E 9 - 6 . **Anatomy of internal and external hemorrhoids.**

(Reproduced, with permission, from DeGowin RL, Brown DD. *DeGowin's Diagnostic Examination*, 7th ed. New York: McGraw-Hill, 2000: 592.)

As with all abscesses, the treatment is drainage, and routine use of antibiotics is not warranted.

TREATMENT

Evaluation, incision and drainage in the operating room.

Perianal and Pilonidal Abscesses

ETIOLOGY

Ingrowing hair induces abscess formation.

CLINICAL FINDINGS

- Pain, swelling, redness, presence of fluctuant mass.
- Perianal is the most common anorectal abscess (40 to 50%).
- Pilonidal abscesses occur in the midline upper edge of the buttock.

TREATMENT

Incision and drainage followed by later surgical excision.

Renal and Genitourinary Emergencies

218

DEFINITIONS

Classified as prerenal, intrinsic, or postrenal.

Prerenal ARF

ETIOLOGY

- Hypovolemia (blood loss, vomiting, diarrhea, burns).
- Decreased cardiac output.
- Sepsis.
- Third spacing.
- Hypoalbuminemia.
- Drugs (nonsteroidal anti-inflammatory drugs [NSAIDs], angiotensin-converting enzyme [ACE] inhibitors).
- Renal artery obstruction.

DIAGNOSIS

- Urine sodium excretion is < 10 and fractional excretion of sodium (FeNa) is < 1%.
- $FeNa = \dfrac{(Urine\ Na) \times (Plasma\ creatinine) \times 100}{(Plasma\ Na) \times (Urine\ creatinine)}$

TREATMENT

- Volume replacement.
- Diuretics for congestive heart failure (CHF).
- Positive inotropics (e.g., dobutamine) or afterload reduction (e.g., ACE inhibitors) for pump failure.
- Mobilize third-space fluid.

Postrenal ARF

DEFINITION

Obstruction anywhere from renal parenchyma to urethra.

ETIOLOGY

- Nephrolithiasis.
- BPH.
- Neurogenic bladder.
- Bladder neck obstruction.
- Urethral strictures.
- Substances causing renal tubular obstruction: Acyclovir, methotrexate, uric acid, oxalate, myeloma (Bence Jones) proteins.

TREATMENT

- Foley catheter.
- Percutaneous nephrostomy tubes for obstructing renal stones.
- Urology consult if catheterization yields no urine.
- Aggressive hydration may be necessary if tubular obstruction is suspected.

Oliguria is the production of < 400 mL of urine in 24 hours.

Most cases of prerenal failure will need FLUID, FLUID, and more FLUID.

Most common cause of postrenal failure is benign prostatic hypertrophy (BPH).

HIGH-YIELD FACTS

Renal and Genitourinary Emergencies

An accurate history is imperative. Drugs, medical history, and family history are all important to help determine a cause of renal failure.

An 81-year-old woman is brought in by ambulance. She was found lying on the floor of her apartment after sustaining a fall 3 days ago. Her creatine kinase is 12,500. Her blood urea nitrogen/creatinine (BUN/Cr) is 100/45. *Think: Rhabdomyolysis.*

A 23-year-old man presents with hypertension and hematuria. He reports having a sore throat 2 weeks ago. His BUN/Cr is 34/2.0. *Think: Poststreptococcal GN.*

Intrinsic ARF

DEFINITION

Insult to the kidney parenchyma from disease states, drugs, or toxins.

ETIOLOGY

- Acute tubular necrosis:
  - Intravenous (IV) contrast
  - Acute ischemia
  - Myoglobinuria from rhabdomyolysis
  - Drugs (aminoglycoside antibiotics, ACE inhibitors, NSAIDs)
- Glomerulonephritis (GN):
  - Antecedent streptococcal (group A, beta-hemolytic)
  - Systemic lupus erythematosus
  - Wegener's granulomatosis
  - Polyarteritis nodosa
  - Goodpasture's
  - Henoch–Schönlein purpura
  - Drugs (gold, penicillamine)
  - Immunoglobulin A nephropathy (Berger's disease)
  - Idiopathic
- Acute interstitial nephritis

TREATMENT

- Treat underlying cause.
- Discontinue any offending agents.
- Increase urine output in oliguric patients with hydration and diuretics (mannitol, furosemide).
- Increase renal perfusion with dopamine if needed.
- Consider dialysis for severe cases.

► **CHRONIC RENAL FAILURE (CRF)**

OVERVIEW

- Patients developing CRF are treated with diet and medications first and progress to use of intermittent dialysis and finally chronic dialysis.
- Uremia, electrolytes, anticoagulants, immunosuppression, vascular access, and cardiovascular stress with hemodialysis all contribute to potential problems.

EMERGENCIES

- Arrhythmias: Due to electrolyte imbalances and drug toxicities:
  - Hyperkalemia is the most common when dialysis appointments are missed.
  - Others include hypocalcemia, hypokalemia (during or immediately after dialysis), and hypermagnesemia.
- Hypertension: Due to increased intravascular volume:
  - Need dialysis but may temporize with IV nitroprusside, hydralazine, or labetalol.

- Hypotension: Due to ultrafiltration during dialysis:
  - Give IV fluid and pressors as necessary.
- Neurological:
  - Lethargy, seizures, coma, headache, and confusion all may occur.
  - Electrolytes, hypoglycemia, and concurrent illness (e.g., sepsis) all may be contributing factors.
  - Must rule out intracranial bleed (especially if projectile vomiting or focal neurological exam) because of use of IV heparin during hemodialysis. Most common intracranial bleed here is subdural hematoma.
- Hemodialysis disequilibrium:
  - Syndrome that occurs toward the end of dialysis, usually after the first dialysis treatment.
  - Characterized by nausea, vomiting, high blood pressure, and a feeling of light-headedness. Can progress to seizures, coma, death.
  - Treatment consists of raising serum osmolality with mannitol and terminating dialysis.
- Gastrointestinal (GI):
  - Upper GI bleeds from anticoagulation, uremic gastritis, and peptic ulcer disease are more common than in the general population.
  - Bowel obstruction may occur due to use of oral phosphate binders. Avoid Mg^{2+}-containing antacids and Fleet enemas (contain phosphate).
- Vascular:
  - External vascular access devices or internal shunts and grafts may become clotted or infected.
  - Strictures, aneurysms, vascular steal syndromes, or excessive bleeding may occur in the extremity where the graft is.
  - Take care to avoid blood draws, blood pressure measurements, or other procedures in that extremity.
- Genitourinary:
  - Uremia: Discussed below.

EMERGENT HEMODIALYSIS

Indications
- Electrolyte abnormalities: Hyperkalemia is the most common and potentially the most dangerous even at moderate levels.
- Volume overload and its various manifestations—the patient may be oliguric; also may have uncontrollable hypertension.
- Intractable acidosis; $HCO_3^- < 10$.
- Severe uremia.
- Dialysis may also be necessary to treat certain drug overdoses.

PERITONEAL DIALYSIS

Problems occur with the intraperitoneal catheter:
- It can become clogged or kinked, resulting in fluid overload and abdominal distention.
- Adhesions may form in the peritoneal space, decreasing fluid drainage.
- If aseptic technique is not followed, peritonitis may occur manifested by abdominal tenderness and GI symptoms:
  - If systemic symptoms are present, IV antibiotics are necessary. Otherwise, antibiotic infusion into the peritoneum suffices as treatment.

CRF patients are sick. Anything out of the ordinary in these patients should be investigated.

► HEMATURIA

ETIOLOGY

- GN
- BPH
- Vascular:
  - Renal vessel thrombosis
  - Abdominal aortic aneurysm (AAA)
  - Arteriovenous malformation
- Urologic cancer:
  - Bladder cancer
  - Renal cell carcinoma
  - Rhabdomyosarcoma
- Urolithiasis/nephrolithiasis
- Acute GN
- Pseudohematuria:
  - Vegetable dyes (e.g., beets, rhubarb)
  - Phenolphthalein
  - Phenazopyridine
  - Porphyria
  - Contamination from menstrual blood
- Sickle cell disease
- Trauma
- Infection:
  - *Schistosoma haematobium*
  - Sexually transmitted disease (STDs)
  - Urinary tract infection (UTI)

CLINICAL FINDINGS

- Start with a urinalysis and go from there.
- Timing of hematuria:
  - At initiation of the stream suggests a urethral source.
  - At the end of the stream suggests prostate or bladder neck problems.
  - Continuous hematuria has a renal, bladder, or ureteral source.
- "Brown" or "Coca-Cola" urine has a renal source: Hematuria, GN, and myoglobinuria.

► PROTEINURIA

DEFINITION

> 150 mg/24 hours in adult patients.

CLINICAL FINDINGS

- Tubular source (impaired reabsorption) has > 2 g/day excretion (e.g., diabetic or hypertensive nephropathy).
- Glomerular source (diffusion across glomerular membrane) may have up to 10 g/day excretion (e.g., nephrosis).
- These criteria apply if proteinuria is isolated. A nephritic picture will have proteinuria but other urinalysis findings as well (e.g., casts).

EPIDEMIOLOGY

- Peak incidence in midlife.
- Three times more common in men.

PATHOPHYSIOLOGY

- ~90% of stones are radiopaque.
- Stone composition:
  - Caoxalate: 75%, radiopaque.
  - Struvite: 15%, radiopaque.
  - Urate: 10%, radiolucent.
  - Cystine: 1%, radiopaque.
- Stones partially obstruct at five different places where the most pain occurs:
  - Renal calyx
  - Ureteropelvic junction
  - Pelvic brim
  - Ureterovesicular junction (tightest space)
  - Vesicular orifice
- Stone passage:
  - Rarely fully obstruct the ureter due to their shapes.
  - < 5-mm stones—almost always pass freely.
  - 5- to 8-mm stones—15% will pass freely.
  - > 8-mm stones—only 5% will pass freely.

RISK FACTORS

- Medications (hydrochlorothiazide, acetazolamide, allopurinol, antacids, excess vitamins)
- Male gender
- Dehydration
- Hot climate
- Family history
- Inflammatory bowel disease
- Gout
- Hyperparathyroidism
- Immobilization
- Sarcoidosis
- Malignancy

DIFFERENTIAL DIAGNOSIS

- AAA
- Testicular/ovarian torsion
- Ectopic pregnancy
- Salpingitis
- Pyelonephritis
- Renal infarction
- Appendicitis
- Drug-seeking behavior
- Musculoskeletal strain

A 34-year-old man presents with left flank pain radiating to his left testicle. The pain does not change with movement or position and is colicky in nature. Urine dip is positive for blood. *Think: Renal colic.*

It is unusual to see first-time presentation of kidney stone in the elderly. Even when the story seems classic, investigate symptoms in the elderly carefully. The most commonly missed diagnosis is AAA.

CLINICAL FINDINGS

- Pain:
  - Flank, abdominal, or back pain, with radiation to groin.
  - Patients are very restless and cannot sit (opposite of patients with peritonitis who tend to lie perfectly still).
  - Waxes and wanes.
- Possibly: Nausea, vomiting, ileus, hematuria (micro- or macroscopic), low-grade fever, urinary urgency/frequency.

DIAGNOSIS

- Although intravenous pyelogram (IVP) is considered the gold standard, in most emergency departments (EDs), noncontrast abdominal CT (Figure 10-1) is the first-line test. Ultrasonography is another good imaging option (Table 10-1).
- KUB (kidney, ureter, and bladder) may be able to detect a stone, but it is not helpful to determine pyelonephritis or hydronephrosis. It may be a good screening tool to look for other abdominal pathology.
- Testing is necessary in first-time presentations and in the elderly.

TREATMENT

- Analgesia with NSAIDs and opiates as needed.
- Hydration.
- Admit for:
  - Obstructing stone
  - Infected stone
  - Intractable pain
  - Patient with single kidney
- Nephrostomy tubes may be necessary to relieve severe hydronephrosis.
- Extracorporeal shockwave lithotripsy, cystoscopy, and ureteroscopy may be necessary to mobilize obstructing stones.
- Discharged patients should be instructed to strain their urine to screen for passage of the stone, and return if fever, vomiting, or severe exacerbation of pain should occur.
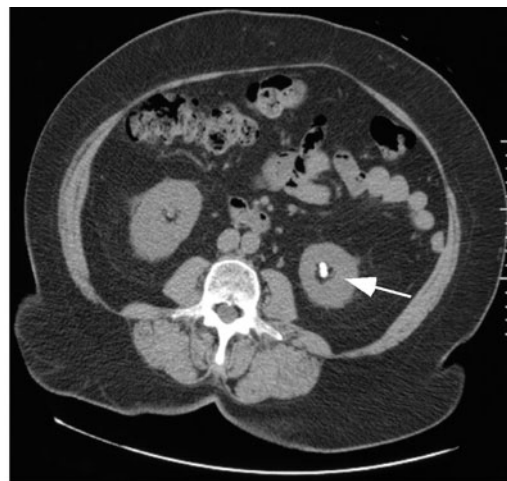
F I G U R E 1 0 - 1 . **Noncontrast abdominal CT demonstrating nephrolithiasis (arrow) in the right kidney, which shows up as radiopaque (white).**

TABLE 10-1. Imaging Modalities for Urolithiasis

	IVP	CT	US
Positive findings	▪ Delay in appearance of nephrogram ▪ Distention of renal pelvis ▪ Distortion of calyx ▪ Extravasation of dye	▪ Presence of suspicious calculi ▪ Dilation of collecting system ▪ Hydronephrosis	▪ Hydronephrosis ▪ Detection of stones in ureter
Sensitivity (%)	▪ 64–90	▪ 95–97	
Specificity (%)	▪ 94–100	▪ 96–98	
Advantages	▪ Provides information on kidney function in addition to anatomy	▪ Fast ▪ No contrast dye	▪ Provides an option for patients who cannot undergo CT or IVP ▪ Especially useful for pregnant women and children (to avoid excess radiation exposure)
Disadvantages	▪ Involves use of contrast dye, which can be nephrotoxic ▪ Time consuming	▪ Does not give you functional information ▪ More radiation than IVP or US	▪ Limited ability to detect smaller stones ▪ Does not give you functional information

► **NEPHROTIC SYNDROME**

DEFINITION

Protein-losing nephropathy with protein loss > 3.5 g/day.

ETIOLOGIES

- Most common cause in adults: Membranous nephropathy.
- Most common cause in children: Minimal change disease.

CLINICAL FINDINGS

- Peripheral edema
- Ascites
- Anasarca
- Hypertension

DIAGNOSIS

- Urinalysis shows oval fat bodies (tubular epithelial casts with cholesterol).
- Hypoalbuminemia.
- Hypercholesterolemia.
- Chest x-ray may demonstrate pleural effusion.

An 8-year-old boy presents with facial edema. Urine dipstick reveals large protein. *Think: Nephrotic syndrome.*

Patients can develop a hypercoagulable state due to nephrotic loss of antithrombin III.

TREATMENT

- Steroids.
- Bed rest.
- Other immunosuppressive agents possible for refractory cases.

▶ TESTICULAR TORSION

DEFINITION

Twisting of a testicle on its root.

EPIDEMIOLOGY

Most common in infants under age 1 and in young adults.

CLINICAL FINDINGS

- Usually occurs during strenuous activity (e.g., athletic event), but sometimes occurs during sleep.
- Pain may be in the lower abdomen, inguinal canal, or testicle. No change in pain with position.
- Twisting is usually in a horizontal direction.
- Physical exam alone is not sufficient to exclude the diagnosis.

DIAGNOSIS

Doppler ultrasound to look for flow to testicle: No flow is highly suggestive of torsion.

TREATMENT

- Immediate urology consult.
- Attempt manual correction of the torsion—untwist from medial to lateral (i.e., patient's right testicle gets rotated counterclockwise and the left clockwise).
- If Doppler ultrasound is equivocal and manual detorsion does not work, immediate exploratory surgery is necessary to prevent death of the testicle.
- Time is testicle! (The longer the delay to definitive treatment, the lower the chance of salvaging the testicle.)

A 19-year-old man presents with severe pain to the right testicle, which occurred suddenly while he was playing baseball. Physical exam reveals a tender, swollen, firm testicle with a transverse lie. There is no cremasteric reflex on that side. *Think: Testicular torsion.*

Manual detorsion is like "opening the book." Imagine a book standing on its spine and the front and back covers are the right and left testes, respectively. "Open" the front cover to untwist the right and likewise for the left.

▶ TORSION OF APPENDIX TESTIS OR APPENDIX EPIDIDYMIS

DEFINITION

Both the testis and epididymis have a small appendix that can become twisted.

CLINICAL FINDINGS

"Blue dot" sign: Palpation of a tender nodule on transillumination of the testes.

TREATMENT

- Get an ultrasound/Doppler to look for blood flow to testes (need to rule out testicular torsion).

- If normal, then appendix testis can be allowed to degenerate.
- Analgesia as needed.

DEFINITION

Inflammation of the testicles.

ETIOLOGIES

- Mumps
- Syphilis

CLINICAL FINDINGS

- Presents with history of bilateral testicular pain.
- Usually will remit after a few days.

TREATMENT

- Treat symptomatically (pain management).
- Disease-specific treatment (e.g., antibiotics for syphilis).

▶ HYDROCELE

DEFINITION

Fluid accumulation in a persistent tunica vaginalis due to obstruction, which impedes lymphatic drainage of the testicles (see Figure 10-2).

ETIOLOGY

- Trauma
- Neoplasia
- Congenital
- Infection: Elephantiasis
- CHF
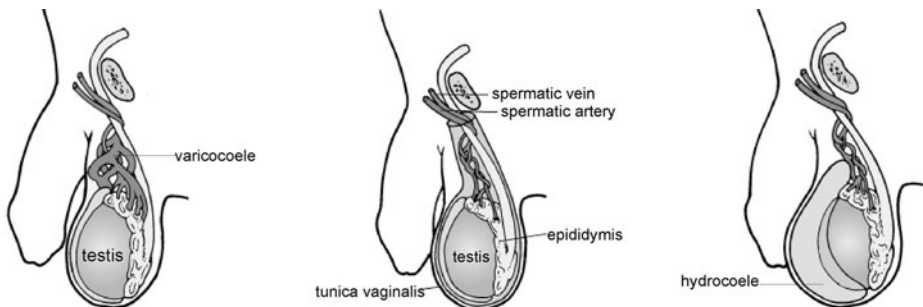
FIGURE 10-2. Varicocele vs. normal vs. hydrocele.

HIGH-YIELD FACTS

Renal and Genitourinary Emergencies

CLINICAL FINDINGS

- May cause much discomfort and pain when distended.
- Other scrotal masses must be ruled out (e.g., torsion).
- Transilluminates on physical exam.

TREATMENT

- Surgical follow-up for drainage.
- Reassurance is usually necessary.

► VARICOCELE

DEFINITION

Varicose vein in scrotal sac (see Figure 10-2).

ETIOLOGY

Caused by venous congestion in the spermatic cord.

CLINICAL FINDINGS

- Palpating "a bag of worms" in the testis.
- Accentuated by Valsalva maneuver and supine position.
- Usually asymptomatic, but persistence has been implicated in sterility.

TREATMENT

May be surgically excised to improve spermatogenesis (elective procedure).

► EPIDIDYMITIS

DEFINITION

Inflammation of the epididymis.

ETIOLOGY

- Bacterial infection.
- Congenital abnormalities with reflux.
- STDs with urethral stricture.

CLINICAL FINDINGS

- Gradual onset of lower abdominal or testicular pain.
- Dysuria.
- There may be isolated firmness or nodularity on the testis.
- May spread to epididymo-orchitis.

TREATMENT

- Antibiotics for infection
- Bed rest
- Scrotal elevation (scrotal support when ambulating)
- Cold compress

A 27-year-old man presents with lower left abdominal pain and left testicular pain for 2 weeks. Palpation of the testes is normal except for isolated tenderness of the epididymis. Cremasteric reflex is normal. *Think: Epididymitis.*

- NSAIDs
- Stool softener

▶ FOURNIER'S GANGRENE

DEFINITION

Rapidly progressive gangrene of groin.

ETIOLOGY

- Usually polymicrobial origin from skin, rectum, or urethra.
- Subcutaneous spread becomes virulent and causes end-artery thrombosis and extensive necrosis in scrotal, medial thigh, and lower abdominal areas.

EPIDEMIOLOGY

Especially prevalent in diabetic and other immunocompromised patients.

TREATMENT

- Broad-spectrum IV antibiotics.
- Surgical debridement.
- Hyperbaric oxygen therapy shown to be of benefit.

A 40-year-old diabetic man presents with severe perineal pain and fever of 103°F. Physical exam demonstrates crepitus over the medial thigh and widespread discoloration of the skin. *Think: Fournier's gangrene.*

The hallmark of Fournier's gangrene is pain of genital area out of proportion to physical exam.

▶ FRACTURE OF PENIS

CLINICAL FINDINGS

- "Snapping sound" during sexual intercourse due to tearing of the tunica albuginea.
- Penis is tender, swollen, and discolored.
- Urethra is usually spared.

TREATMENT

Surgery necessary to evacuate hematoma and to repair the tunica.

A 41-year-old man presents stating he heard his penis "crack" while having intercourse. Physical exam reveals an edematous, purplish penis. *Think: Penile fracture.*

▶ BALANOPOSTHITIS

DEFINITION

Inflammation of the glans penis and foreskin:
- Balanitis = inflammation of glans
- Posthitis = inflammation of foreskin

ETIOLOGY

- Allergy to latex condoms.
- Diabetes mellitus.
- Infection—most commonly with *Candida albicans*.
- Drugs—sulfonamides, tetracyclines, phenobarbital.

Often seen in diabetics and in children who have not learned to clean themselves properly.

CLINICAL FINDINGS

Areas are purulent, excoriated, malodorous, and tender.

TREATMENT

- Preventative therapy with adequate cleaning and drying.
- Topical therapy useful.
- Consider circumcision, especially if recurrent.
- Look for phimosis/paraphimosis.

▶ PEYRONIE'S DISEASE

DEFINITION

Gradual or sudden dorsal curvature of the penis.

ETIOLOGY

- Due to thickened plaque on tunica—may be associated with Dupuytren contractures in the hand.
- May be painful and preclude sexual intercourse.

TREATMENT

Spares urethra and not emergent; reassurance and referral.

▶ PHIMOSIS

DEFINITION

Inability to retract foreskin over glans (proximally).

ETIOLOGY

May be from infection, poor hygiene, old injury with scarring.

CLINICAL FINDINGS

May cause urinary retention secondary to pain or obstruction of urethra.

TREATMENT

Patient will need circumcision or dorsal slit to foreskin.

Do not force retraction of foreskin. May lead to paraphimosis.

▶ PARAPHIMOSIS

DEFINITION

Inability to reduce proximal foreskin over glans (distally).

PATHOPHYSIOLOGY

Edema of the glans leads to venous engorgement, decreased arterial flow, and eventual gangrene.

Paraphimosis is a true urologic emergency!

TREATMENT

Attempt manual reduction or emergent circumcision.

- Manual reduction: Wrap glans with elastic banding for several minutes. Alternatively, several small punctures to edematous area can be made with a 27G needle to express fluid. Local anesthetic block prior to making the punctures is advisable.
- Dorsal slit: See Procedures chapter.

▶ PRIAPISM

DEFINITION

Pathologic erection.

ETIOLOGY

- Sickle cell disease: Sickling in corpus cavernosum.
- Drugs (e.g., prostaglandin E, papaverine, phentolamine, sildenafil, phenothiazines, trazodone).
- Leukemic infiltrate.
- Idiopathic.
- Spinal cord injury.

CLINICAL FINDINGS

Corpus cavernosum with stagnant blood—spongiosum and glans are usually soft.

TREATMENT

- Intramuscular (IM) terbutaline (smooth muscle relaxer).
- Aspiration of blood from cavernosum.
- Hydration, exchange transfusion, and hyperbaric oxygen for sickle cell disease.
- Urologic consult.

COMPLICATIONS

Urinary retention, infection, and impotence.

▶ BENIGN PROSTATIC HYPERPLASIA (BPH)

DEFINITION

The prostate undergoes two growth spurts during life, the second of which begins at around 40 years. This second spurt focuses around the urethra and later in life may cause urinary obstruction.

CLINICAL FINDINGS

- Decreased urinary stream
- Hesitancy
- Dribbling
- Incomplete emptying of bladder

Replace the foreskin when you insert Foley catheters in uncircumcised patients to prevent paraphimosis.

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- Nocturia
- Overflow incontinence
- Chronic urinary retention
- Obstruction
- Enlarged prostate on rectal exam

ETIOLOGY

Infection, drugs (e.g., alpha agonists), and alcohol may exacerbate symptoms to the point where patients are seen in the ED.

DIAGNOSIS

- Urinalysis to look for infection.
- BUN/Cr to look for postrenal failure.
- Prostate-specific antigen (PSA) to monitor for prostate cancer (usually not done in the ED).
- Foley to check post voiding residual volume.
- Sonogram to measure prostate size and look for hydronephrosis.
- Urodynamic studies to determine effect of BPH on urinary flow (outpatient).
- Other outpatient studies such as cystoscopy and IVP are helpful for planning surgical procedures.

TREATMENT

- Avoid things that exacerbate symptoms (e.g., caffeine).
- Leuprolide or finasteride to decrease testosterone levels (factor in growth), which results in decrease of prostate size.
- Alpha blockers to decrease internal sphincter tone (doxazosin, terazosin, tamsulosin, prazosin).
- Transurethral resection of the prostate (TURP) for definitive treatment.

▶ PROSTATITIS

DEFINITION

Inflammation of the prostate.

ETIOLOGY

UTI and STD pathogens.

CLINICAL FINDINGS

- May present with chills, back pain, perineal pain.
- Recurrent UTIs despite treatment.
- Rectal exam will reveal a firm, warm, swollen, tender prostate.
- If exudate is expressed via the urethra, send it for culture.

TREATMENT

- Requires 1 month of total antibiotic therapy because of typically poor penetration into the prostate.
- Acute prostatitis is susceptible to antibiotic treatment with usual UTI antibiotics since inflammation renders the prostate more penetrable.

- Chronically infected prostate without acute inflammation is a relatively "protected" area. Choose your antibiotics wisely—fluoroquinolones have good penetration.

▶ INGUINAL HERNIA

CLINICAL FINDINGS

- Presents as a palpable mass in the inguinal canal or as a scrotal mass.
- The mass is usually reducible (either spontaneously or manually).
- Emergent situations occur when signs and symptoms of intestinal obstruction or severe pain or inability to reduce the mass lead to the diagnosis of incarcerated hernia.

TREATMENT

- Firmer manual reduction may be attempted in the ED, but if irreducible, surgical intervention is necessary.
- Reducible hernias should be referred for eventual repair to avoid incarceration.

Inguinal hernia is a common cause of scrotal or inguinal mass. Listen for bowel sounds over the mass. Transilluminate to rule out hydrocele. When you're convinced, try to reduce.

▶ URETHRAL STRICTURE

DEFINITION

Fibrotic narrowing of urethral lumen.

ETIOLOGY

- Often due to STDs (urethral inflammation → fibrosis).
- Urethral instrumentation.

CLINICAL FINDINGS

- Urinary retention, difficulty voiding.
- Difficulty placing Foley or coudé catheter.

TREATMENT

Catheterization or expansion of urethra with filiform rods.

A 25-year-old man presents with right-sided groin pain that occurred after he attempted to lift a refrigerator. On physical exam, there is a bulge in the inguinal canal. Bowel sounds can be heard over the canal. *Think: Inguinal hernia.*

▶ URINARY RETENTION

DEFINITION

Inability to void completely.

ETIOLOGY

- BPH.
- Drugs (anticholinergics, antihistamines, antispasmodics, alpha-adrenergic agonists, antipsychotics, tricyclic antidepressants).
- Mechanical: Stenosis of urethral meatus, bladder neck contracture, urethral stricture.

Urethral stricture: Differential includes voluntary tightening, bladder neck contraction, and BPH.

- Cancer (bladder and prostate).
- Neurogenic bladder.

CLINICAL FINDINGS

- Inability to void for > 7 hours
- Hesitancy
- Decreased urinary stream
- Lower abdominal pain
- Distended bladder

DIAGNOSIS

- Urinalysis to look for infection.
- BUN/Cr to evaluate renal function.

TREATMENT

- Catheterization is both diagnostic and therapeutic. Patients can be discharged after observation for a few hours with outpatient urology referral.
- Antibiotics for concurrent UTI.

COMPLICATIONS

- Patients with chronic urinary retention can develop post obstructive diuresis when retention is relieved by Foley.
- Post obstructive diuresis is characterized by massive urine output, which can lead to hypotension due to hypovolemia and electrolyte imbalances.

▶ **URINARY TRACT INFECTION (UTI)**

DEFINITION

Infection anywhere from kidney parenchyma (pyelonephritis) to urethral orifice (urethritis).

ETIOLOGY

- UTIs in men often due to anatomical defect.
- Usual culprits are gram-negative aerobes (e.g., *E. coli*) but spectrum varies: *Staphylococcus saprophyticus*, *Proteus* (alkaline urine), *Klebsiella, Enterobacter*.

EPIDEMIOLOGY

- Women: 10 to 20% lifetime incidence.
- Men: 1 to 10% lifetime incidence.
- Institutionalized patients: 50% incidence.

Consider nosocomial
infections with
Pseudomonas and
methicillin-resistant
Staphylococcus aureus in
institutionalized or recently
hospitalized patients.

CLINICAL FINDINGS

- Presentation varies with sites of involvement.
- Pyuria.
- Bacteriuria.
- Dipstick may be positive for leukocyte esterase and nitrites.
- Microscopic or gross hematuria.
- Urine culture and sensitivity.
- White blood cells (WBC) in urinary sediment (> 5 to 10 WBC/hpf).

DIAGNOSIS

- Imaging studies (IVP, ultrasound, CT scan, retrograde urogram) necessary for:
    - All children under 5 (rule out pyelonephritis).
    - All male children (rule out anatomic defect).
    - Recurrent UTIs in women.
    - Fever for more than 3 days with treatment.
    - Recurrent pyelonephritis.
- In severely ill patients: Must rule out things such as perinephric abscess and pyoureter.

TREATMENT

- Trimethoprim–sulfamethoxazole, fluoroquinolones, and aminopenicillins have all been used and are effective; may need culture to streamline treatment.
- Patients with chronic Foley catheters can have asymptomatic bacteruria and need not be treated; if symptomatic, change catheter and treat (probably need admission for IV antibiotics; also beware of fungal infection in these patients if unresponsive to therapy).
- Stable pyelonephritis (no signs and symptoms of sepsis) may be treated as an outpatient with close follow-up.
- In pregnant patients, all bacteriuria must be treated and all pyelonephritis must be admitted for IV antibiotics; higher risk of miscarriage with UTI.

▶ **SEXUALLY TRANSMITTED DISEASES (STDs)**

Gonorrhea

ETIOLOGY

Neisseria gonorrhoeae.

SIGNS AND SYMPTOMS

- Purulent discharge.
- Dysuria, epididymitis, inguinal lymphadenitis, proctitis (in homosexuals).
- Oral or pharyngeal lesions may be present if acquired through oral sex.
- Systemic infection may present as fever, rash, or monoarticular arthritis (usually the knee).

TREATMENT

- Ceftriaxone 125 mg single IM injection

or

- Cefixime 400 mg PO once

or

- Ciprofloxacin 500 mg PO once

or

- Ofloxacin 400 mg PO once

or

- Levofloxacin 250 mg PO once

Due to a high rate (~60%) of concurrent chlamydia infection, treatment for chlamydia should always be included with that for gonorrhea.

Fluoroquinolones such as ciprofloxacin and levofloxacin are contraindicated in pregnancy, lactation, and children due to their potential for damaging cartilage and interference with musculoskeletal development.

Due to a high rate (~60%) of concurrent gonorrhea infection, treatment for gonorrhea should always be included with that for chlamydia.

PainFUL ulcers:
- Chancroid
- Herpes

PainLESS ulcers:
- LGV
- Syphilis

Urethral discharge:
- *Gonococcus*
- *Chlamydia*
- *Trichomonas*

Chlamydia

ETIOLOGY

Specific serotypes of *Chlamydia trachomatis*.

SIGNS AND SYMPTOMS

- Dyspareunia.
- Pelvic pain.
- Yellow mucopurulent discharge.
- Friable, erythematous cervix.
- Tender epididymis if causing epididymitis.

TREATMENT

First-Line
- Azithromycin 1 g PO once

or

- Doxycycline 100 mg PO bid × 7 days

Alternatives
- Ofloxacin 300 mg PO bid × 7 days

or

- Erythromycin base 500 mg PO qid × 7 days

or

- Erythromycin ethylsuccinate 800 mg PO × 7 days

or

- Levofloxacin 500 mg PO × 7 days

Lymphogranuloma Venereum (LGV)

ETIOLOGY

Specific serotypes of *Chlamydia trachomatis* (different from the ones that cause chlamydia).

SIGNS AND SYMPTOMS

- Initial small painless papule that quickly disappears.
- Inguinal lymphadenopathy appears 2 to 6 weeks later, which may suppurate through the skin—these remain painless.
- Extensive scarring and strictures may result.

TREATMENT

Three weeks of doxycycline or erythromycin.

Chancroid

ETIOLOGY

Haemophilus ducreyi.

SIGNS AND SYMPTOMS

- Painful inguinal adenopathy.
- Tender, shallow ulcers with irregular reddish borders.
- Inguinal mass/abscess from coalesced nodes (bubo).

TREATMENT

- Azithromycin 1 g PO once

or

- Ceftriaxone 250 mg IM once

or

- Erythromycin 500 mg PO tid × 7 days

or

- Ciprofloxacin 500 mg PO bid × 3 days

Syphilis

ETIOLOGY

Treponema pallidum.

1° Stage

Painless ulcer (chancre), which is highly ineffective; ulcers heal spontaneously 3 to 6 weeks after primary infection.

2° Stage

Fever, sore throat, rash (trunk with spread to palms and soles), malaise, warts (condylomata lata), aseptic meningitis, lymphadenopathy; also spontaneously resolves.

3° Stage

- May occur after many years of latency.
- Various manifestations in multiple systems:
  - *Argyll Robertson pupil* (small pupil that reacts to accommodation but not light).
  - *Tabes dorsalis* (posterior column disease presenting with loss of position, deep pain and temperature sensation, ataxia, decreased or absent deep tendon reflexes, wide-based gait, urinary retention or incontinence, impotence, and sharp leg pain).
  - *Gumma* (granulomatous, necrotic lesions on the skin and submucosa involving the palate, nasal septum, or other organ).
  - Thoracic aortic aneurysm/dissection (due to spirochetes in aortic vasa vasorum).

TREATMENT

For 1° and 2°
- Benzathine penicillin G 2.4 million units IM once for *early* latent syphilis (Bicillin LA). Repeat the dose three times a 1-week intervals for *late* latent syphilis.
- Dose for children is 500,000 U/kg IM up to adult dose.
- If penicillin-allergic, consider desensitization or doxycycline or tetracycline PO × 2 weeks.

For Neurosyphilis
- Aqueous penicillin G 2.4 million units every 4 hours × 10 to 14 days

or

- Ceftriaxone 1 g IV/IM daily × 14 days

The Jarisch–Herxheimer reaction is an acute febrile reaction that occurs ~24 hours after treatment for early syphilis. It presents with headache and severe myalgias. It can cause early labor and fetal distress in pregnancy.

There are several preparations of penicillin G, including benzathine, aqueous procaine, and aqueous crystalline. The mixed forms are not appropriate therapy for syphilis.

Human Papillomavirus (HPV)

ETIOLOGY

HPV.

SIGNS AND SYMPTOMS

- Genital and anal warts causing discomfort but not pain.
- Condylomata acuminata when warts coalesce.

TREATMENT

Self-Treatments (Applied by Patient)
- Podofilox 0.5% solution or gel:
  - Applied to visible genital warts bid × 3 days, followed by 4 days of no therapy; cycle can be repeated up to four times.
  - Total wart area treated should not exceed 10 cm^2.
  - Total volume of podofilox should be limited to 0.5 mL/day.
  - Safety of podofilox during pregnancy has not been established.
- Imiquimod 5% cream:
  - Applied once daily at bedtime, three times a week for up to 16 weeks.
  - Treatment area should be washed with soap and water 6 to 10 hours after the application.

> The safety of podofilox and imiquimod during pregnancy has not been established.

Physician Treatments
- Cryotherapy with liquid nitrogen or cryoprobe. Applications repeated every 1 to 2 weeks until eradicated.
- Podophyllin resin 10 to 25% in a compound tincture of benzoin. Treatment can be repeated weekly if needed. A small amount should be applied to each wart and allowed to air dry.
- Trichloroacetic acid (TCA) or bichloroacetic acid (BCA) 80 to 90%: Small amount should be applied only to warts and allowed to dry, at which time a white "frosting" develops. If an excess amount of acid is applied, the treated area should be powdered with talc, sodium bicarbonate (i.e., baking soda), or liquid soap preparations to remove unreacted acid. This treatment can be repeated weeky as needed.
- Surgical removal either by tangential scissor excision, tangential shave excision, curettage, or electrosurgery.

Trichomoniasis

ETIOLOGY

Trichomonas vaginalis.

> *Trichomoniasis:*
> Flagellated organisms are seen swimming under the wet mount.

SIGNS AND SYMPTOMS

- Copious, foamy, yellow-green, malodorous discharge with pH > 5.5.
- Punctate red spots on cervix or vaginal wall ("strawberry cervix").
- Labial irritation or swelling.
- Dyspareunia.
- Dysuria.
- Men may be asymptomatic.

> "Whiff test": Two drops of KOH mixed with the discharge and heated onto a slide produces a fishy smell. This is characteristic of both *Trichomonas* and bacterial vaginosis.

TREATMENT

- Single-dose metronidazole (2 g) or 500 mg bid × 1 week. Abstain from alcohol while on drug.
- Clotrimazole for first-trimester pregnancy.

Herpes

EPIDEMIOLOGY

More than 50 million people in the United States are infected.

ETIOLOGY

Herpes simplex virus (HSV). Most caused by HSV-2. Infections caused by HSV-1 tend to have lower recurrence rates.

SIGNS AND SYMPTOMS

- Painful pustular or ulcerative lesions.
- Initial infection more severe than recurrences.
- May have systemic effects (fever, headache, myalgias), left axis deviation, aseptic meningitis.
- Also a causative agent in encephalitis and esophagitis.

TREATMENT

- Oral antiviral therapy helps to control acute symptoms and is also useful for chronic suppressive therapy. However, they do not eradicate the virus and they do not decrease the risk, frequency, or severity of recurrences.
  - Acyclovir 400 mg PO tid × 7 to 10 days
  - Acyclovir 200 mg PO five times a day × 7 to 10 days
  - Famciclovir 250 mg PO tid × 7 to 10 days
  - Valacyclovir 1 g PO bid × 7 to 10 days
- Duration of oral treatment may be extended if healing is imcomplete at 10 days.
- Topical antiviral therapy is not recommended.

▶ **SEXUAL ASSAULT**

EPIDEMIOLOGY

- An estimated 6% of crimes are rapes.
- Approximately one in eight women have been raped, with only 25% of cases reported.
- Two to 4% of rapes are committed against males.

CLINICAL FINDINGS

- The interviewer must determine from the patient the identity and number of persons involved, details on what happened (what kind of assault), areas of pain, how long ago it happened, what happened after the incident, last menstrual period, oral contraceptive use, last consensual intercourse, and allergies.
- Other signs of physical abuse:
  - Facial and extremity injuries are more common than actual injuries to the genitalia.
  - When males are sodomized, they may have thorax and abdomen abrasions because of the position they are in; also anal fissures and lacerations may be seen.
  - Decreased sphincter tone or severe hemorrhoids may indicate chronic sodomy.

The most important aspect of dealing with sexual assault victims is to ensure their psychological well-being. This very traumatic experience could potentially be made worse by the victim's being surrounded and "interrogated" by hospital and police personnel, so every effort should be made to make the patient as comfortable as possible.

- When examining a rape victim, most EDs use a standardized kit provided by the police:
  - All physical injuries (all lacerations and bruises) are documented.
  - A pelvic exam is done, and all mucosal surfaces (oral, vaginal, and anal) are sampled.
  - Testing for STDs is done.
  - Skin and fingernail scrapings are collected.
  - Semen can be sampled if found with the use of a Wood's lamp (fluoresces).

TREATMENT

- Addressing all physical injuries (admission if necessary).
- Tetanus prophylaxis.
- STD and pregnancy testing (offer prophylaxis).
- Hepatitis B prophylaxis.
- Human immunodeficiency virus counseling (offer prophylaxis).
- Most physicians will empirically treat for gonorrhea and chlamydia.
- Medical and psychiatric follow-up should be arranged within 2 weeks.
- Ensure that patient has a safe place to go; arrange for social worker to see patient if needed.

Hematologic and Oncologic Emergencies

1° and 2° Hemostasis

- 1° hemostasis is the initial superficial clotting performed by **platelets.** Defects (e.g., thrombocytopenia) typically result in oozing from IV sites and bleeding from mucous membranes, nose, and gastrointestinal (GI) tract. Also manifest and petechiae and ecchymoses.
- 2° hemostasis is a function of the coagulation cascade and clotting factors. Defects in this process (e.g., hemophilia) result in large, deep bleeds such as hemarthrosis (bleeding into a joint).

Coagulation Cascade

See Figure 11-1.

Heparin

- Increases activated partial thromboplastin time (aPTT).
- Affects intrinsic pathway.
- Decreases fibrinogen levels.
- Primarily affects factors **VIII,** IX, X, XI, XII.
- Low-molecular-weight heparins have 10 times activity against factor Xa.
- Safe in pregnancy.
- Adverse effects include bleeding, thrombocytopenia, and osteoporosis.

PiTT = intrinsic pathway

Heparin goes with **I**ntrinsic pathway because **H** comes right before **I.**

PeT = PT measures extrinsic pathway.
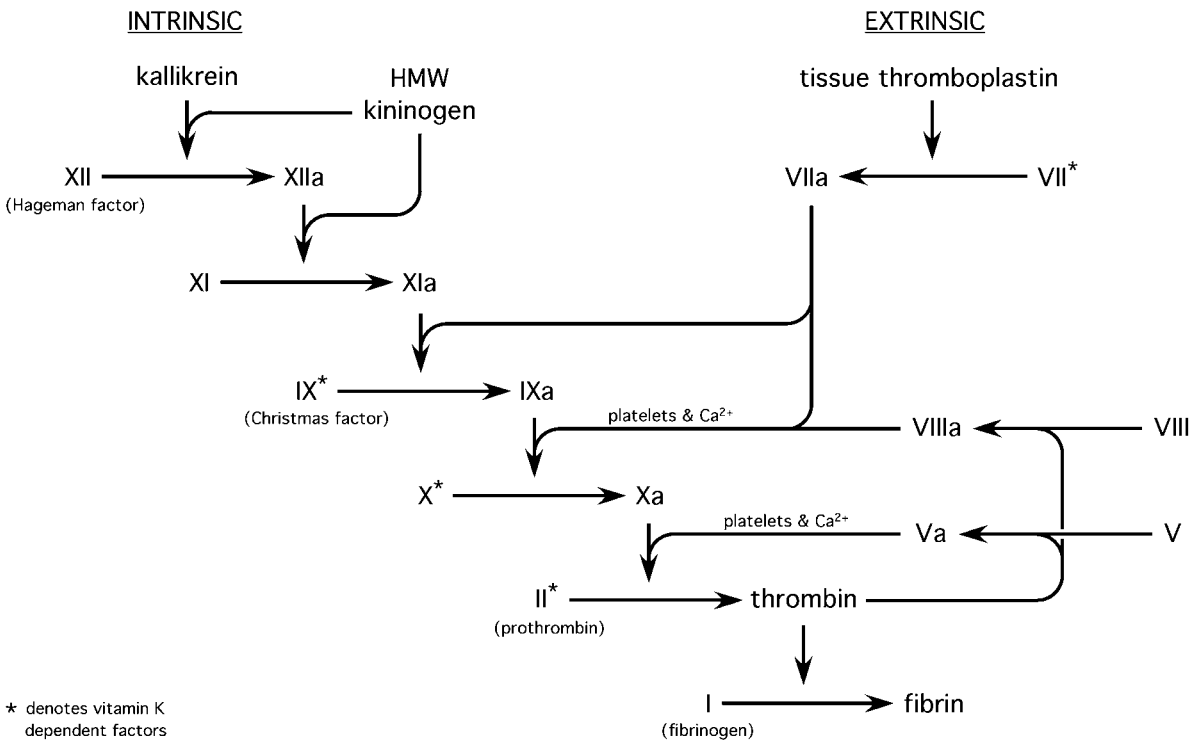
FIGURE 11-1. Coagulation cascade.

Leafy green vegetables have high vitamin K content and can make the international normalized ratio (INR) become subtherapeutic for patients on warfarin.

Warfarin

- Increases prothrombin time (PT).
- Affects extrinsic pathway.
- Decreases vitamin K.
- Primarily affects II, V, **VII.**
- Teratogenic.
- Has an initial *pro*coagulant effect, taking 48 to 72 hours to become anticoagulant. Concurrent coverage with heparin during this time is needed, and oral warfarin dose is titrated slowly.

aPTT

- Tests extrinsic and common pathways.
- Isolated elevation of aPTT (with normal PT) seen in:
  - Heparin therapy.
  - Deficiencies of factors VIII (hemophilia A), factor IX (hemophilia B), factor XI, and factor XII (asymptomatic).

PT

- Tests intrinsic and common pathways.
- Isolated elevation of PT (with normal PTT) seen in:
  - Vitamin K deficiency
  - Warfarin therapy
  - Liver disease (decreased factor production)
  - Congenital (rare)

Thrombin Time

- Measures the time it takes to convert fibrinogen into a fibrin clot.
- Elevated in:
  - Diffuse intravascular coagulation (DIC) (consumes fibrinogen)
  - Liver disease (decreased production of fibrinogen)
  - Heparin therapy (inhibits fibrinogen formation)
  - Hypofibrinogenemia (low fibrinogen to start)

Bleeding Time

- Measures time from start of skin incision to formation of clot (normal = 3 to 8 minutes).
- Independent of coagulation cascade.
- Elevated in:
  - Thrombocytopenia
  - Qualitative platelet disorders
  - von Willebrand's disease (VWD)

▶ HEMOPHILIA A

PATHOPHYSIOLOGY

Sex-linked recessive disease causing a deficiency of factor VIII.

Signs and Symptoms

- Severity of disease varies depending on amount of factor VIII activity.
- Deep tissue bleeding, hemarthrosis (2° hemostasis problems).

Diagnosis

- Prolonged aPTT, normal bleeding time.
- Clinical picture, family history, and the factor VIII coagulant activity level.

Treatment

- Recombinant factor VIII
- Cryoprecipitate

Unlike in VWD, bleeding time in hemophilia A is unaffected because no abnormality with platelets is present.

► HEMOPHILIA B (CHRISTMAS DISEASE)

Pathophysiology

X-linked recessive disease that causes a deficiency of factor IX.

Signs and Symptoms

Identical to hemophilia A.

Diagnosis

Factor IX assay.

Treatment

- Fresh frozen plasma (FFP)
- Recombinant factor IX

► VON WILLEBRAND'S DISEASE (VWD)

Definition

- Type I: Partial quantitative deficiency of von Willebrand factor (VWF) (most common).
- Type II: Qualitative defect of VWF.
- Type III: Almost total absence of VWF.

VWD is the most common inherited bleeding disorder.

Pathophysiology

- VWF is a glycoprotein that is synthesized, stored, and secreted by vascular endothelial cells.
- It functions to (1) allow platelets to adhere to the damaged endothelium and (2) carry factor VIII in the plasma.

Etiology

Usually autosomal dominant inheritance.

EPIDEMIOLOGY

- One in 100 live births have some defect in VWF.
- Only 1 in 10,000 manifests a clinically significant bleeding disorder.

SYMPTOMS AND SIGNS

1° hemostasis problems: Epistaxis, GI bleeding, easy bruising, menorrhagia, prolonged bleeding after dental extraction.

DIAGNOSIS

- Prolonged bleeding time (platelets don't adhere well).
- Prolonged aPTT (factor VIII is decreased).
- Normal PT.
- Normal platelet count.
- Definitive diagnosis made with abnormal assay of VWF, VWF:antigen, or factor VIII:C (usually not in the emergency department [ED]).

TREATMENT

- Type I: Desmopressin
- Types II and III:
  - Factor VIII concentrates with large amounts of VWF:
    - Synthetic-treated product, no risk of infection.
    - Provides VWF most efficiently, with the least amount of volume.
  - Cryoprecipitate and FFP:
    - Will also work, but carry risk of infection and provide low concentration of VWF for given volume, resulting in volume overload for severe cases.

▶ **ANTIPHOSPHOLIPID ANTIBODY SYNDROME**

- Defined as presence of lupus anticoagulant or anticardiolipin antibodies in addition to thrombosis and/or pregnancy complications.
- Most commonly manifests as recurrent fetal losses in women of childbearing age.
- Antiphospholipid antibody impairs in vivo anticoagulant pathways.
- Diagnosis is by lupus anticoagulant and anticardiolipin antibody detection. Tests to detect presence: dilute Russell's viper venom time (dRVVT), kaolin clotting time, dilute phospholipid time.
- Asymptomatic patients need no treatment. Patients with thrombosis need long-term anticoagulation. Pregnant patients can be treated with heparin.

▶ **THROMBOCYTOPENIA**

DEFINITION

Platelet count < 140,000.

ETIOLOGY

Increased Destruction
- Antibody-coated platelets removed by macrophages:
  - Idiopathic thrombocytopenic purpura (ITP).

Causes of thrombocytopenia:
PLATELETS
Platelet disorders: TTP, HUS, ITP, DIC
Leukemia
Anemia
Trauma
Enlarged spleen
Liver disease
EtOH
Toxins (benzene, heparins, aspirin, chemotherapy agents, etc.)
Sepsis

- Human immunodeficiency virus (HIV)-associated thrombocytopenia.
    - Transfusion reactions.
    - Some drug-induced thrombocytopenias.
- Thrombin-induced platelet damage:
    - DIC (seen with obstetrical complications, metastatic malignancy, septicemia, and traumatic brain injury).
- Removal by acute vascular abnormalities:
    - Thrombotic thrombocytopenic purpura (TTP).
    - Hemolytic uremic syndrome (HUS).
    - Adult respiratory distress syndrome–induced thrombocytopenia.

Decreased Production
- Decreased megakaryocytes in marrow:
    - Leukemia
    - Aplastic anemia
- Normal megakaryocytes:
    - Alcohol-induced reactions
    - Megaloblastic anemias
    - Some myelodysplastic syndromes
    - Medications (e.g., antibiotics)

Sequestration in Spleen
- Cirrhosis with congestive splenomegaly
- Myelofibrosis with myeloid metaplasia

RISK FACTORS

- Drugs (chemotherapeutic agents, ethanol, thiazides, antibiotics)
- Prior thrombocytopenic episodes
- Underlying immunologic disorder
- Massive blood transfusions
- Significant EtOH consumption
- Term pregnancy

SIGNS AND SYMPTOMS

1° hemostasis signs:
- Petechiae
- Purpura
- Heme-positive stool
- Recurrent epistaxis, gingival bleeding, or menorrhagia
- Hepatosplenomegaly (jaundice, spider angiomas, and palmar erythema may be present if condition is due to EtOH abuse)

DIAGNOSIS

- Complete blood count (CBC) with platelet morphology and manual platelet count.
- PT/PTT.
- Bleeding time.
- Liver function tests (LFTs).

TREATMENT

- Treatment depends on cause.
- Typically, no need to give platelets unless < 10,000/μL or active bleed.
- Platelets are contraindicated in TTP.

Antiplatelet drugs:
CAFE PORN
C[4]: Cardiovascular drugs
Cholesterol-lowering
drugs
Cough suppressants
Chemotherapy
A[4]: Antimicrobials
Antihistamines
Anticoagulants
Anesthetics
F: Food
E: Ethanol

P: Psychotropics
O: Opiates
R: Radiocontrast agents
N: NSAIDs

A 42-year-old woman with no past medical history presents due to petechiae that have erupted over her arms and legs in the past 2 days. She also reports gingival bleeding. Physical exam reveals petechiae within the oral cavity as well. Labs demonstrate a platelet count of 7,000/mm³, normal PT/aPTT, and a prolonged bleeding time. *Think: ITP.*

► **CHEMICALLY INDUCED PLATELET DYSFUNCTION**

Antiplatelet Therapy

- Platelets are significant components of the thrombotic response to damaged coronary and cerebral artery plaques.
- Prompt antiplatelet therapy can halt progression and significantly reduce morbidity/mortality from acute myocardial infarction and cerebrovascular accident.

ASPIRIN (ASA)

Irreversibly acetylates platelet cyclooxygenase: As platelets have no biosynthetic machinery, it is *inactivated for the life span of the platelet* (8 to 10 days).

TICLOPIDINE (TICLID)

- Irreversibly inhibits conversion of platelet surface receptor to its high-affinity binding state.
- Prevents fibrinogen receptor expression.
- Lasts for life span of platelet.

CLOPIDOGREL (PLAVIX)

- Ticlopidine analog, similar mechanism
- Rapid onset of action, can be used acutely
- Intravenous (IV) administration possible

TREATMENT

With all of these drugs, if bleeding occurs, you must give platelets because although the patient may have a normal count, the platelets are dysfunctional.

► **IDIOPATHIC THROMBOCYTOPENIC PURPURA (ITP)**

DEFINITION

An autoimmune-mediated destructin of platelets.

ETIOLOGY

- Adult ITP usually results from antibody development against a structural antigen present on the platelet surface.
- Childhood ITP is thought to be triggered by a virus, which produces an antibody that cross-reacts with an antigen on the platelet surface.

DIAGNOSIS

- Peripheral blood smear should be unremarkable with the exception of decreased platelets.
- Bone marrow is normal with the exception of possibly increased megakaryocytes.

Adults
- Initial treatment is prednisone 1 mg/kg/day. Platelet levels usually rise over the coming weeks, during which time the steroid dosage is tapered.
- Most patients fail to have a sustained response and will go on to have a splenectomy. This causes remission in 50 to 60% of patients.

Patients with ITP and Life-Threatening Bleeding
- High-dose steroids.
- Suppress mononuclear phagocyte clearance of platelets by administering IV immunoglobulin at 1 g/kg for 1 day.
- Platelet transfusion for life-threatening bleeding.

▶ **THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP)**

DEFINITION

Severe disorder in which fibrin strands are deposited in multiple small vessels: This damages passing RBCs and platelets and results in thrombocytopenia and microangiopathic hemolytic anemia.

EPIDEMIOLOGY

- Female more than male.
- Age 10 to 45 years.

RISK FACTORS

- Pregnancy, often indistinguishable from severe preeclampsia (see HELLP syndrome).
- Drugs: Quinine, cyclosporine, mitomycin C, ticlopidine, H_2 blockers, oral contraceptives, penicillin.
- Autoimmune disorders (e.g., systemic lupus erythematosus).
- Infection (including *E. coli* O157: H7, *Shigella dysenteriae*, and HIV).
- Allogeneic bone marrow transplantation.
- Malignancy.

SIGNS AND SYMPTOMS

- Fever.
- Waxing and waning mental status (correlates with lodging and dislodging of thrombus in cerebral vessels).
- Pallor.
- Petechiae.
- Colicky pain of various body parts (again due to thrombus in vessels).

DIAGNOSIS

- Diagnosis requires a combination of clinical suspicion and correlation with appropriate lab analyses.
- Peripheral blood smear: Schistocytes and helmet cells.
- CBC: Anemia, thrombocytopenia, elevated reticulocyte count.

TTP diagnostic pentad:
- Fever
- Altered mental status
- Renal dysfunction
- Microangiopathic hemolytic anemia
- Thrombocytopenia

You do *not* need all present for diagnosis!

A 33-year-old woman is brought to the ED after her sister found her febrile and confused. Physical exam reveals fever, tachycardia, some mucosal bruising, a waxing and waning mental status, and trace heme-positive stool. Labs demonstrate a platelet count of 22,000/mm³, normal PT/aPTT, elevated bilirubin, and a BUN/Cr of 40/2.0. Peripheral smear shows schistocytes. *Think: TTP.*

DO NOT transfuse platelets in patients with TTP, this could kill them.

- Blood urea nitrogen/creatinine (BUN/Cr): Azotemia.
- Urinalysis: Hematuria, red cell casts, and proteinuria.
- LFTs: Elevated lactic dehydrogenase, elevated bilirubin (unconjugated > conjugated), low haptoglobin.

TREATMENT

- **Do not transfuse platelets.**
- Plasmapheresis is mainstay of treatment (given daily, until platelet count rises to normal).
- May give FFP if plasmapheresis not available.
- Transfuse packed RBCs if anemia is symptomatic (tachycardia, orthostatic hypotension, hypoxia).
- Consider corticosteroids, vincristine, antiplatelet agents, and splenectomy for refractory cases.
- Monitor for and treat acute bleeds (remember to look for intracranial bleed as well).
- Admit patients to the intensive care unit.

► HEMOLYTIC UREMIC SYNDROME (HUS)

DEFINITION

Disorder thought to be on the same continuum as TTP (earlier) with renal dysfunction as its primary feature.

ETIOLOGY

Unknown.

EPIDEMIOLOGY

- Most common in childhood.
- Adult form also seen.

RISK FACTORS

- Infection with *E. coli* O157: H7 or *Shigella dysenteriae*.
- Ingestion of undercooked meats and unpasteurized products.

SIGNS AND SYMPTOMS

- GI symptoms (nausea, vomiting, abdominal pain).
- Oliguria.
- Pallor.
- GI bleeding.
- Seizures can result as a complication of renal failure, due to hypertension, hyponatremia, fluid overload, and electrolyte imbalances.

DIAGNOSIS

Same as TTP. Test for *E. coli* O157 infection.

TTP is a disease with high mortality when not diagnosed and treated early. Also, it is a disease that recurs, with each subsequent episode being slightly worse. Patients eventually die of multiple thrombi lodged in the brain, kidneys, and other organs.

HUS diagnostic triad:
- Renal failure
- Microangiopathic hemolytic anemia
- Thrombocytopenia

A 6-year-old boy presents with abdominal pain, oliguria, diarrhea, and fever. Several kids from his school came down with the same thing after his birthday party at a local hamburger chain. Labs demonstrate acute renal failure. *Think: HUS.*

Platelet transfusion is not contraindicated for HUS as it is for TTP.

TREATMENT

- Dialysis and supportive care.
- Plasmapheresis is sometimes used in adults.

PROGNOSIS

- The time for highest mortality is during the course of the disease, when central nervous system (CNS) complications can result.
- Most children recover without sequelae after the acute illness.
- Some children will have progressive renal dysfunction and hypertension and should be monitored for a period of at least 5 years.
- Adults do not recover so well and usually have residual renal failure.

▶ DISSEMINATED INTRAVASCULAR COAGULATION (DIC)

DEFINITION

DIC is a coagulopathy that happens when both the fibrinolytic and coagulation cascades are activated.

RISK FACTORS

- Infection: Usually from gram-negative organisms (endotoxin causes generation of tissue factor activity on the plasma membrane of monocytes and macrophages). Probably the most common cause.
- Trauma:
  - Crush injuries, brain trauma, burns.
- Obstetrical complications:
  - Abruptio placentae.
  - Saline-induced therapeutic termination.
  - Retained products of conception.
  - Amniotic fluid embolism.
- Malignancy:
  - Mucin-secreting adenocarcinomas of pancreas and prostate.
  - Acute promyelocytic leukemia.
- Shock from any cause.
- Snake bites.
- Heat stroke.
- Severe transfusion reaction.
- Drugs.
- Foreign bodies such as peritoneovenous shunts.

PATHOGENESIS

- Results from generation of tissue factor in the blood or the introduction of tissue factor–rich substances into the circulation.
- Tissue factor is the most fibrinogenic substance known, and it initiates coagulation.
- Coagulative activity is difficult to regulate once it is begun in this fashion, and soon the factors of coagulation have been exhausted, resulting in coagulopathy.

A 27-year-old woman who is 39 weeks pregnant is the victim of a high-speed motor vehicle crash. She presents with vaginal bleeding and uterine irritability. A few hours later, she goes into shock and begins bleeding profusely from her multiple lacerations. Labs show prolonged PT/aPTT, thrombocytopenia, and fibrin split products. *Think: DIC due to abruptio placentae.*

PT/aPTT are normal in ITP, TTP, and HUS. They are abnormally elevated in DIC.

SIGNS AND SYMPTOMS

- Sites of recent surgery or phlebotomy bleed profusely and cannot be controlled with local measures.
- Ecchymoses form at sites of parenteral injections.
- Serious GI bleeding may ensue at sites of erosion of the gastric mucosa.

DIAGNOSIS

- Presence of fibrin split products.
- Thrombocytopenia.
- Markedly prolonged PT/PTT.
- Low fibrinogen concentration.
- Elevated plasma D-dimers (are the cross-linked fibrin degradation by products).
- Schistocytes on peripheral smear.

TREATMENT

- Treat underlying cause.
- If a transient process, give cryoprecipitate (has fibrinogen) and platelets for temporary support.
- For DIC-associated bleeding with raised PT and aPTT, use FFP.

▶ **HEPARIN-INDUCED THROMBOCYTOPENIA (HIT)**

DEFINITION AND CLINICAL MANIFESTATIONS

- Type I HIT is a transient, harmless, and mild drop in platelets, usually 1 or 2 days after initiation of heparin. Patient may be kept on heparin and platelets will return to normal.
- Type II HIT (true HIT) is an immune-mediated process against the heparin–platelet complex. It is manifested by both arterial and venous **thrombosis,** as well as thrombocytopenia. It is life threatening. It typically presents 4 to 10 days after heparin exposure *or* after several hours if patient has had prior heparin exposure.

DIAGNOSIS

- Clinical suspicion.
- Heparin-induced platelet aggregation assay (HIT assay). This is specific but insensitive.
- Serotonin release assay.

TREATMENT

- Immediate removal of any heparin exposure (including low-molecular-weight heparins), including flushes and locks.
- Anticoagulation is the mainstay of treatment, as most complications are caused by thrombosis. Lepirudin and argatroban are direct thrombin inhibitors used in this setting. Warfarin **should not** be used initially as it can precipitate worsening thrombosis.

Several entities must be considered in the differential. The outcomes and treatments are very different.

- **Gestational thrombocytopenia:** Mild and asymptomatic, usually with platelet count > 70,000 μL, typically occurring in the third trimester. There are no sequelae, and it resolves after delivery.
- **Preeclampsia** (discussed in greater detail in the Obstetric Emergencies chapter): Hypertension, proteinuria, and edema typically developing in the third trimester. Eclampsia is the above with the addition of seizures. Typically, platelets are decreased in these patients more than usual gestational thrombocytopenia. Delivery of the child is the mainstay of treatment.
- **HELLP syndrome** (hemolytic anemia, elevated liver enzymes, low platelets): This has the same features as preeclampsia with the addition of hemolysis and elevated liver function tests (LFTs) (thrombocytopenia can be seen in both). This is life threatening, and delivery is indicated. Diagnose by platelet count, lactic dehydrogenase (LDH) (high indicates hemolysis), LFTs, and peripheral blood smear (schistocytes indicate microangiopathic hemolysis).

Sickle Cell Anemia (SCA)

DEFINITION

- Genetic disease characterized by the presence of hemoglobin S in RBCs.
- Hemoglobin S is formed by substitution of valine for glutamine in the sixth position of the β-hemoglobin chain.
- During periods of high oxygen consumption, this abnormal hemoglobin distorts and causes cell to sickle.
- Sickle cell trait: Heterozygous for sickle gene.
- Sickle cell disease: Homozygous for sickle gene.

EPIDEMIOLOGY

- More common in blacks than whites.
- Increased incidence in populations from Africa, the Mediterranean, Middle East, and India.

PATHOPHYSIOLOGY

- Deoxygenated hemoglobin S undergoes a conformational change with low O_2 tension.
- When enough hemoglobin S molecules change conformation, the hemoglobin molecules crystallize, forming a semisolid gel in the interior of the RBC.
- This causes the RBC to adopt a sickle shape (Figure 11-2).
- The distorted RBCs are inflexible and plug small capillaries, leading to occlusion and ischemia/infarction.

> *Features of SCA:*
> **SICKLE**
> **S**plenomegaly, sludging
> **I**nfection
> **C**holelithiasis
> **K**idney — hematuria
> **L**iver congestion, leg ulcers
> **E**ye changes

> Patients with SCA are prone to infection with encapsulated organisms. *Salmonella* osteomyelitis is most common among patients with SCA.

HIGH-YIELD FACTS

Hematologic and Oncologic Emergencies

FIGURE 11-2. Peripheral smear demonstrating sickled RBCs (arrows).

A 24-year-old man with known sickle cell disease presents with pain in his lower back and knees. He is afebrile, has an O₂ saturation of 100%, and normal labs. *Think: Vaso-occlusive crisis of sickle cell disease.*

- The sickled cells also have an increased propensity to adhere to the capillary endothelium.
- The distortion also results in a weakening of the RBC membrane and the cells have a decreased lifespan in the circulation, causing the chronic hemolytic anemia.
- Early in the sickling process, the RBCs can resume their normal shape if O_2 tension is restored; later, the sickling becomes irreversible.
- Low RBC H_2O content can also trigger sickling.
- Early in life, the spleen removes most of the sickled cells from the circulation, causing splenomegaly. Eventually, the toll of continuous sequestration damages the spleen to the point of infarction. The spleen fibroses and shrivels to a fraction of its normal size, often termed *autosplenectomy.*
- Absence of splenic function renders these patients more susceptible to infections, particularly by encapsulated organisms *(Haemophilius influenzae, Pneumococcus, Meningococcus, Klebsiella).*

DIAGNOSIS

- All newborns at risk in the United States are screened for the disease.
- Peripheral smear: Howell–Jolly bodies (cytoplasmic remnants of nuclear chromatin that are normally removed by the spleen), sickled cells.
- Blood tests show anemia, increased reticulocyte count, and increased indirect bilirubin.
- Hemoglobin electrophoresis will show hemoglobin S.

Typical Vaso-occlusive Crisis

- Symptoms include arthralgias and pain.
- Caused by vascular sludging and thrombosis. Uncomplicated crisis is treated with hydration and analgesics. Crisis is often precipitated by infection, so one should have a low threshold for antibiotics.

Sickle Cell Emergencies

Most sickle cell emergencies are due to vaso-occlusive crisis infarcting a particular organ.

Acute Chest Syndrome

- Fever, chest pain, cough, shortness of breath, and pulmonary infiltrates on chest radiograph.
- Thought to be due to infection and occlusion of pulmonary microvasculature.

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- This is a major cause of mortality in patients > 5 years old.
- Diagnosis of pulmonary embolism should usually be excluded with ventilation–perfusion scanning or computed tomographic (CT) angiography.
- Oxygenation should be monitored. Antibiotics and hydration are treatment. Exchange transfusion if severe. Always give supplemental oxygen as well.

Aplastic Crisis

- Life-threatening complication characterized by severe pancytopenia.
- Due to medullary sickling, common complication of infection with parvovirus B19.

CNS Crisis

- This is the only type of vaso-occlusive crisis that is painless (no pain receptors).
- Cerebral vascular occlusion is more common in children, and cerebral hemorrhage is more common in adults.
- Evaluation should include CT scanning.
- Lumbar puncture should be performed if CT is negative and headache is present to exclude diagnosis of subarachnoid hemorrhage.

Priapism

Sickling in corpus cavernosum of penis causing protracted painful erection. This can lead to impotence. Draining of corpora cavernosa can be done. Also, subcutaneous terbutaline can be used.

Acute Hepatic Sequestration Crisis

Sequestration and sickling of RBCs in liver leading to high bilirubin and severe anemia. Aggressive transfusion is treatment as well as exchange transfusion if severe.

Acute Splenic Sequestration

- Second most common cause of death in children with SCA (by adulthood, autosplenectomy will have disposed of this source of morbidity/mortality).
- Sickled cells block outflow from the spleen, causing pooling of blood and platelets in the spleen. In major sequestration crisis, the hemoglobin drops three points from baseline or to a level less than 6 g/dL.

Renal Papillary Necrosis

- Characterized by flank pain and hematuria.
- Occurs because of the very high osmolalities in renal medulla needed to pull the water from the collecting ducts causing the RBCs to sickle.

Thalassemias

ETIOLOGY

- Defective globin chain synthesis.
- Patients with beta-thalassemia have decreased production of beta-globin chain. Likewise, patients with alpha-thalassemia have defective production of a chains and accumulate excessive beta-globin chains.

The best treatment for SCA patients is education about their disease, specifically, how to avoid vaso-occlusive crises.

Hematologic and Oncologic Emergencies

CLINICAL FEATURES

- Severity of anemia dependent on extent of disease..
- Hepatosplenomegaly.
- Bone marrow expansion causing osteopenia.

DIAGNOSIS

- Microcytosis, hypochromasia on peripheral smear.
- CBC may or may not reveal anemia.
- Hemoglobin electrophoresis.

TREATMENT

- Thalassemia carrier and traits are asymptomatic and need no treatment.
- Patients with hemoglobin H (one functional alpha-globin chain) and beta-thalassemia major may or may not require intermittent transfusions.
- In all such cases presenting to the ED with manifestations of severe anemia or hemolysis, a thorough search for precipitating events like infection or oxidative stress should be undertaken.

Glucose-6-Phosphate Dehydrogenase (G6PD) Deficiency

- G6PD maintains glutathione in reduced state to prevent oxidative damage to RBCs.
- Males are affected more commonly as the gene for G6PD is carried on X chromosome.
- Severity is proportional to magnitude of enzyme deficiency.
- Majority of patients stay asymptomatic unless exposed to infections or oxidative stress.
- Diagnosis is by quantitative assay.
- Treatment is primarily preventive and symptomatic management.

► TRANSFUSION REACTIONS

ABO Incompatibility

- Most common transfusion reaction.
- Almost invariably due to **human error.**
- Patients are immunized against A/B antigen (Ag) without prior exposure because endogenous bacteria produce glycoproteins with structures similar to the A/B Ag.
- Which antibody (Ab) form is actually determined by the patient's own A/B status.

Rh immunoprophylaxis should be considered for all pregnant patients who sustain any abdominal trauma regardless of the amount of vaginal bleeding.

Non-ABO Incompatibility

- Uncommon, occurring mostly in multitransfused patients.
- One to 1.5% risk of red cell alloimmunization per unit transfused.
- Fifteen to 20% incidence in multitransfused patients.
- Most commonly Ab to Rh and Kell (K) Ag, less frequently Duffy (Fy) and Kidd (Jk) Ag.

- Rh-negative mothers who give birth to Rh-positive children have a 15 to 20% chance of developing anti-Rh Ab due to fetal–maternal hemorrhage.
- Anti-Rh$_o$ immune globulin is routinely given to Rh-negative mothers pre- and perinatally to prevent Rh immunization.

► COMMON ED PRESENTATIONS OF CANCER COMPLICATIONS

Malignant Pericardial Effusions

- Seen with cancers of lung and breast, Hodgkin's and non-Hodgkin's lymphoma, leukemia, malignant melanoma, and postradiation pericarditis.
- Large effusions can lead to cardiac tamponade (see Trauma chapter).

Syndrome of Inappropriate Antidiuretic Hormone (SIADH)

- Most commonly associated with small cell carcinoma.
- Other cancers that can cause SIADH are brain, thymus, pancreas, duodenum, prostate, and lymphosarcoma.
- See Diagnostics chapter for description.

Adrenal Crisis

- Seen with malignant melanoma and cancers of breast, lungs, and retroperitoneal organs.
- See Endocrine Emergencies chapter for description.

Neutropenic Fever

- Defined as fever and absolute granulocyte count < 500/mm^3.
- Patients are at great risk of developing overwhelming sepsis.
- Treated with hydration, broad-spectrum antibiotics, and reverse isolation. Start antibiotics immediately; do not wait for a source.

Superior Vena Cava (SVC) Syndrome, Acute Spinal Cord Compression, and Tumor Lysis Syndrome

See individual sections below.

► SUPERIOR VENA CAVA (SVC) SYNDROME

DEFINITION

Acute or subacute obstruction of the SVC due to compression, infiltration, or thrombosis.

A 53-year-old female with known breast cancer presents with facial and upper extremity edema and distended neck veins. She states the symptoms are worse in the morning. She is afebrile; her lungs reveal mild crackles bilaterally to auscultation. *Think: SVC syndrome.*

EPIDEMIOLOGY

- Usually from a malignant tumor (90%). Lung cancer and lymphoma are most common causes.
- Nonmalignant causes are more rare. Thrombosis, goiter, and aortic aneurysm are examples.

PHYSIOLOGY

- Blood flow: Internal jugular and subclavian veins → brachiocephalic (innominate) veins → SVC ← azygous vein ← bronchial veins.
- Blockade above the azygous vein manifests less severely, as chest wall collaterals allow bypass of the obstruction and because unobstructed drainage of the bronchial veins avoids many of the pulmonary problems.
- Due to lack of gravitational assistance with drainage, many symptoms are more severe in the recumbent position and in the morning after sleep.
- Slow-growing tumors allow more time for collateral development, and thus a less severe picture, than rapidly growing ones.

SIGNS AND SYMPTOMS

- Distended neck veins (67%).
- Isolated upper extremity edema, particularly periorbital and facial (56%).
- Pulmonary manifestations (40%): Shortness of breath, tachypnea, cyanosis, crackles, rales.
- Less commonly, sequelae of increased intracranial pressure (ICP): Papilledema, cerebral edema, altered mental status, visual disturbances, headache, seizures, coma, cerebral hemorrhage, death.

DIAGNOSIS

- Chest x-ray: Pleural effusion, mass apparent in only 10% of cases.
- Chest CT/magnetic resonance imaging (MRI) for mass.
- If mass detected, biopsy for definitive diagnosis.

TREATMENT

- Supportive measures.
- Increased ICP may require emergent surgical resection.
- Definitive treatment depends on etiology of obstruction.
- Steroids are often used to decrease any inflammatory component.

► SPINAL CORD COMPRESSION

DEFINITION

Malignancy metastasizing to and destroying vertebral bodies, causing cord impingement.

ETIOLOGY

- Most common malignancies: Lung, breast, prostate.

- Other causes: Melanoma, myeloma, renal cell cancer, lymphoma, vertebral subluxation, epidural hematomas, and intramedullary metastasis.
- Spinal distribution:
  - 15% cervical
  - 68% thoracic
  - 19% lumbosacral

SIGNS AND SYMPTOMS

- Back pain is most common symptom. Usually, it is localized to level of compression but later stages may have radicular quality.
- Weakness is often present, usually in lower extremities.
- Loss of sensation is also common. Saddle anesthesia is classic for cauda equina syndrome.
- Loss of bladder and bowel function is a late finding.

DIAGNOSIS

- Diagnostic study of choice is magnetic resonance imaging (MRI).
- Myelography can also be used. Positive myelogram shows dye obstruction at level of lesion.
- Plain films of spine (90% show evidence of tumor, but not whether it is compressing the spinal cord).

TREATMENT

- Rapid treatment is essential as duration of symptoms is inversely proportional to chances of recovery. Pretreatment neurologic status is most important prognostic factor (i.e., the more compromised at presentation, the less likely to recover).
- High-dose steroids to control inflammation/edema.
- Oncology consult.
- Radiation is usual therapy of choice, but depends on the radiosensitivity of the tumor.
- Surgery is superior in certain scenarios (e.g., a protuberant bone from a pathologic fracture).

▶ TUMOR LYSIS SYNDROME

DEFINITION

Acute life-threatening condition arising from massive release of lysed tumor cell cytosol and nucleic acids. Complications include renal failure and life-threatening electrolyte disturbances.

PATHOLOGY

- Usually occurs 1 to 5 days after instituting antineoplastic therapy (chemotherapy or radiation).
- Likelihood of syndrome increases with tumor bulk and sensitivity to antineoplastic therapy.
- Generally low risk with solid tumors.
- Most common with hematologic malignancies: Acute leukemias, lymphomas (particularly Burkitt's).

- Hyperuricemia:
  - Due to DNA breakdown.
  - Due to urate nephropathy.
- Hyperkalemia:
  - Due to release of cytosol.
  - Cardiac dysrhythmias.
- Hyperphosphatemia:
  - Due to protein breakdown.
  - Nephropathy due to precipitation of calcium phosphate crystals.
- Hypocalcemia:
  - Due to hyperphosphatemia driving renal excretion of Ca^{2+}.
  - Neuromuscular symptoms: Muscle cramps, tetany, convulsions.
  - Cardiac dysrhythmias.
  - Confusion.
- Uremia:
  - Due to protein breakdown.
- Renal failure:
  - Due to urate and Ca^{2+} crystal deposition.

TREATMENT

- Hydration.
- Allopurinol to decrease the uric acid.
- Alkalination of urine with bicarbonate to make the uric acid more soluble.
- Close monitoring and correction of electrolytes.

Gynecologic Emergencies

Ovarian Cysts

TYPES

Follicular Cysts
- First 2 weeks of menstrual cycle (most common).
- Pain secondary to stretching of capsule/rupture of cyst.
- Usually regress spontaneously in 1 to 3 months.

Corpus Luteum Cysts
- Last 2 weeks of menstrual cycle (less common).
- Bleeding into cyst cavity may cause stretching or rupture of capsule.
- Usually regress at end of menstrual cycle.

PCO classic triad:
- Obesity
- Hirsutism
- Oligomenorrhea

Polycystic Ovaries (PCO)
- Endocrine disorder: Hyperandrogenism and anovulation.
- Menses occur infrequently but are heavy and painful.
- Ovarian cysts possibly secondary to chronic anovulation.
- Long-term management is with oral contraceptives.

SIGNS AND SYMPTOMS

Cysts are usually asymptomatic unless complicated by rupture, torsion, or hemorrhage.

DIAGNOSIS

Ultrasound is useful for visualizing cysts and signs of rupture (free fluid in pelvis).

TREATMENT

Most complications are treated surgically.

Ovarian Torsion

DEFINITION

Twisting of the ovary on its stalk.

EPIDEMIOLOGY

Most common in the mid-20s.

PATHOPHYSIOLOGY

- Venous drainage is occluded, but arterial supply remains patent.
- Ovarian edema, hemorrhage, and necrosis may occur rapidly.

SIGNS AND SYMPTOMS

- Sudden onset of severe unilateral pelvic pain.
- More commonly associated with the presence of enlarged ovary (tumor, cyst, abscess, or hyperstimulated with fertility drugs).
- Patients often give history of similar pain that resolved spontaneously (twisting/untwisting).
- Unilateral adnexal tenderness on pelvic exam.

DIAGNOSIS

Doppler ultrasound reveals decreased or absent flow to ovary and can demonstrate location of an adnexal mass.

TREATMENT

- Laparotomy/laparoscopy usually successful early on.
- Advanced cases may require oophorectomy.

Ovarian Tumors

- Malignant tumors are less common than benign ones but have the highest mortality of all gynecologic malignancies.
- They usually present late in course with abdominal distention (secondary to massive ascites).

▶ VAGINAL DISORDERS

Bacterial Vaginosis

DEFINITION

Most common vulvovaginitis.

ETIOLOGY

- Marked decrease in numbers of lactobacilli (protective).
- Infection with organisms such as *Peptostreptococcus* species, *Bacteroides* species, and *Gardnerella vaginalis*.

SIGNS AND SYMPTOMS

Fishy-smelling itchy discharge.

DIAGNOSIS

Via wet mount of vaginal smear.

Diagnostic Criteria for Bacterial Vaginosis
- White, noninflammatory vaginal discharge (relative absence of white blood cells [WBCs]).
- Clue cells (epithelial cells coated by bacteria) on microscope (Figure 12-1).
- pH > 4.5.
- "Whiff test" (fishy odor to discharge after adding KOH).

TREATMENT

First-Line
- Metronidazole 500 mg PO bid × 7 days.
- Metronidazole 0.75% gel intravaginally bid × 5 days.
- Clindamycin cream 2% intravaginally hs × 3 days.

Adnexal torsion is a gynecologic emergency.

The most common ovarian tumor is a benign cystic teratoma (dermoid cyst).

Lactobacilli are abundant in yogurt. A Yoplait a day keeps vaginitis away.

Bacterial vaginosis in pregnancy can cause preterm labor, so it should always be treated, even if the patient is asymptomatic.
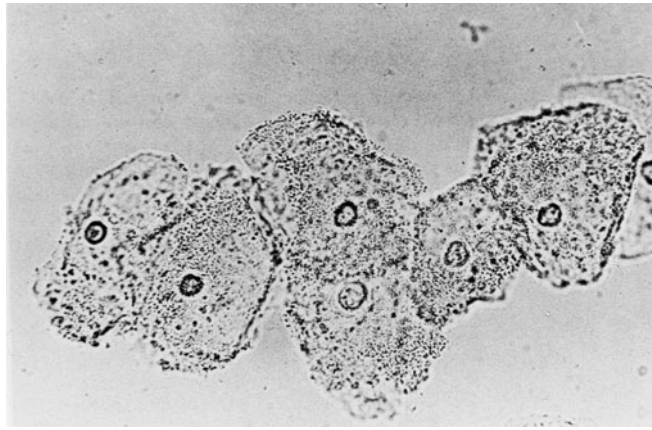
FIGURE 12-1. Clue cells in bacterial vaginosis.

(Reproduced, with permission, from Knoop K, Stack LB, Storrow AB. *Atlas of Emergency Medicine*. New York: McGraw-Hill, 1997: 488.)

> Warn patients against having *any* alcohol while on metronidazole (not even a teaspoonful of alcohol-containing mouthwash). It can cause a disulfiram-like reaction when co-ingested with alcohol, resulting in severe retching.

> *T. vaginalis* is the most common cause of vaginitis worldwide.

Alternatives
- Single 2-g PO metronidazole (causes extreme nausea).
- Clindamycin 300 mg PO bid × 7 days.
- Clindamycin ovules 100 g intravaginally hs × 3 days.

During Pregnancy
- Metronidazole 250 mg PO tid × 7 days

or

- Clindamycin 300 mg PO bid × 7 days

Trichomoniasis

DEFINITION

Sexually transmitted vulvovaginitis.

ETIOLOGY

Trichomonas vaginalis, a flagellated protozoan.

SIGNS AND SYMPTOMS

- Copious, yellow-green, malodorous, foamy vaginal discharge (pH > 5.5).
- Punctate hemorrhages on cervix or vaginal wall ("strawberry cervix").
- Dyspareunia.
- Dysuria.
- Labial irritation or swelling.
- Ninety percent of infected men are asymptomatic. The trichomonads live in the seminal fluid.

DIAGNOSIS

Presence of motile trichomonads on wet mount (Figure 12-2).
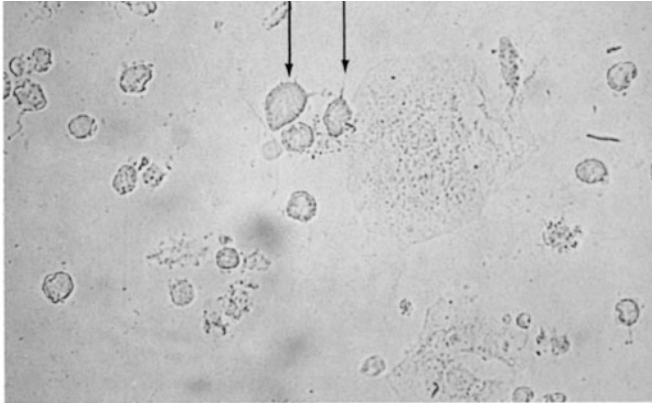
TREATMENT

Metronidazole 2 g PO once or 500 mg PO bid × 7 days.

COMPLICATIONS

Associated with increased risk of:
- Premature rupture of membranes
- Preterm delivery
- Postpartum endometritis

Candidiasis

DEFINITION

Most common fungal infection.

ETIOLOGY

Candida albicans.

RISK FACTORS

- Diabetes mellitus
- Stress
- Human immunodeficiency virus (HIV)
- Post antibiotic therapy
- Pregnancy
- Oral contraceptive therapy

SIGNS AND SYMPTOMS

- White "cottage cheese" discharge (pH < 4.5).
- Beefy red swollen labia.
- Pruritus.

DIAGNOSIS

Presence of pseudohyphae on 10% KOH prep (Figure 12-3).

A woman presents with a recurrent vaginal candidiasis that is refractory to treatment. *Think: Diabetes mellitus.* Get a blood glucose.
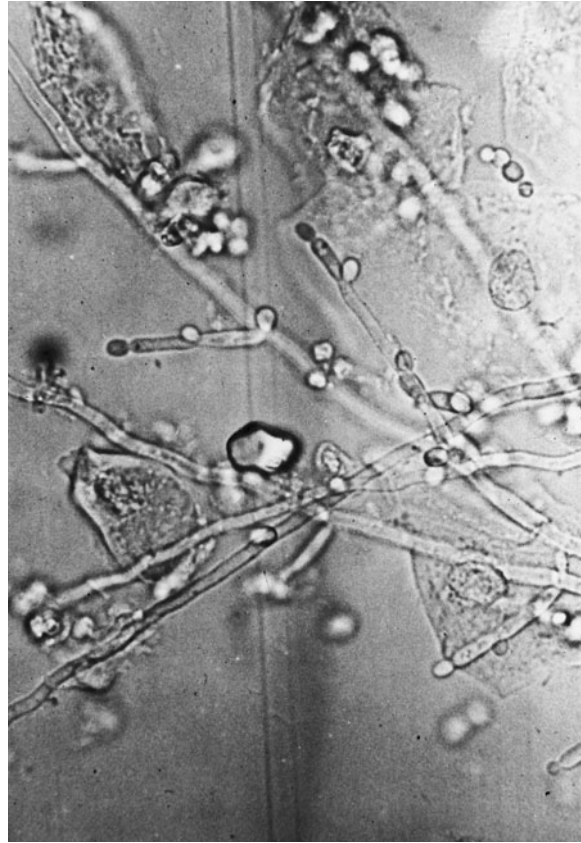
FIGURE 12-3. Pseudohyphae seen in candidiasis.

(Reproduced, with permission, from Pearlman MD, Tintinalli JE, eds. *Emergency Care of the Woman.* New York: McGraw-Hill, 1998: 544.)

TREATMENT

Multiple antifungal preparations (oral and intravaginal) are available.

Contact Vulvovaginitis

DEFINITION

Vulvovaginitis caused by exposure to chemical irritant or allergen (douches, soaps, tampons, underwear, topical antibiotics).

SIGNS AND SYMPTOMS

- Erythema and edema of labia.
- Clear watery discharge.

TREATMENT

- Removal of offending substance.
- Sitz baths for mild cases.
- Topical steroids for severe cases.

Atrophic Vaginitis

DEFINITION

Decreased estrogen stimulation of vagina leads to mucosal atrophy.

ETIOLOGY

- Pregnancy and lactation
- Postmenopause

SIGNS AND SYMPTOMS

- Red, dry-appearing labial mucosa.
- Atrophic vagina is predisposed to ulceration and superinfection.

TREATMENT

- Topical vaginal estrogen cream.
- Hormone replacement therapy for postmenopausal women.

▶ UTERINE DISORDERS

Endometriosis

DEFINITION

Presence of endometrial glands/stroma outside the uterus that may affect ovaries, fallopian tubes, bladder, rectum, or appendix.

PATHOPHYSIOLOGY

Most commonly accepted hypothesis is "retrograde menstruation":
- During menses, uterus contracts against partially closed cervix.
- Menstrual flow passes retrograde into fallopian tubes and pelvic cavity.
- Ectopic endometrial tissue then responds to cyclic hormonal influence.

SIGNS AND SYMPTOMS

- Pain most often occurs just before and during menses.
- Classic triad of endometriosis:
  - Dysmenorrhea
  - Dyspareunia
  - Dyschezia

DIAGNOSIS

Is suspected clinically, confirmed by direct visualization (laparoscopy).

TREATMENT

- Analgesia for acute episodes (nonsteroidal anti-inflammatory drugs or opiates).
- Hormonal therapy (suppress normal menstrual cycle) for long-term control.
- Surgery for cases refractory to medical management.

Every woman who presents with abdominal/pelvic pain or vaginal bleeding should have a documented β-hCG test. All women are pregnant until proven otherwise.

Bleeding in postmenopausal women (over 6 months after cessation of menopause) may represent early signs of cervical or endometrial neoplasia and should be referred for urgent gynecologic follow-up.

Warn patients that heavy withdrawal bleeding may follow cessation of oral contraceptive therapy.

Dysfunctional Uterine Bleeding

TYPES AND CAUSES

- Ovulatory regular menstrual periods with intermenstrual bleeding:
  - Causes include oral contraceptives, persistent corpus luteum, and uterine fibroids.
- Anovulatory chronic estrogen stimulation without cyclic progesterone:
  - Hyperstimulated endometrium thickens and sheds irregularly.
  - Most common during menarche and menopause.
  - Increased risk of endometrial hyperplasia/adenocarcinoma.
- Miscellaneous:
  - Carcinoma, polyps, condylomata, lacerations (trauma), retained foreign bodies, endometriosis, blood dyscrasias, anticoagulant use.

TREATMENT

- Treatment in the hemodynamically stable, nonpregnant patient is primarily supportive.
- Oral contraceptive pills (several regimens) will stop the bleeding and may be useful to "jump start" a regular cycle, although still anovulatory.
- Refer for gynecologic follow-up.

▶ **CERVIX**

Anatomy

- The cervix is the lowest portion of the uterus, composed primarily of collagen with 10% smooth muscle.
- Cervical dysplasia may occur secondary to infection, inflammation, or neoplasia.
- Abnormalities noted on speculum exam should be referred for gynecologic follow-up.

Cervicitis

DEFINITION

Inflammation of the cervix, most often due to infection.

ETIOLOGY

- *Neisseria gonorrhoeae*
- *Chlamydia trachomatis*
- *Trichomonas vaginalis*

RISK FACTORS

Unsafe sexual practices.

SIGNS AND SYMPTOMS

- Yellow mucopurulent discharge
- Dysuria
- Friable cervix

DIAGNOSIS

- Culture of discharge.
- Wet mount to look for WBCs or motile trichomonads.

TREATMENT

See Renal and Genitourinary Emergencies chapter for specific treatment regimens for gonorrhea, chlamydia, and trichomoniasis.

► BARTHOLIN'S GLAND ABSCESS

PATHOPHYSIOLOGY

- Bartholin's (vestibular) gland lies at 5 and 7 o'clock positions of vestibule.
- Secretions normally provide lubrication during intercourse.
- Obstruction of gland leads to cyst formation, which may develop into abscess.

ETIOLOGY

Most common pathogens:
- *Neisseria gonorrhoeae*
- *Chlamydia trachomatis*
- *Staphylococcus aureus*
- *Streptococcus faecalis*
- *Escherichia coli*
- Anaerobes
- Normal vaginal flora

TREATMENT

- Incision and drainage (usually under conscious sedation).
- Once the abscess cavity is drained, a balloon catheter is left in the cavity for continuous drainage while healing (6 weeks). The patient may engage in all activities including intercourse while it is in place.
- Antibiotics.
- Definitive surgical excision may be indicated for recurrent abscesses.

► PELVIC INFLAMMATORY DISEASE (PID)

DEFINITION

Ascending infection of the vagina, fallopian tubes, and ovaries.

PATHOPHYSIOLOGY

Bacterial infection involving female upper reproductive tract cases are presumed to originate with a sexually transmitted disease of the lower genital tract, resulting in inflammation and scarring.

RISK FACTORS

- Previous PID
- Multiple sexual partners

Due to the high rate of concurrent chlamydia and gonorrhea infection, treatment for both is given when either is suspected.

There is a high rate of recurrence of Bartholin's abscess secondary to fistulous tract formation.

PID is a leading cause of female infertility.

PID is a risk factor for infertility, chronic pelvic pain, and ectopic pregnancy.

The most accurate way to diagnose PID is via laparoscopy.

A 19-year-old woman presents with bilateral abdominal pain, fever (101°F), nausea, vomiting, and general malaise. Her last menstrual period was 3 days ago. Pelvic exam reveals exquisite cervical motion and bilateral adnexal tenderness. *Think: PID.*

PID is uncommon in pregnancy due to the plug formed by the fusion of the chorion and decidua, providing an additional natural barrier to infection.

- Intrauterine device use
- Douching
- Instrumentation of cervix

ETIOLOGY

- The most common pathogens are *Neisseria gonorrhoeae* and *Chlamydia trachomatis*.
- Anaerobes, gram-negatives, and mycoplasma are less common.

CLINICAL PRESENTATION

- Can be subtle.
- Adnexal/uterine tenderness.
- Cervical motion tenderness.
- Fever may be present.

DIAGNOSIS

Most Common
- Abnormal cervical or vaginal mucopurulent discharge.
- Presence of WBC on saline microscopy of vaginal secretions (wet mount).
- Laboratory documentation of cervical infection with *N. gonorrhoeae* or *C. trachomatis*.

Most Specific Criteria
- Endometrial biopsy with histopathologic evidence of endometritis.
- Transvaginal sonography or magnetic resonance imaging (MRI) techniques showing thickened, fluid-filled tubes with or without free pelvic fluid or tubo-ovarian complex.
- Laparoscopic abnormalities consistent with PID.

Nonspecific
- Elevated erythrocyte sedimentation rate (ESR) or C-reactive protein.

CENTERS FOR DISEASE CONTROL GUIDELINES FOR INPATIENT ADMISSION

- Uncertain diagnosis
- Suspected tubo-ovarian abscess (TOA)
- Fever > 102.2°F
- Failure of outpatient therapy
- Pregnancy
- First episode in a nulligravida
- Inability to tolerate PO intake
- Inability to follow up in 48 hours
- Immunosuppressed patient
- Other considerations for admission:
  - Pediatric patient
  - Presence of infected foreign body

TREATMENT

Inpatient Therapy (Parenteral)
- Cefotetan or cefoxitin plus doxycycline

or

- Clindamycin plus gentamicin

Outpatient Therapy (14-Day Course)

- Ceftriaxone or cefoxitin IM plus doxycycline

or

- Ofloxacin plus metronidazole

COMPLICATIONS

- TOA
- Fitz-Hugh–Curtis syndrome
- Septic abortion
- Intrauterine growth retardation
- Premature rupture of membranes
- Preterm delivery

► **TUBO-OVARIAN ABSCESS (TOA)**

- Common and potentially fatal complication of PID.
- Intravenous antibiotics curative in 60 to 80% of cases.
- Surgical drainage or salpingectomy/oophorectomy in resistant cases.
- Ruptured TOA presents with shock and 15% mortality rate.

The most common organism in TOA is *Bacteroides.*

► **FITZ-HUGH–CURTIS SYNDROME**

- "Perihepatitis" secondary to ascending gonorrhea/chlamydia infection (infection tracks up fallopian tubes into paracolic gutters).
- Mild elevations of liver function tests with symptoms of diaphragmatic irritation.
- "Violin string" adhesions are classic anatomic findings.
- Treatment with intravenous antibiotics (as for PID) is usually curative.

Obstetric Emergencies

Human Chorionic Gonadotropin (hCG)

- Presence of beta subunit of hCG is used as criteria for positive pregnancy test.
- Produced by trophoblastic tissue ~8 to 9 days after ovulation.
- Maintains corpus luteum (which maintains progesterone production).
- After 6 to 8 weeks, progesterone production shifts to placenta.

Human Placental Lactogen (hPL)

- Produced by placenta, increases throughout pregnancy.
- Antagonizes insulin → increased glucose levels.

Prolactin

- Rises in response to increasing maternal estrogen.
- Stimulates milk production.

Progesterone

- Produced by the ovaries (up to 8 weeks) and placenta (after 8 weeks).
- Prevents uterine contractions.

Estrogens

- Produced by both fetus and placenta.
- Limited role in monitoring course of pregnancy and fetal well-being.

Cortisol

- Both maternal and fetal adrenal production.
- Responsible for differentiation of type II alveoli → surfactant production.
- Antagonizes insulin → increased glucose levels.

Cardiovascular

- Plasma volume increases to ~150% of pregestational levels.
- Increase in red blood cell (RBC) mass less than plasma volume → hemoglobin/hematocrit will drop slightly ("anemia of pregnancy").
- Heart rate and stroke volume both rise → increased cardiac output.
- Systolic BP/diastolic BP/mean arterial pressure all decrease until 20 weeks, then rise again.
- Gravid uterus obstructs venous return from lower extremities.
- Increased blood flow to kidneys (waste) and skin (heat).

Patients in second and third trimester of pregnancy may experience a significant drop in blood pressure (BP) when lying down. This "supine hypotensive syndrome" is relieved by turning onto the left side, taking weight of the uterus off the vena cava.

HIGH-YIELD FACTS

Obstetric Emergencies

Respiratory

- Functional residual capacity decreases due to effects of gravid uterus.
- Increased tidal volume and minute ventilation.
- Hyperventilation leads to chronic respiratory alkalosis.

Renal

- Progesterone causes smooth muscle dilatation (ureters, bladder).
- Renal bicarbonate excretion compensates for respiratory alkalosis.
- Both renal plasma flow and glomerular filtration rate increase.
- Renin levels are elevated → increased angiotensin levels.

Metabolic

- By 10th week, increased insulin levels and anabolic activity.
- Insulin resistance and hPL/cortisol activity → elevated glucose levels.

Endocrine

- Estrogen stimulates thyroxine-binding globulin → increased triiodothyronine (T_3)/thyroxine (T_4) levels.
- Both adrenocorticotropic hormone and cortisol levels increased after 3 months.

▶ **PRENATAL CARE**

Routine Tests

- Blood tests (blood counts, type and screen, Rh factor, glucose screen).
- Syphilis, rubella, hepatitis B, human immunodeficiency virus (HIV).
- Serum alpha-fetoprotein (between 16 and 20 weeks).
- Ultrasonography (around 16 to 20 weeks).
- Cervical cultures for gonorrhea, chlamydia, group B strep, and cytology.

With sudden weight gain in the third trimester, consider preeclampsia.

Monitoring

- Weight gain (26 to 28 pounds is average).
- Urinalysis (glucose, protein).
- Blood pressure (BP).
- Fundal height (after 22 weeks) approximates age of fetus.
- Fundal height above pubic symphysis in centimeters approximates weeks gestation.
- Fetal assessment: Attitude, lie, presentation, position.
- Nonstress test (NST): Assess for fetal heart rate accelerations in response to movement.
- Amniotic fluid index (AFI): Assess for oligohydramnios (AFI < 5 cm).
- Reactive NST and adequate AFI with normal fetal movement constitutes a normal biophysical profile.

Asymptomatic bacteriuria in the pregnant woman is treated with antibiotics (e.g., nitrofurantoin).

Ectopic Pregnancy

EPIDEMIOLOGY

- Leading cause of pregnancy-related death in the first trimester.
- Second leading cause of all maternal mortality.
- ~10% mortality rate.

PATHOPHYSIOLOGY

- Zygote implants outside uterus (95% in fallopian tubes).
- Aborts when vascular supply to abnormal placenta disrupted, but may rupture as well.

RISK FACTORS

Risk: APPRAISE IT!
- **A**ge
- **P**revious ectopic
- **P**elvic inflammatory disease (PID)
- **R**ace
- **A**IDS and other sexually transmitted diseases (STDs)
- **I**ntrauterine device (IUD)
- **S**moking
- **E**lective abortion
- **I**nfertility treatment
- **T**ubal surgery or scarring

SIGNS AND SYMPTOMS

Classic triad:
- Abdominal pain
- Vaginal bleeding
- Amenorrhea

Spectrum anywhere from asymptomatic up to hemorrhagic shock.

DIAGNOSIS

β-hCG
- Sensitivity of pregnancy tests: Urine positive > 20 mIU/mL.
- Serum normal pregnancy—β-hCG increases by at least 66% for the first 6 to 7 weeks from day 9. If it does not, suspect ectopic pregnancy.

Progesterone in the Presence of Classic Triad
- < 5 ng/mL highly suggestive of EP.
- > 25 ng/mL highly suggestive of IUP.

Ultrasound
- Used to establish presence or absence of IUP (Figure 13-1).
- Presence of echogenic adnexal mass and pelvic free fluid is highly suggestive of EP (Figure 13-2).
- Can usually visualize IUP (gestational sac) by transvaginal sonogram at β-hCG > 1,000 (approximately 5 weeks) and by transabdominal sonogram at 5,000.

> A 29-year-old woman with history of PID presents due to abdominal pain and vaginal spotting. Her last menstrual period was 10 weeks ago. No intrauterine pregnancy (IUP) can be detected on ultrasonography. *Think: Ectopic pregnancy (EP).*

> *Transvaginal IUP sono findings:*
> Gestational sac: ~1,000)
> Yolk sac: ~2,500)
> Heart tones: ~10,500–17,000

FIGURE 13-1. Intrauterine pregnancy.

Arrowhead shows gestational sac; small arrow shows yolk sac.

Eating small frequent meals and things such as toast and crackers may help the ease the nausea and vomiting and keep some food down.

- Incidence of coexisting EP/IUP (heterotopic) is 1/4,000, but increases to 1/100 in women on fertility drugs.

TREATMENT

- Medical management: Methotrexate for termination.
- Surgery for hemodynamic instability or if medical management not feasible.
- Rh-immune globulin for Rh-negative women.

Hyperemesis Gravidarum

DEFINITION

- Syndrome of intractable nausea and vomiting in a pregnant woman.
- Usually occurs early in pregnancy and resolves by end of first trimester.

TREATMENT

- Fluid and electrolyte abnormalities are common and should be replaced as indicated.
- Metoclopramide is class B and effective treatment for nausea and vomiting.
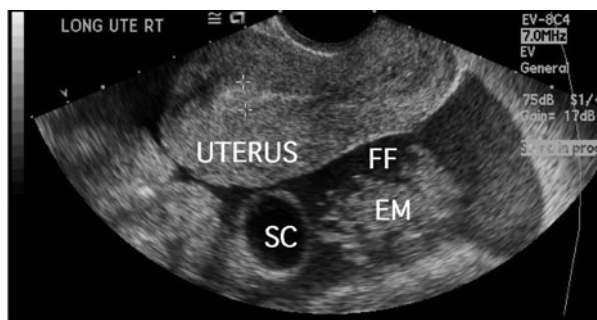
FIGURE 13-2. Transvaginal sonogram demonstrating an ectopic pregnancy.

Note the large amount of free fluid (FF) in the pelvis. No intrauterine pregnancy was seen. A large complex echogenic mass (EM) was seen in the left adnexa, consistent with an ectopic pregnancy. A simple cyst (SC) is also seen in the right adnexa. The area within the uterus represents a small fibroid.

Rhesus (Rh) Isoimmunization

DEFINITION

Immunologic disorder that affects Rh-negative mothers of Rh-positive fetuses.

PATHOPHYSIOLOGY

- Occurs with maternal exposure to fetal Rh-positive blood cells in the setting of transplacental hemorrhage (typically occurs during delivery, may also occur with abortions and trauma).
- Initial exposure leads to primary sensitization with production of immunoglobulin M antibodies.
- In subsequent pregnancies, maternal immunoglobulin G antibody crosses placenta and attacks Rh-positive fetal RBCs.

PREVENTION

- Prevention of Rh isoimmunization is by administering Rh_o immune globulin (RhoGAM) to mothers during time of potential antigen exposure (amniocentesis, threatened abortion, trauma, delivery, etc.).
- RhoGAM is also administered prophylactically to all Rh-negative mothers ~28 weeks' gestation.

> ▶ ABORTION

Threatened Abortion

DEFINITION

Abdominal pain or vaginal bleeding in first 20 weeks' gestation.

SIGNS AND SYMPTOMS

- Closed cervix.
- No passage of fetal tissue by history or exam.

DIAGNOSIS

β-hCG and ultrasound to confirm IUP and rule out EP.

TREATMENT

- Bed rest for 24 hours.
- Avoid intercourse, tampons, and douching until bleeding stops.
- Arrange outpatient follow-up for repeat β-hCG/sonogram in 24 to 48 hours.
- Rh isoimmunization prophylaxis as needed.

First-trimester vaginal bleeding occurs in approximately 40% of first-time pregnancies, one half of which will eventually result in miscarriage.

Inevitable Abortion

DEFINITION

Vaginal bleeding with open cervical os but no passage of fetal products.

TREATMENT

- Dilation and curettage (D&C, evacuation of pregnancy).
- Rh isoimmunization as needed.

Incomplete Abortion

DEFINITION

Incomplete passage of fetal products, usually between 6 and 15 weeks of gestation.

SIGNS AND SYMPTOMS

- Open cervical os
- Pain and bleeding

TREATMENT

- D&C.
- Rh isoimmunization prophylaxis as needed.

Complete Abortion

DEFINITION

Complete passage of fetal products and placenta.

SIGNS AND SYMPTOMS

- Closed cervical os.
- Uterus contracts.
- Pregnancy-induced changes begin to resolve.

TREATMENT

- Supportive management with outpatient follow-up for ultrasound-confirmed complete abortion.
- D&C if unsure all products have been passed.
- Rh isoimmunization prophylaxis as needed.

Septic Abortion

DEFINITION

Uterine infection during any stage of an abortion.

CAUSES

Bowel and genital flora are most often implicated.

SIGNS AND SYMPTOMS

- Fever
- Bleeding
- Cramping pain
- Purulent discharge from cervix
- Boggy, tender, enlarged uterus

TREATMENT

- Prompt evacuation.
- Broad-spectrum antibiotics.
- Rh isoimmunization prophylaxis as needed.

Missed Abortion

DEFINITION

Uterine retention of dead fetal products for several weeks after abortion.

SIGNS AND SYMPTOMS

May progress to spontaneous abortion with expulsion of products.

TREATMENT

- D&C.
- Rh isoimmunization prophylaxis as needed.
- Complications may occur secondary to infection or coagulopathy.

► **THIRD-TRIMESTER BLEEDING (28+ WEEKS)**

Abruptio Placentae

DEFINITION

Premature separation of normally implanted placenta from uterine wall (usually third trimester).

RISK FACTORS

- Previous abruptio placentae
- Abdominal trauma
- Hypertension
- Cocaine use
- Smoking
- Multiparity
- Advanced maternal age

Hypertension is the most common risk factor for abruptio placentae.

SIGNS AND SYMPTOMS

- Vaginal bleeding with dark clots.
- Abdominal pain.
- Uterine pain/irritability.
- Uterus may be soft or very hard.

DIAGNOSIS

- Ultrasound is not useful in the diagnosis of abruptio placentae.

TREATMENT

- Emergent obstetrical consultation for maternal/fetal monitoring and possible delivery.
- Rh isoimmunization prophylaxis as needed.

COMPLICATIONS

- Fetal distress
- Diffuse intravascular coagulation
- Amniotic fluid embolism
- Maternal/fetal death

Placenta Previa

DEFINITION

Implantation of placenta overlying internal cervical os.

RISK FACTORS

- Previous placenta previa
- Prior C-section
- Multiple gestations
- Multiple induced abortions
- Advanced maternal age

SIGNS AND SYMPTOMS

- Painless vaginal bleeding
- Soft, nontender uterus

DIAGNOSIS

Ultrasonography will confirm placental location.

TREATMENT

- Pelvic/cervical exam is *not* performed in the emergency department (ED), as this can precipitate massive bleeding. It is done in the operating room (OR) where emergency C-section can be performed if massive bleeding does occur.
- Emergent obstetrical consultation for maternal/fetal monitoring and possible delivery.
- Rh isoimmunization prophylaxis as needed.

▶ **PREGNANCY-INDUCED HYPERTENSION**

Defined as BP > 140/90, increase in systolic BP > 20, or increase in diastolic BP > 10.

Eclampsia can occur up to 10 days postpartum.

HELLP *syndrome complicates ~10% of preeclampsia:*
Hemolysis,
Elevated **L**iver enzymes,
Low **P**latelets

Preeclampsia and Eclampsia

DEFINITIONS

- Preeclampsia is a syndrome of hypertension, proteinuria, and generalized edema that occurs in weeks 20 to 24 of pregnancy.
- Eclampsia is preeclampsia plus seizures.

RISK FACTORS

- Primigravida.
- Very young or advanced maternal age.
- History of hypertension or kidney disease.
- Diabetes mellitus.
- Hydatidiform mole.
- Multiple gestations.
- Family history of pregnancy-induced hypertension.

SIGNS AND SYMPTOMS

- Weight gain > 5 lbs/wk
- Headache, visual disturbances
- Peripheral edema
- Pulmonary edema
- Oliguria

DIAGNOSIS

Diagnostic Triad
- Hypertension as defined above
- Proteinuria > 300 mg/24 hr
- Generalized edema

Laboratory Findings
- Elevated uric acid, serum creatinine, liver function tests, and bilirubin.
- Thrombocytopenia.

TREATMENT

- Absolute bed rest.
- Left lateral decubitus position to increase blood flow to uterus.
- Hydralazine for BP control (labetalol for refractory cases).
- Magnesium sulfate for seizure and secondary BP control (phenytoin or diazepam for refractory cases). Watch for signs of magnesium toxicity.
- Maintain urine output at 30 cc/hr, as many of the drugs used are cleared through the kidney.
- Definitive treatment is delivery of the fetus.

> Rapid BP reduction may decrease uterine blood flow and lead to fetal distress.

> *Signs of magnesium toxicity:*
> - **Hyporeflexia or loss of deep tendon reflexes**
> - **Respiratory depression**
> - **Bradydysrhythmias**

▶ HYDATIDIFORM MOLE

DEFINITION

- "Molar pregnancy" secondary to overproduction of chorionic villi.
- Partial mole: Triploid (two sets paternal, one maternal), presence of fetal parts, higher tendency to progress to choriocarcinoma.
- Complete mole: Diploid (two sets maternal), absence of fetal parts.

RISK FACTORS

- Previous history of molar pregnancy.
- Very young or advanced maternal age.

SIGNS AND SYMPTOMS

- Severe nausea and vomiting.
- Uterus larger than expected for dates.
- Passage of grapelike clusters of vesicles through vagina.
- Intermittent vaginal bleeding during early pregnancy.
- Preeclampsia before 20 weeks' gestation.

DIAGNOSIS

- Anemia.
- β-hCG higher than expected.
- Snowstorm appearance on ultrasound.

> A 17-year-old girl who is 11 weeks pregnant by dates presents with severe vomiting. Her BP is 190/120. Her fundal height is at the umbilicus. Sonogram reveals echogenic material in the uterus, but no gestational sac or fetal heart beat is present. *Think: Hydatidiform mole.*

- D&C.
- Follow-up to monitor for choriocarcinoma.

▶ **NORMAL LABOR AND DELIVERY**

- Progressive cervical effacement and/or dilatation in the presence of uterine contractions occurring < 5 minutes apart and lasting 30 to 60 seconds at a time (Table 13-1).
- "False" labor—during last 4 to 8 weeks of pregnancy, contractions (Braxton Hicks) occur in the absence of cervical dilatation or effacement.

First Stage

- Starts with onset of labor, ends with complete dilatation (10 cm) of cervix.
- "Latent" phase—effacement with minimal dilatation.
- "Active" phase—accelerated rate of cervical dilatation.

Second Stage

- Starts with complete cervical dilatation, ends with delivery of baby.
- Six cardinal movements of labor: Descent, flexion, internal rotation, extension, external rotation, expulsion.

Third Stage

- Starts with delivery of baby, ends with delivery of placenta.
- Assess external genitalia for signs of perineal/rectal tears.

Fourth Stage

- Starts with delivery of placenta, ends with stabilization of mother.
- Monitor for hemodynamic instability and postpartum hemorrhage.

T A B L E   1 3 - 1 .   Duration of the Stages of Labor

	PRIMIPAROUS	**MULTIPAROUS**
First stage	6–18 hours	2–10 hours
Cervical dilatation (active phase)	1.0 cm/hr	1.2 cm/hr
Second stage	0.5–3.0 hours	5–20 minutes
Third stage	0–30 minutes	0–30 minutes
Fourth stage	~1 hour	~1 hour

Premature Rupture of Membranes (PROM)

DEFINITION

Rupture of fetal membranes before labor begins.

SIGNS AND SYMPTOMS

Leakage of amniotic fluid prior to onset of labor at any stage of gestation.

DIAGNOSIS

- Pooling of amniotic fluid in vaginal fornix.
- Nitrazine paper test: Turns blue in presence of amniotic fluid (false positive seen with use of lubricant).
- Ferning pattern on microscopic paper (false negative seen with blood) (Figure 13-3).

TREATMENT

- If fetus > 37 weeks, delivery within 24 hours.
- If fetus < 37 weeks, timing of delivery is weighed against risks of fetal immaturity.

Patients with PROM are at risk for chorioamnionitis.

Preterm Labor

DEFINITION

Defined as labor occurring after 20 weeks' and before 37 weeks' gestation.
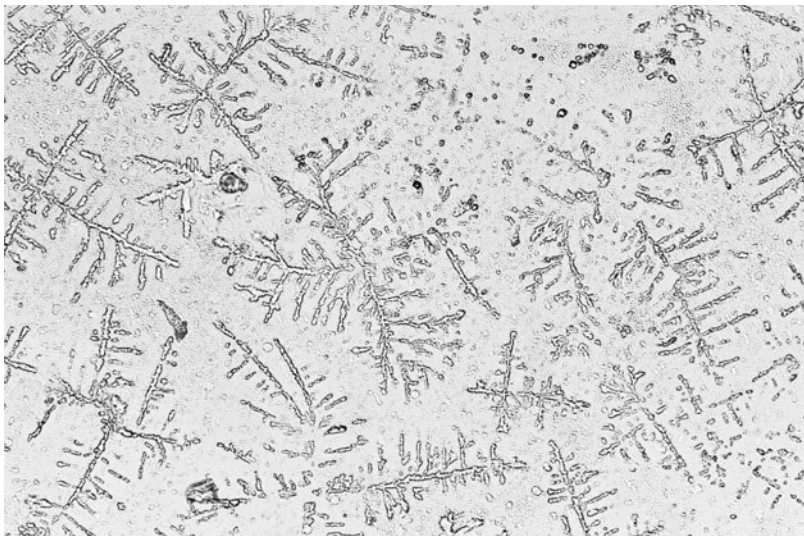
FIGURE 13-3. "Ferning" pattern of amniotic fluid when exposed to air. Can be useful for diagnosing PROM.

(Reproduced, with permission, from Knoop K, Stack LB, Storrow AB. *Atlas of Emergency Medicine.* New York: McGraw-Hill, 1997: 265.)

HIGH-YIELD FACTS

Obstetric Emergencies
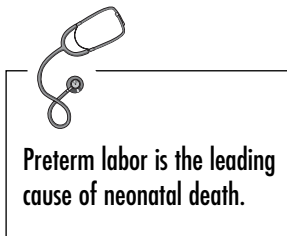
DIAGNOSIS

Diagnosed by regular uterine contractions in the presence of cervical dilatation/effacement.

RISK FACTORS

- PROM
- Abruptio placentae
- Multiple gestation
- Drug use
- Polyhydramnios
- Incompetent cervix
- Infection (including STDs)

TREATMENT

- Hydration and bed rest (successful in ~20% cases).
- Glucocorticoids (Celestone) to accelerate fetal lung maturity.
- Tocolysis with magnesium sulfate, beta blockers (terbutaline, ritodrine), and prostaglandin synthetase inhibitors (indomethacin).
- Contraindications to tocolysis:
  - Severe preeclampsia.
  - Severe bleeding from placenta previa or abruptio placentae.
  - Chorioamnionitis.

Strategies for Assessing Potential Fetal Distress

1. Assess for short-term (beat-to-beat) and long-term variability of fetal heart rate (normal).
2. Assess for response of fetal heart rate to uterine contractions:
   - Accelerations are a normal response to uterine contractions.
   - Early decelerations are usually related to head compression (normal).
   - Variable decelerations may be related to intermittent cord compression.
   - Late decelerations are often related to uteroplacental insufficiency.
3. Fetal tachycardia may be a sign of maternal fever or intrauterine infections.
4. Presence of "heavy" meconium in amniotic fluid increases risk of aspiration.

▶ POSTPARTUM COMPLICATIONS

Postpartum Hemorrhage

DEFINITION

- Classified as early (within 24 hours of delivery) or late (up to 1 to 2 weeks postpartum).
- > 500 mL blood loss (vaginal delivery) or 1,000 mL (cesarean delivery).

CAUSES OF EARLY POSTPARTUM HEMORRHAGE

- **Uterine atony:** Most common cause (overdistended uterus, prolonged labor, oxytocin use).

- **Genital tract trauma:** Vaginal or rectal lacerations.
- **Retained products of conception:** Acts as a wedge preventing uterine contractions, leading to increased bleeding.
- **Uterine inversion:** Uterus turns inside out, leading to vasodilatation and increased bleeding.

CAUSES OF LATE POSTPARTUM HEMORRHAGE

- Endometritis.
- Retained products of conception.

SIGNS AND SYMPTOMS

- Vaginal bleeding
- Soft, atonic uterus

TREATMENT

- Repair any lacerations.
- Manually remove placenta if it does not pass.
- Bimanual massage and/or intravenous oxytocin to stimulate uterine contractions.
- Metyhylergonovine for refractory cases.

Endometritis

DEFINITION

Infection of the endometrium.

CAUSES

Majority of infections are caused by normal vaginal/cervical flora (enterococci, streptococci, anaerobes).

SIGNS AND SYMPTOMS

- Fever
- Tender, swollen uterus
- Foul-smelling lochia

DIFFERENTIAL

In patients who do not respond to antibiotic therapy, consider:
- Pelvic abscess (requires surgical drainage)

or
- Pelvic thrombophlebitis (requires anticoagulation)

TREATMENT

- Broad-spectrum antibiotics
- Hospitalization

HIGH-YIELD FACTS

Obstetric Emergencies

Musculoskeletal Emergencies

DEFINITIONS

Sprain
- A partial or complete rupture of the fibers of a ligament.
- First degree: Joint is stable; integrity of the ligament is maintained with a few fibers torn.
- Second degree: Joint stability is maintained but ligamentous function is decreased.
- Third degree: Joint instability with complete tearing of ligament.

Strain
- A partial or complete rupture of the fibers of the muscle–tendon junction.
- First degree: Mild.
- Second degree: Moderate, associated with a weakened muscle.
- Third degree: Complete tear of the muscle–tendon junction with severe pain and inability to contract the involved muscle.

ETIOLOGY

Trauma: Indirect or direct, causing the ligaments of any joint to stretch beyond their elastic limit.

SIGNS AND SYMPTOMS

- Pain and swelling over area involved.
- Patient may have experienced a snap or pop at time of injury.

TREATMENT

For First- and Most Second-Degree Sprains
- Rest, ice, compression, and elevation ("RICE" therapy) for 24 to 36 hours.
- Weight bearing as tolerated in the case of lower extremity sprain.
- Pressure dressing can also be applied.
- Crutches as needed.
- Orthopedic follow-up is not always necessary.
- Analgesia (e.g., ibuprofen—to be taken with food to decrease gastric irritation).

For Third-Degree Sprains
- Splint that prevents range of motion (ROM) of joint.
- Crutches: Provide non-weight-bearing status in lower extremity injuries.
- RICE and pain medications.
- Orthopedic follow-up for appropriate treatment, which usually includes operative repair in the young.

RICE *therapy:*
Rest
Ice
Compression (splint)
Elevation

Nontraumatic Conditions

> ▶ INFECTIONS

Herpetic Whitlow

DEFINITION

Painful infection of the terminal phalanx.

ETIOLOGY

- Initiated by viral inoculation.
- Sixty percent of cases are herpes simplex virus type 1 (HSV-1); 40% are HSV-2.
- Incubation period is 2 to 20 days.

SIGNS AND SYMPTOMS

- Prodrome of fever and malaise.
- Initial pain and burning or tingling of the infected digit, followed by erythema, edema, and the development of 1- to 3-mm grouped vesicles on an erythematous base over the next 7 to 10 days.
- Complete resolution occurs over subsequent 5 to 7 days.
- Recurrences observed in 20 to 50% of cases are usually milder and shorter in duration.

DIAGNOSIS

- Primarily clinical diagnosis
- Laboratory tests:
  - Tzanck test.
  - Viral culture of aspirated vesicle (requires 24 to 48 hours).
  - Serum antibody titers.

TREATMENT

- Self-limited disease.
- Symptomatic relief.
- Acyclovir may be beneficial and may prevent recurrrence.
- Use antibiotic in bacterial superinfection.
- Deep surgical incision is contraindicated.

Felon

DEFINITION

Infection of the pulp space of any of the distal phalanges (Figure 14-1).

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.
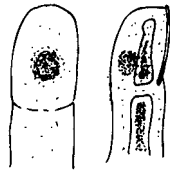
FIGURE 14-1. Felon (infection of pulp space).

(Reproduced, with permission, from DeGowin RL, Brown DD. *DeGowin's Diagnostic Examination.* 7th ed. New York: McGraw-Hill, 2000: 703.)

TREATMENT

- Using a digital block, perform incision and drainage with longitudinal incision over the area of greatest induration but not over the flexor crease of the distal interphalangeal (DIP).
- A drain may be placed and the wound checked in 2 days.
- Seven- to 10-day course of antibiotics: Usually first-generation cephalosporin or anti-*Staphylococcus* penicillin.

Paronychia

DEFINITION

Infection of the lateral nail fold (Figure 14-2).

ETIOLOGY

Caused by minor trauma such as nail biting or manicure.

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 incise and drain at area of most fluctuance using a digital block.
- Pus below the nailbed may require partial or total removal of the nail.
- Advise patient to do warm soaks and return for wound check in 2 days.
- Antibiotics are usually not necessary unless area is cellulitic.
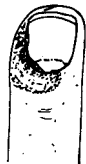
FIGURE 14-2. Paronychia.

(Reproduced, with permission, from DeGowin RL, Brown DD. *DeGowin's Diagnostic Examination*, 7th ed. New York: McGraw-Hill, 2000: 703.)

Flexor Tenosynovitis

DEFINITION

- This is a surgical emergency requiring prompt identification.
- Infection of the flexor tendon and sheath is caused by penetrating trauma and dirty wounds (i.e., dog bite).
- Infection spreads along the tendon sheath, allowing involvement of other digits and even the entire hand, causing significant disability.

ETIOLOGY

- Polymicrobial.
- *Staphylococcus* most common.
- *Neisseria gonorrhoeae* with history of sexually transmitted disease.

SIGNS AND SYMPTOMS

Kanavel criteria:
- Digit is flexed at rest.
- Passive extension produces pain.
- Symmetrical swelling of finger.
- Tenderness over flexor tendon sheath.

TREATMENT

- Immobilize and elevate hand.
- Immediate consultation with hand surgeon.
- Intravenous (IV) antibiotics.

Kanavel criteria for tenosynovitis:
STEP
Symmetrical swelling of finger
Tenderness over flexor tendon sheath
Extension (passive) of digit is painful.
Posture of digit at rest is flexed.

Cellulitis

DEFINITION

A local erythematous inflammatory reaction of the subcutaneous tissue following a cutaneous breach, which leads to infection.

ETIOLOGY

- *Streptococcus pyogenes* (most common).
- *Staphylococcus*.
- *Haemophilus influenzae* in unimmunized individuals.
- Enterobacteriaceae in diabetics.

SIGNS AND SYMPTOMS

- Localized tenderness and swelling.
- Warmth.
- Erythema.
- If immunocompromised, may see fever and leukocytosis.

TREATMENT

- For uncomplicated cellulitis in healthy individuals, cephalexin or dicloxacillin 500 mg qid × 10 days or azithromycin 500 mg × 1, then 250 mg qd × 4 days.
- IV antibiotics for head and face involvement and the immunocompromised: Cefazolin 1 g IV qid and nafcillin or oxacillin 2 g IV q4h. Ceftriaxone or imipenem for severe cases.

Gas Gangrene

ETIOLOGY

A life- and limb-threatening soft-tissue infection caused by one of the spore-forming *Clostridium* spp., resulting in myonecrosis, gas production, and sepsis.

ORGANISM

- *Clostridium perfringens* (80 to 90%).
- *Clostridium septicum*.
- These are spore-forming G+ anaerobic bacilli found in soil, gastrointestinal tract, and female genitourinary tract.

PATHOPHYSIOLOGY

- Dirty wounds with jagged edges become infected with the ubiquitous organism that produces exotoxins.
- These cause systemic toxicity and cellular destruction.
- Bacteremia is rare.

SIGNS AND SYMPTOMS

- Three-day incubation period.
- Patient complains of **pain out of proportion** to physical findings.
- Limb feels heavy.
- Skin becomes discolored (brown).
- Crepitance.
- Fever.
- Tachycardia.
- Diabetics are particularly susceptible due to immunocompromise (impaired white blood cell [WBC] chemotaxis), peripheral neuropathy (delaying detection of small wounds), and impaired peripheral perfusion.

DIAGNOSIS

- Metabolic acidosis.
- Leukocytosis.
- Myoglobinuria, myoglobinemia.
- Coagulopathy.
- Elevated creatine phosphokinase (CPK).
- Gas in soft-tissue planes on radiograph.

TREATMENT

- Fluid resuscitation, may require transfusion.
- Monitor intake and output.
- Antibiotics: Penicillin G or clindamycin; metronidazole or chloramphenicol if penicillin allergic.
- Surgical debridement is definitive treatment.
- Hyperbaric O_2 has been shown to help.

Septic Arthritis

DEFINITION

Infection of joint space.

> *Features of gas gangrene:*
> **CLOSTRIDiuM P**
> *C*lostridium spp., crepitance of skin:
> **L**eukocytosis
> c**O**agulopathy
> **S**pore-forming G+ anaerobic bacilli, found in soil
> **T**hrombocytopenia, tachycardia
> **R**adiograph shows gas in soft tissue.
> **I**rritability
> **D**iscoloration of skin
> **M**yoglobinemia
> **P**ainful

The most common organism to cause septic arthritis overall across all age groups is *S. aureus*.

The most common joint involved is the knee, followed by hip, shoulder, and wrist.

ETIOLOGY

- Neonates: *Staphylococcus aureus,* group B strep.
- Children and adolescents: *S. aureus, Haemophilus.*
- Adults < 50: *N. gonorrhea, S. aureus.*
- Adults > 50: *S. aureus, E. coli.*
- IV drug users: *Pseudomonas aeruginosa.*

EPIDEMIOLOGY

- Two peaks: In children and the elderly.
- Males affected twice as often.

RISK FACTORS

- Rheumatoid arthritis (RA).
- Osteoarthritis (OA).
- Risky sexual behavior *(N. gonorrhoeae).*
- Immunocompromised states: Alcoholism, liver or kidney disease, diabetes, cancer.

SIGNS AND SYMPTOMS

- Fever, chills.
- Acute joint pain.
- Joint stiffness.
- Recent urethritis, salpingitis, or hemorrhagic vesicular skin lesions *(N. gonorrhoeae).*
- Maculopapular or vesicular rash *(N. gonorrhoeae).*
- Tenosynovitis → migratory polyarthritis → oligoarthritis *(N. gonorrhoeae).*
- Pain with passive motion of the involved joint.
- Joint is warm, tender, and swollen, with evidence of effusion.

DIAGNOSIS

- Via arthrocentesis (see Procedures chapter).
- A WBC count of 50,000 in the joint fluid with 75% granulocytosis is diagnostic.
- Erythrocyte sedimentation rate (ESR) and C-reactive protein often elevated, blood cultures usually positive.
- Plain radiographs of joint should be obtained to look for underlying osteomyelitis or joint disease.
- If *N. gonorrhoeae* is suspected, culture the cervix, anus, and eye.

TREATMENT

- IV antibiotics.
- Splinting of joint.
- Analgesia.
- Surgery is recommended in children and for joints with loculated effusions.
- Shoulder and hip septic arthritis are drained openly in the operating room due to risk of avascular necrosis (AVN).

Osteomyelitis

DEFINITION

Inflammation or infection of bone.

ETIOLOGY

- *S. aureus*
- *Streptococcus* species
- *Pseudomonas aeruginosa* (especially in IV drug users and foot puncture wounds)

EPIDEMIOLOGY

More common in males.

RISK FACTORS

- Trauma (including surgery)
- Immunocompromise (diabetes, sickle cell disease, alcoholism, etc.)
- Soft-tissue infection

SIGNS AND SYMPTOMS

- Pain, swelling, and warmth of bone or joint.
- Decreased ROM.
- Fever.

DIAGNOSIS

- Bone scan will detect osteomyelitis within 48 hours.
- Radiograph will demonstrate periosteal elevation within 10 days. (See Figure 14-3.)

A 42-year-old male athlete steps on a nail through his sneaker. Two weeks later, he presents to the emergency department (ED) with pain and swelling of his left foot. *Think: Osteomyelitis due to* Pseudomonas.

HIGH-YIELD FACTS

Musculoskeletal Emergencies
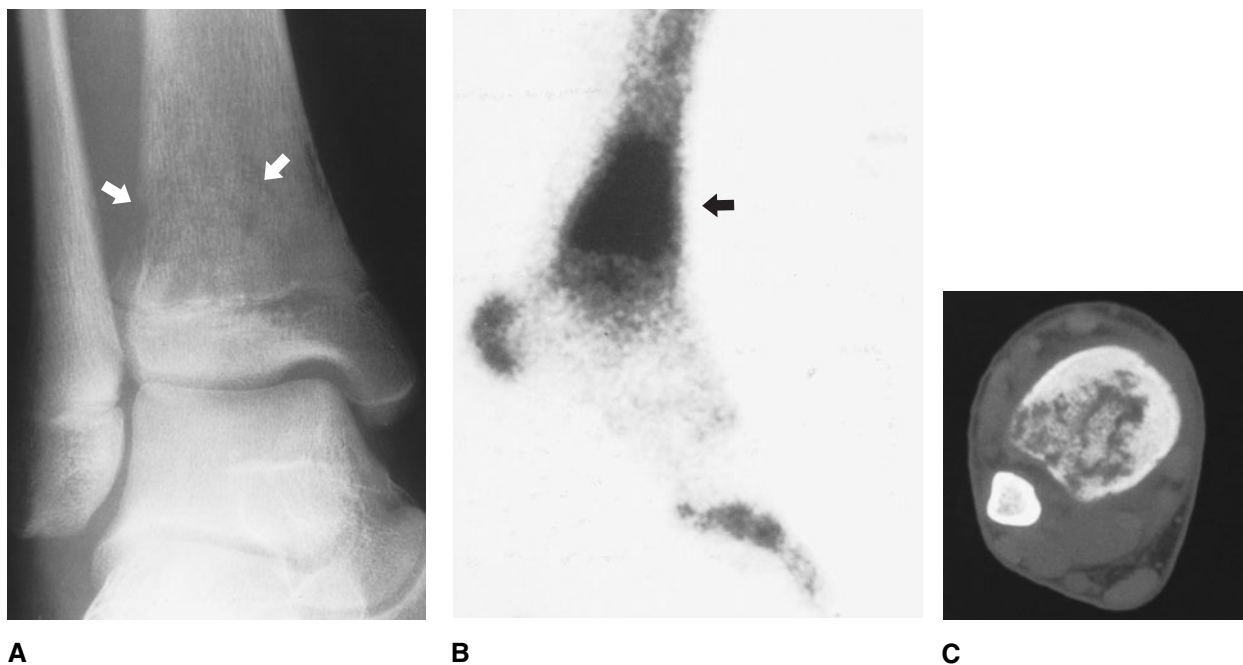
FIGURE 14-3. **Osteomyelitis of the distal tibia.**

A. Plain film of tibia demonstrating slight lucency at arrow. **B.** Bone scan of the same tibia demonstrating increased uptake. **C.** Computed tomography scan (cross-section) demonstrating mottled appearance of the bony cortex. (Reproduced, with permission, from Schwartz DT, Reisdorff EJ. *Emergency Radiology.* New York: McGraw-Hill, 2000: 23.)

- Blood cultures will demonstrate causative organism in 50% of cases.
- ESR and C-reactive protein support presence of inflammation.

TREATMENT

- Antibiotics for 6 weeks (some bugs may need shorter courses).
- Splinting of joint.

► **NONINFECTIOUS CONDITIONS**

Carpal Tunnel Syndrome

DEFINITION

Compression of the median nerve, resulting in pain along the distribution of the nerve.

ETIOLOGY

- Tumor (fibroma, lipoma).
- Ganglion cyst.
- Tenosynovitis of flexor tendons due to RA or trauma.
- Edema due to pregnancy or 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).

DIAGNOSIS

- Tinel's sign: Tapping over median nerve at wrist produces pain and paresthesia. (See Figure 14-4.)
- Phalen's test: One minute of maximal palmar flexion produces pain and paresthesia. (See Figure 14-4.)
- Consider ESR, thyroid function tests, serum glucose, and uric acid level to look for underlying cause.

TREATMENT

- Treat underlying condition.
- Rest and splint.
- NSAIDs for analgesia.
- Surgery for crippling pain, thenar atrophy, and failure of nonoperative management.

It is important to differentiate osteomyelitis from septic joint, which is an emergency.

Carpal tunnel syndrome is the most common entrapment neuropathy.

A 37-year-old woman 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*

Twenty to 50% of the population normally have a positive Phalen's test and Tinel's sign.

Median Nerve
Flexor Digitorum Sublimus
Flexor Carpi Radialis
Flexor Carpi Ulnaris
Flexor Retinaculum

1. Carpal Tunnel   2. Sensory Spread of Median Nerve   3. Phalen's Test

FIGURE 14-4. **Carpal tunnel syndrome.**

1. The flexor retinaculum in the wrist compresses the median nerve to produce hyperesthesia in the radial 3.5 digits. **2.** Tinel's sign: Percussion on the radial side of the palmaris longus tendon produces tingling in the 3.5 digit region. **3.** Phalen's test: Hyperflexion of the wrist for 60 seconds may produce pain in the median nerve distribution, which is relieved by extension of the wrist. (Reproduced, with permission, from DeGowin RL, Brown DD. *DeGowin's Diagnostic Examination*, 7th ed. New York: McGraw-Hill, 2000: 720.)

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.

Differential diagnosis includes bone tumor, arthritis, and intraosseous ganglion.

F I G U R E   1 4 - 5 .   Trigger finger.

Usually involves third or fourth digit. Flexion is normal, but extension involves a painful "snap." (Reproduced, with permission, from DeGowin RL, Brown DD. *DeGowin's Diagnostic Examination*, 7th ed. New York: McGraw-Hill, 2000: 701.)

Trigger Finger

DEFINITION

Stenosis of tendon sheath flexor digitorum leading to nodule formation within the sheath.

RISK FACTORS

- RA
- Middle-aged women
- Congenital

SIGNS AND SYMPTOMS

Snapping sensation or click when flexing and extending the digit (Figure 14-5).

TREATMENT

- Splinting of MCP joint in extension.
- Injection of corticosteroid into tendon sheath.
- Surgical repair if above fail.

See Table 14-1 for more upper extremity problems.

▶ **LOW BACK PAIN**

Leading Causes of Lower Back Pain

- **F**racture
- **A**bdominal aortic aneurysm
- **C**auda equina syndrome
- **T**umor (cord compression)
- **O**ther (OA, severe musculoskeletal pain, other neurological syndromes)
- **I**nfection (e.g., epidural abscess)
- **D**isk herniation/rupture

Emergent causes of low back pain: **FACTOID**

INJURY	DESCRIPTION	TREATMENT

Boxer's fracture	Fracture of neck of fifth metacarpal sustained in a closed-fist injury	■ Thumb spica cast for 3–6 weeks for partial tears
		■ Surgical repair for:
		  ■ Any rotational deformity
		  ■ Angulation of fourth > 20°/fifth metacarpal > 40°
		  ■ Angulation of second/third metacarpal > 10°–15°

Bennett's fracture	Fracture–dislocation of base of thumb	■ Initially immobilization in thumb spica cast ■ Definitive treatment is with surgical fixation
Rolando's fracture	Comminuted fracture of the base of the thumb	■ Initially immobilization in thumb spica cast ■ Definitive treatment is with surgical fixation
Scaphoid fracture	■ Most commonly caused by fall on outstretched hand ■ Snuffbox 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

Nightstick fracture	■ Isolated fracture of the ulnar shaft	■ Long arm cast for 3–6 weeks
		■ Surgical repair for:
		  ■ Angulation > 10°
		  ■ Displacement > 50%

Lunate dislocation	■ Often missed ■ Fall on outstretched hand ■ Pain and swelling anteriorly over wrist	■ Surgical repair

Lumbar Disk Herniation

DEFINITION

- Disk herniation is a common cause of chronic lower back pain.
- L4–5 and L5–S1 are the most common sites affected.
- Herniation occurs when the nucleus pulposus prolapses posteriorly through the annulus fibrosis.
- More common in men.

SIGNS AND SYMPTOMS

- Limited spinal flexion.
- Pain and paresthesia with a dermatomal distribution.
- Specific signs depend on nerve root involved:
  - L4: Decreased knee jerk, weakness of anterior tibialis.
  - L5: Weakness of extensor hallucis longus, decreased sensation over lateral aspect of calf and first web space.
  - S1: Decreased ankle jerk, decreased plantar flexion, decreased sensation over lateral aspect of foot.

OA can lead to osteophytes and hypertrophy of spinal facets, which compress nerve roots.

The nucleus pulposus is a thick gel. Herniation of the nucleus pulposus is like toothpaste being squeezed out of the tube.

Spondylolysis

DEFINITION

Defect or fracture of the pars interarticularis in the lumbar spine.

ETIOLOGY

Possible etiology is a stress fracture that occurs during childhood that does not heal completely.

DIAGNOSIS

- Oblique view on radiograph will show a characteristic "Scotty dog with a collar" (Figure 14-6).
- Most often occurs at L4–5, L5–S1.

TREATMENT

Rest, nonsteroidal anti-inflammatory drugs (NSAIDs), possible bracing.
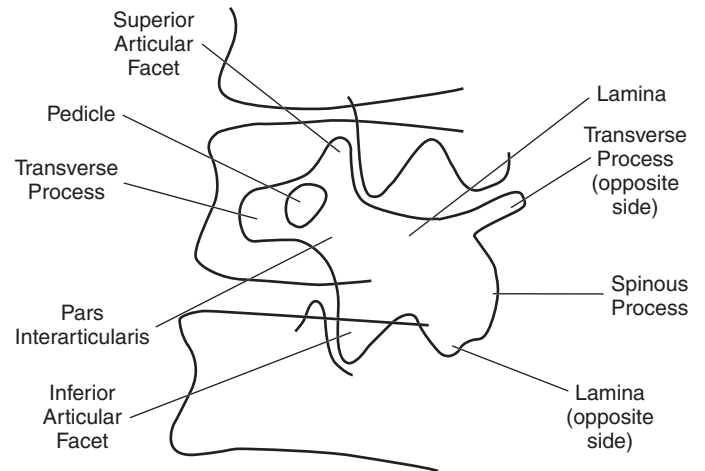
FIGURE 14-6. Normal lumbosacral spine with intact "Scotty dog."

(Reproduced, with permission, from Schwartz DT, Reisdorff EJ. *Emergency Radiology.* New York: McGraw-Hill, 2000: 344.)

Spondylolisthesis

DEFINITION

- Forward displacement of one vertebra over another.
- Usually occurs when spondylolysis is bilateral and becomes unstable.

DIAGNOSIS

- Oblique view on radiograph will show forward displacement of one vertebra with fracture at bilateral pars interarticularis or "Scotty dog decapitated."
- Felt as a stepoff when palpating the lumbar spine.
- Most common at L4–5, L5–S1.

TREATMENT

Orthopedic consultation, bracing.

Vertebral Compression Fracture

- Most common manifestation of osteoporosis.
- Also seen in patients on long-term steroids and in patients with lytic bony metastases.
- The thoracic spine is the most common site affected.

SIGNS AND SYMPTOMS

- Height loss.
- Sudden back pain after mild trauma.
- Local radiation of pain—the extremities are rarely affected (unlike a herniated disk).

DIAGNOSIS

Plain radiographs of lumbosacral spine will not show compression fracture until there is loss of 25 to 30% of bone height.

TREATMENT

- Symptomatic relief with NSAIDs.
- Treatment of osteoporosis prevents compression fractures:
  - Recommend weight-bearing exercises.
  - Estrogen replacement therapy.
  - Calcium supplementation.
  - Calcitonin (intramuscular) inhibits bone resorption.
  - Bisphosphonates increase bone mass by inhibiting osteoclast activity.

Bisphosphonates such as alendronate (Fosamax) irritate gastric mucosa, so advise patients to eat beforehand and stay upright for 30 minutes after taking it.

Epidural Abscess

- Spinal abscesses are most commonly found in the immunosuppressed, IV drug users, and the elderly.
- An abscess can form anywhere along the spinal cord, and as it expands, it compresses against the spinal cord and occludes the vasculature.

ETIOLOGY

- The infection is generally spread from the skin or other tissue.
- *Staphylococcus aureus,* gram-negative bacilli, and tuberculosis bacillus are the leading organisms involved.

SIGNS AND SYMPTOMS

- Triad of pain, fever, and progressive weakness.
- The pain develops over the course of a week or two, and the fever is often accompanied by an elevated WBC count.

DIAGNOSIS AND TREATMENT

- Magnetic resonance imaging (MRI) can localize the lesion. Lumbar puncture is not required unless meningitis is suspected.
- Emergent decompressive laminectomy can prevent permanent sequelae. This should be followed up with long-term antibiotics.

> Spinal cord compression is an oncologic emergency. Missed diagnosis can lead to permanent paralysis.

Spinal Metastasis

- Metastatic lesions invade the spinal bone marrow, leading to compression of the spinal cord.
- Typically involves the thoracic spine.
- The most common primary tumors involved include breast, lung, prostate, kidney, lymphoma, and multiple myeloma.

SIGNS AND SYMPTOMS

- Pain is the primary symptom.
- Weakness and sensory loss follow.
- Upper motor neuron signs are seen:
  - Hyperreflexia
  - Upward Babinski sign

DIAGNOSIS

MRI is the preferred imaging technique.

TREATMENT

- Glucocorticoids are used to reduce inflammation and edema.
- Radiation therapy should be started as soon as possible.
- Surgery is indicated only if radiation fails to improve the symptoms.

> Any cancer patient who develops back pain should be investigated for spinal metastases.

Cauda Equina Syndrome

DEFINITION

Compression of the lumbar and sacral nerve roots that comprise the cauda equina.

ETIOLOGY

- Tumor.
- Midline disk herniations (rare).
- Congenital narrowing of the lumbar canal.

> **Saddle anesthesia:** Loss of sensation over the buttocks, perineum, and thighs. Frequently seen in cauda equina syndrome.

- Typically present with *saddle anesthesia*.
- Sensory and motor disturbances of the lower extremities can occur, as well as urinary and bladder incontinence.

TREATMENT

- Bed rest on a hard surface and analgesia.
- Neurosurgical evaluation for potential laminectomy.

► SYSTEMIC PROBLEMS

Osteoarthritis (OA)

DEFINITION

- OA is the result of mechanical and biological factors that destabilize articular cartilage and subchondral bone.
- There is softening, ulceration, and loss of articular cartilage, eburnation (sclerosis) of subchondral bone, osteophytes, and subchondral cysts.
- Cause is unclear but is probably multifactorial.

EPIDEMIOLOGY

- Most common form of arthritis.
- Affects males and females equally.
- Peak age is 45 to 55, but after 55 more common in women.
- Fifty percent of people over 65 have radiographic changes in the knees.
- Obesity correlates with OA of the knees.
- Weight-bearing joints, including the lumbosacral spine, hips, knees, and feet, are most commonly involved.
- Cervical spine and proximal interphalangeal (PIP) and DIP joints are frequently involved.
- Elbows and shoulders are effected only when involved in trauma or overuse.

SIGNS AND SYMPTOMS

- Pain and stiffness in and around the joint.
- Limitation of function.
- Insidious onset.
- Worse with activity, relieved by rest.
- Worse in rainy, damp, and cool weather.
- Gel phenomenon (stiffness after periods of rest) that resolves within several minutes.
- Knee instability and buckling.
- Hip-gait disturbance and pain in groin or radiation to anterior thigh and knee.
- Hands: PIP (Bouchard's nodes) and DIP (Heberden's nodes) (Figure 14-7).
- Facet joints of cervical spine and lumbosacral spine cause neck and low back pain.
- Symptoms are localized, and limitation of function is secondary to osteophytes, cartilage loss, and muscle spasm.
- Locking of joint is secondary to loose bodies.
- Crepitation is present in 90%.

> OA can cause morning stiffness but is usually short lived (in contrast to RA).

> **O**A affects the **O**uter joints on the hand — the DIPs. RA affects the inner joints — MCPs and PIPs.

HIGH-YIELD FACTS

Musculoskeletal Emergencies

FIGURE 14-7. OA of the hands.

Note the bony swelling at the base of the thumb and Heberden's nodes at the DIPs. (Reproduced, with permission, from Simon RR, Koenigsknecht SJ. *Emergency Orthopedics: The Extremities*, 4th ed. New York: McGraw-Hill, 2001: 43.)

DIAGNOSIS

- Osteophytes and spurs at joint margin.
- Asymmetric joint space narrowing.
- Subchondral cysts and bone remodeling later on in the disease.

TREATMENT

- Goals of treatment: Relieve symptoms, limit disability, improve function.
- Physical and occupational therapy for ROM and strengthening exercises and providing assistive devices.
- Weight loss.
- Nonopioid analgesics (acetaminophen).
- NSAIDs.
- Topical analgesics (capsaicin).
- Intra-articular steroid injections.
- Surgical intervention: Arthroscopic debridement and lavage, osteotomy, and arthroplasty.

Rheumatoid Arthritis (RA)

DEFINITION

- RA is a chronic, inflammatory, systemic disease that is manifested in the diarthrodial and peripheral joints.
- Etiology is still unknown; may be infectious.
- A combination of genetic and environmental factors control the progression.
- Disease process ranges from self-limited to progressively chronic with severe debilitation.

EPIDEMIOLOGY

- All ethnic groups affected; may be higher in Native Americans.
- Worldwide distribution.
- Affects all ages, prevalence increases with age, and peak incidence is between the fourth and sixth decades.
- Females affected more commonly than males (2:1).
- Associated with HLA-DR4 and HLA-DRB1 genes.

SIGNS AND SYMPTOMS

Common deformities:
- **Ulnar deviation** of the digits.
- **Boutonniere's deformity**—hyperextension of the DIP and flexion of the PIP (Figure 14-8).
- **Swan's neck**—flexion of the DIP and extension of the PIP.

FIGURE 14-8. RA of the hands.

Note inflammation and fusion of the PIP and metacarpointerphalangeal joints with relative sparing of the DIP joint, and the boutonniere deformities on the fifth digits. (Reproduced, with permission, from Simon RR, Koenigsknecht SJ. *Emergency Orthopedics: The Extremities*, 4th ed. New York: McGraw-Hill, 2001: 50.)

DIAGNOSIS

- Diagnosis is based on a constellation of findings over several weeks to months or longer.
- Rheumatoid factor (RF) is present in 70% of patients.
- ESR correlates with the degree of inflammation and is useful in following the course of the disease.
- C-reactive protein can monitor inflammation.

CRITERIA FOR CLASSIFICATION OF RA

At least four of the following seven criteria must be present to diagnose RA; criteria 1 through 4 must have been present for ≥ 6 weeks.

1. Morning stiffness for ≥ 1 hour.
2. Arthritis of ≥ three joint areas.
3. Arthritis of hand joints (see Figure 14-8).
4. Symmetric arthritis.
5. Rheumatoid nodules.
6. Positive serum RF.
7. Radiographic changes: Erosions or bony decalcifications on posteroanterior hand and wrist.

TREATMENT

- Fifty percent of patients are refractory to treatment and display systemic disease.
- Physical and occupational therapy to maintain strength and flexibility and splinting of inflamed joints.
- NSAIDs.
- Corticosteroids.
- Disease-modifying antirheumatic drugs: Gold compounds, hydroxychloroquine (Plaquenil) penicillamine, methotrexate, azathioprine (Imuran), sulfasalazine, cyclophosphamide (Cytoxan), cyclosporine.

Most arthritis patients (regardless of the type of arthritis) have taken NSAIDs for long periods of time. This places them at high risk for ulcers.

Gout

DEFINITION

A disorder in purine metabolism, resulting in the deposition of urate crystals in joint spaces, resulting in joint inflammation and exquisite pain.

EPIDEMIOLOGY

Seen most commonly in middle-aged men.

RISK FACTORS

- Age
- Hyperuricemia
- Alcohol consumption
- Drugs (e.g., thiazide diuretics)

The most common and frequent manifestation of gout is podagra.

SIGNS AND SYMPTOMS

- Acute onset of extreme pain in small joints, accompanied by redness and swelling.
- **Tophi** are aggregates of gouty crystals and giant cells. They can erode away tissue.

- **Podagra** is inflammation of the first metatarsophalangeal joint, which presents in 50 to 75% of all patients as an exquisitely painful nodule on the medial aspect of the foot.

DIAGNOSIS

- Presence of **negatively birefringent crystals** in synovial fluid.
- Elevated serum uric acid levels between attacks (may be low or normal during an acute attack).

TREATMENT

ED:
- Indomethacin to decrease inflammation is first line.
- Colchicine to inhibit chemotaxis is second line.

Outpatient:
- Allopurinol, a xanthine oxidase inhibitor, used as prophylaxis. Do not give in acute phase as it may induce an attack.
- Uricosuric agents (probenecid).

Pseudogout

DEFINITION

Deposition of calcium pyrophosphate dihydrate (CPPD) crystals in joint spaces.

ETIOLOGY

- Acute inflammatory reaction to the deposition of CPPD in joint spaces.
- Changes related to age that make the synovial fluid environment more hospitable to CPPD growth.

SIGNS AND SYMPTOMS

The most common presentation is erythema and swelling of the knee.

DIAGNOSIS

Presence of **positively birefringent crystals** in synovial fluid.

TREATMENT

- Splint joint.
- Aspiration is both diagnostic and therapeutic.
- NSAIDs.

Polymyositis and Dermatomyositis

DEFINITIONS

Connective tissue diseases that result in proximal muscle weakness. Dermatomyositis differs only in that there is a rash, typically affecting the face, neck, and shoulders. There is also a significant risk of an occult malignancy associated with dermatomyositis.

Pseudogout:
Mostly large joints such as shoulder, wrist, and knee are involved.

Gout:
Small joints
Negative birefringence
Pseudogout:
Large joints
Positive birefringence

Polymyositis and dermatomyositis are both more common in women.

ETIOLOGY

Etiology unknown. Many viruses including *Toxoplasma*, influenza, and coxsackie have been implicated. Family history of autoimmune disease or vasculitis is a risk factor.

SIGNS AND SYMPTOMS

- Symmetrical proximal muscle weakness.
- Dysphagia.
- Difficulty getting out of a chair, climbing or descending stairs, kneeling, raising arms.

DIAGNOSIS

Look for the following four criteria:

1. Proximal muscle weakness.
2. Elevated CPK (from necrotic muscle fibers).
3. Low-amplitude action potentials and fibrillations on electromyography (EMG).
4. Increased muscle fiber size on muscle biopsy.

LABORATORY

- Positive antinuclear antibody.
- Elevated CPK, lactic dehydrogenase, serum glutamic oxaloacetic transaminase, aldolase.
- ESR is elevated in only 50% of cases.
- Abnormal EMG.
- Muscle biopsy shows inflammatory infiltrates.
- One fifth of patients have myositis-specific antibodies (Anti-Jo-1).
- Chest x-ray may show interstitial pulmonary disease.

PROGNOSIS

Presentation is usually insidious and progresses slowly, but disease can be fatal. Seventy-five percent survival at 5 years with long-term corticosteroid therapy.

TREATMENT

- ROM exercises.
- Daily steroids.
- If refractory to steroids, azathioprine or methotrexate is given.

> Polymyositis and dermatomyositis can be distinguished from myasthenia gravis by the lack of ocular involvement (ptosis).

> Both azathioprine and methotrexate suppress the bone marrow. Azathioprine is also hepatotoxic.

▶ UPPER EXTREMITY TRAUMA

Anterior Shoulder Dislocation

ETIOLOGY

- Forcible external rotation and abduction of the arm.
- 95 to 97% of all shoulder dislocations.

SIGNS AND SYMPTOMS

- Shoulder pain.
- Patient maintains shoulder in elevated position.

- Difficulty with internal rotation of arm.
- Axillary nerve palsy:
  - Decreased sensation over deltoid.
  - Decreased ability to abduct shoulder.

DIAGNOSIS

See Figure 14-9.

TREATMENT

- Closed reduction under conscious sedation.
- Sling and swathe for 4 weeks.
- ROM exercises.
- Surgical repair for nonreducible dislocations.

COMPLICATIONS

Associated fractures (occur about 40% of the time):
- Bankart: Fracture of glenoid margin.
- Hill–Sachs: Fracture of humeral head.

Posterior Shoulder Dislocation

RISK FACTORS

- Lightning injury.
- Seizures.
- Anterior blow to shoulder or fall on outstretched hand.

SIGNS AND SYMPTOMS

Arm is internally rotated and adducted.

DIAGNOSIS

Lightbulb sign: Lightbulb appearance of internally rotated proximal humerus (Figure 14-10).

Posterior shoulder dislocations account for only 2% of all dislocations.

Posterior shoulder dislocations are frequently missed. Surgical fixation is necessary when the diagnosis is delayed for 2 weeks or more.
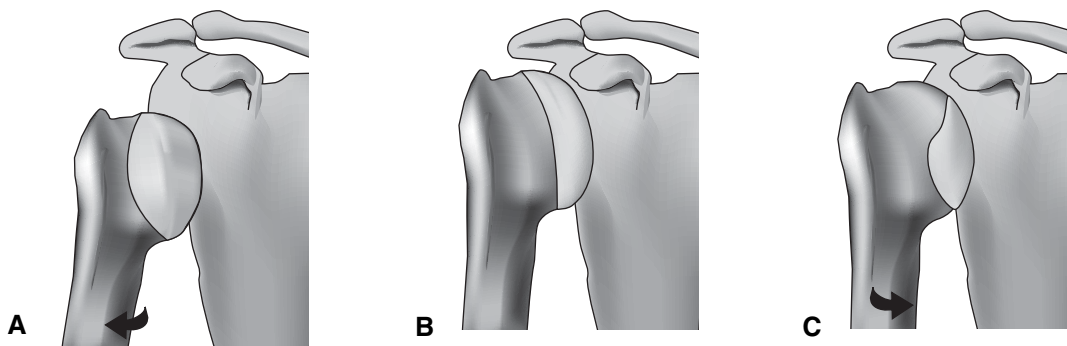
FIGURE 14-9. Anatomy of shoulder dislocations.

A. Anterior dislocation. **B.** Normal shoulder. **C.** Posterior dislocation.

TREATMENT

- Closed reduction under conscious sedation.
- Sling and swathe for 4 weeks.
- ROM exercises.
- Surgical repair for nonreducible dislocations.

Colles' Fracture

- Distal radius fracture with dorsal angulation.
- Most commonly caused by fall on outstretched hand.
- "Dinner fork deformity" is classic.
- More common in elderly women.

TREATMENT

- Short arm cast 4–6 weeks with volar flexion and ulnar deviation.
- Surgical repair for:
  - Open fracture.
  - Comminuted fracture.
  - Intra-articular displaced fracture > 5 mm.

Smith's Fracture

- Distal radius fracture with volar angulation.
- Most commonly caused by direct trauma to dorsal forearm.
- Garden spade deformity.

TREATMENT

Surgical repair needed for most cases.

Galeazzi's Fracture

- Distal one-third radial fracture with dislocation of distal radioulnar joint.

- Commonly caused by fall on outstretched hand with forearm in forced pronation or direct blow to back of wrist.

TREATMENT

Surgical repair needed for most cases.

Monteggia's Fracture

- Proximal one-third ulnar fracture with dislocation of the radial head.
- Commonly caused by fall on outstretched hand with forearm in forced pronation or direct blow to posterior ulna.
- May note injury of radial nerve.

TREATMENT

- Surgical repair for adults.
- Closed reduction for children (children can tolerate a greater degree of displacement).

Radial Head/Neck Fracture

- Fall on outstretched hand.
- Limited flexion and extension of elbow.
- Look for elbow effusion.
- Posterior fat pad visible on x-ray.

TREATMENT

- Collar and cuff if undisplaced.
- Displaced or comminuted fractures may need surgery.

Gamekeeper's Thumb

DEFINITION

Avulsion of ulnar collateral ligament of first metacarpophalangeal (MCP) joint (Figure 14-11).

ETIOLOGY

- Forced radial 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 to 6 weeks for partial tears.
- Surgical repair for complete tears.

If one forearm bone is fractured, look for a fracture of dislocation in the other one.

If posterior fat pad is visible on x-ray, always assume radial head/neck fracture even if not visible.

Gamekeeper's thumb is commonly associated with ski pole injury.
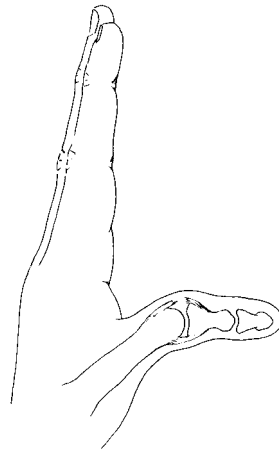
FIGURE 14-11. Gamekeeper's thumb.

(Reproduced, with permission, from Scaletta TA, et al. *Emergent Management of Trauma.* New York: McGraw-Hill, 1996: 220.)

Mallet Finger

DEFINITION

Rupture of extensor tendon at its insertion into base of distal phalanx (Figure 14-12).

A   **B**
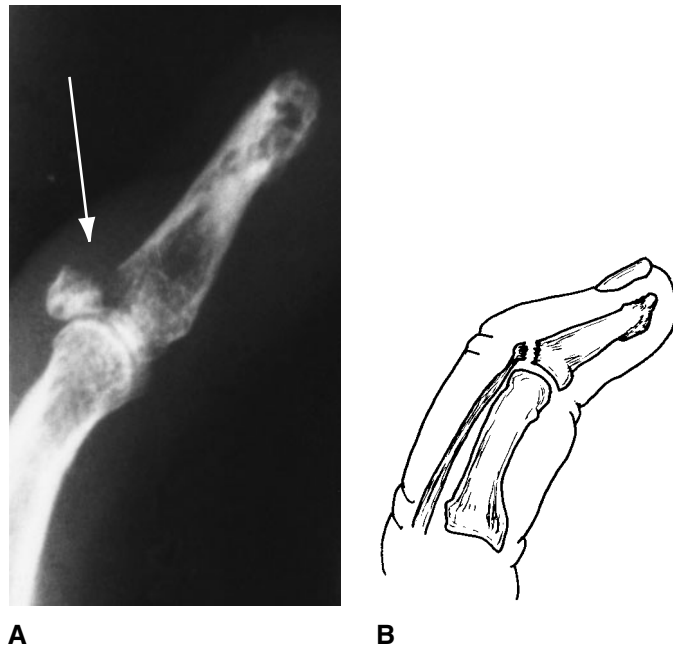
FIGURE 14-12. Mallet finger.

A. Radiograph of an avulsion fracture of the base of the distal phalanx (arrow), which is often associated with mallet finger. **B.** Avulsion of the extensor tendon. (Reproduced, with permission, from Schwartz DT, Reisdorff EJ. *Emergency Radiology.* New York: McGraw-Hill, 2000: 40.)

ETIOLOGY

- Avulsion fracture of distal phalanx.
- Other trauma.

SIGNS AND SYMPTOMS

Inability to extend DIP joint.

TREATMENT

- Splint finger in extension for 6 to 8 weeks.
- Surgery may be required for large avulsions of distal phalanx and for injuries that were not splinted early.

> **LOWER EXTREMITY TRAUMA**

See Table 14-2 for lower extremity injuries.

Pelvis Fracture

- Requires high-impact injury to fracture pelvis (e.g., motor vehicle accident).
- Usually one of multiple injuries.

> If left untreated, mallet finger results in permanent boutonniere deformity.

> Always x-ray the pelvis in patients with multisystem trauma.

T A B L E   1 4 - 2 .   **Common Lower Extremity Injuries**

INJURY	DESCRIPTION	TREATMENT
Lisfranc fracture	■ Fracture through base of second metatarsal ■ The second metatarsal is the stabilizing force of the tarsometatarsal joint	■ Surgical fixation (can be open or closed)
Maisonneuve fracture	■ Malleolar (ankle) and proximal fibula fracture with disruption of the medial deltoid ligament	■ Long leg cast for 6–12 weeks ■ Surgical fixation for: 　■ Medial malleolar fracture 　■ Widened medial joint space
Baker's cyst	■ Cyst in the medial popliteal fossa ■ Associated with arthritis and joint trauma ■ Rupture of cyst can mimic symptoms of deep vein thrombosis	■ Treat underlying cause (adults) ■ Symptomatic relief with NSAIDs
Calcaneal fracture	■ Most frequently injured foot bone ■ Usually occurs due to fall from a height with patient landing on his feet	■ Posterior splint for nondisplaced fractures ■ Surgical repair for displaced fractures
Jones fracture	■ Fracture of diaphysis of fifth metatarsal ■ Usually occurs due to force applied to ball of foot, as in pivoting or dancing ■ Often complicated by nonunion	■ Short leg cast for nondisplaced fractures ■ Surgical repair for displaced fractures

CLINICAL FEATURES

- Pain.
- Crepitus, bruising.
- Pelvic instability.
- May lead to extensive blood loss due to disruption of blood vessels, especially with open fractures.

TREATMENT

- Aggressive resuscitation with intravenous fluids and blood as required.
- Stabilize fracture with bed sheet or external fixation device.
- Consult orthopedic team early.

Hip Fracture

- Incidence increases with age.
- Male-to-female ratio is 1:3.
- Twenty to 30% of elderly patients die in the first year after hip fracture.

CLINICAL FEATURES

- History of fall.
- Inability to bear weight.
- Leg shortened and externally rotated.

DIAGNOSIS

- Anteroposterior (AP) and lateral x-rays of hip.
- Classified as intracapsular or extracapsular.

TREATMENT

- Intracapsular fractures are at risk of avascular necrosis of femoral head and often receive 1° arthroplasty if displaced.
- Extracapsular fractures are usually treated by internal fixation.

Hip Dislocation

- Result of high-energy trauma.
- Associated with other injuries.
- Ninety percent are posterior dislocations. Also called "dashboard dislocations."
- At risk of AVN if left untreated for longer than 6 hours.

Check for sciatic nerve damage: Dorsiflexion of foot and sensation below knee.

DIAGNOSIS

- Posterior dislocation causes shortening, adduction, and internal rotation of extremity.
- Anterior dislocation leaves hip flexed and extremity abducted and externally rotated.

Early closed reduction.

Femoral Shaft Fracture

- Very high energy injury.
- Often occurs in association with hip dislocation or pelvis fracture.

CLINICAL FEATURES

- Deformity.
- Shortened, externally rotated leg.

TREATMENT

- Can be associated with marked blood loss.
- Resuscitate as appropriate.
- Splint with Thomas splint or other traction device.
- Orthopedic consultation.

Proximal Tibial Fracture

- Also known as "bumper fracture."
- Fall onto extended leg or pedestrian hit by car bumper.

CLINICAL FEATURES

- Swollen knee.
- Tenderness over proximal tibia.

TREATMENT

- Immobilize in long leg cast.
- Orthopedic consultation.

Ankle Fracture and Dislocation

- One of the most common orthopedic injuries.
- Classified as unimalleolar, bimalleolar, and trimalleolar.
  - Unimalleolar fractures are stable, requiring only splinting.
  - Bimalleolar and trimalleolar fractures usually require open reduction and internal fixation (ORIF).
- Ottawa Rules: See Tables 14-3 and 14-4 and Figure 14-13.

Ankle injuries are a very common presentation to the ED. The majority are soft-tissue injuries and do not require x-ray.

Ankle dislocations are orthopedic emergencies requiring urgent reduction to prevent neurovascular compromise.

HIGH-YIELD FACTS

Musculoskeletal Emergencies

TABLE 14-3. Ottawa Ankle Rule

CRITERIA FOR ANKLE RADIOGRAPH
- Inability to bear weight immediately after injury and in emergency department
- Bony tenderness at posterior edge of distal 6 cm or tip of medial or lateral malleolus

TABLE 14-4. Ottawa Foot Rule

CRITERIA FOR FOOT RADIOGRAPH
- Bony tenderness at base of fifth metatarsal
- Bony tenderness over navicular
- Inability to bear weight immediately and in emergency department

An antalgic gait is one that results from pain on weight bearing in which the stance phase of the gait is shortened on the affected side.

A 7-year-old boy presents with a limp and complains of groin and hip pain. Radiographs demonstrate a slight widening of the joint space. *Think: Legg–Calvé–Perthes disease.*

▶ **PEDIATRIC MUSCULOSKELETAL PROBLEMS**

Legg–Calvé–Perthes Disease

DEFINITION

- Also called coxa plana, juvenile osteochondrosis.
- Childhood hip disorder that involves AVN of femoral head (Figure 14-14).
- Bilateral in 10 to 15% of patients.

EPIDEMIOLOGY

- 1 in 1,200 children ages 3 to 10 years.
- Peak incidence at age 6 years.
- More common in males by factor of 4:1, but when it affects females, there is more extensive involvement of the epiphysis.

ETIOLOGY

Etiology is unknown but may be due to chronic synovitis, repeated trauma to hip in athletic children, infection, or congenital anomaly.
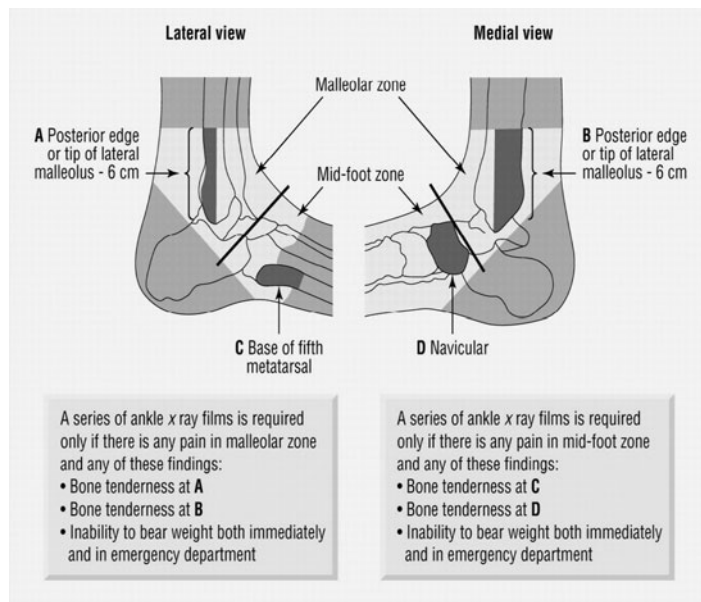
FIGURE 14-13. Ottawa ankle rules.

(Reproduced, with permission, from Bachman LM et al. *BMJ* 2003 Feb 22;326(7386):417.)

FIGURE 14-14. AP view of pelvis demonstrating subchondral lucency of femoral head 2° to avascular necrosis.

This is known as the "crescent sign" of Caffey. (Reproduced, with permission, from Schwartz DT, Reisdorff EJ. *Emergency Radiology.* New York: McGraw-Hill, 2000: 238.)

SIGNS AND SYMPTOMS

- Antalgic gait.
- May or may not identify history of trauma or strenuous activity followed by sudden onset of limping and pain in the anterior groin, anterior thigh or knee, making diagnosis difficult.
- Hip is held in external rotation and is limited in internal rotation. Muscle spasm may also be present.
- Buttock and thigh atrophy may be present on the affected side.

DIAGNOSIS

- Stage 1: No findings to slight widening of the joint space and lateral displacement of the head.
- Stage 2: Radiolucent and radiodense areas with epiphyseal fragmentation: This is the stage when most children present.
- Stage 3: Areas of radiolucency secondary to resorption of necrotic bone; this stage may last for several years in older children as new bone grows.
- Stage 4: Normal-appearing radiograph; however, femoral neck and epiphysis may remain widened. This is the healed phase.

TREATMENT

- Depends on the severity of the disease.
- Most cases are self-limited and no intervention is required.
- If ROM is limited, an abduction cast is applied. Traction may be used to relax adductor spasm and to maintain 45 degrees of abduction and slight internal rotation, which is the best position to center the femoral head to facilitate normal growth.
- The child may be mobilized using crutches and partial weight bearing with the cast. Cast should be removed periodically to mobilize the knee and ankle.
- Surgical intervention may be required for severe cases.

Slipped Capital Femoral Epiphysis (SCFE)

DEFINITION

Condition in which the femoral head maintains its position in the acetabulum but the femoral neck is displaced anteriorly.

EPIDEMIOLOGY

- Occurs between the ages of 10 and 15.
- Male-to-female ratio 2:1.
- Bilateral in 15 to 20%.

An 11-year-old obese boy presents with thigh and knee pain that worsens when he plays sports. He is walking with a limp and holds his hip in slight external rotation. *Think: SCFE.*

ETIOLOGY

Cause is unclear, may involve:
- History of trauma
- Weakened physis
- Obesity
- Endocrine disorder

SIGNS AND SYMPTOMS

- Antalgic gait that may have been present for months or may have occurred acutely.
- Hip, medial thigh, or knee pain intermittently worsened with activity.
- Hip may be held in external rotation, and internal rotation is limited.

DIAGNOSIS

AP radiograph of the pelvis (Figure 14-15) and lateral and frogleg views (if no acute slippage) to look for:
- Irregular widening of the epiphyseal plate.
- Globular swelling of the joint capsule.
- Posterior and inferior displacement of the femoral head (if slippage).
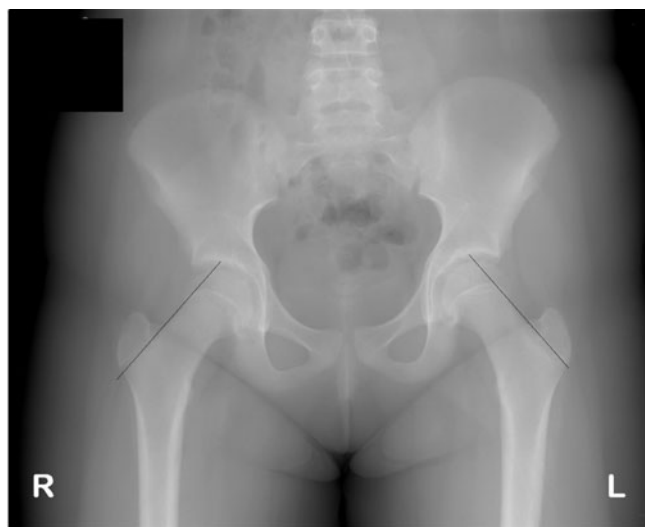
ANTERO-POSTERIOR (AP) VIEW

FROG LEG VIEW

F I G U R E 1 4 - 1 5 . **Hip radiographs in a 13-year-old girl with mildly slipped capital femoral epiphysis (SCFE) on the right.**

Note on the AP view that a line drawn along the superior border of the femoral neck (Klein line) shows less femoral head superior to the line on the right than it does in the normal hip on the left.

Surgical stabilization.

Osgood–Schlatter Disease

EPIDEMIOLOGY

- A cause of adolescent knee pain.
- Cause is uncertain but may be due to:
    - Apophysitis at the insertion of the patellar tendon at the tibial tuberosity.
    - Repeated quadriceps contraction resulting in tendonitis or partial avulsions at the tibial tubercle.
- Seen at ages 9 to 15, when the apophysis is most prone to injury.
- Males are affected more commonly than females.

SIGNS AND SYMPTOMS

- Pain at tibial tuberosity.
- Symptoms are worsened by palpation and knee extension against resistance.
- Prominent tibial tubercle.
- Soft-tissue swelling.

DIAGNOSIS

Radiographs demonstrate:
- Prominence of the tibial tubercle.
- Heterotopic ossification, which appears as irregularities.

TREATMENT

- Self-limited and usually resolves with complete ossification of the tibial tuberosity by age 15.
- NSAIDs.
- Cryotherapy.
- Stretching of the quadriceps.
- Limit activity to pain tolerance.
- Avoid kneeling, running, and jumping.

Pediatric Fractures

- Two important differences between children's bones and adults':
    - Presence of epiphyses.
    - They are softer, so it is more common to have a fracture than a significant ligament injury.
- Also unique to children are buckle fractures (incomplete fracture with buckling of cortex) and greenstick fractures (only one cortical surface breaks).
- Epiphyseal injuries: See Table 14-5.

Supracondylar Humeral Fracture

- Common in children.
- Fall on outstretched hand.

> A 12-year-old boy presents complaining of knee pain for 2 weeks. Physical exam demonstrates a prominent tibial tubercle. *Think: Osgood–Schlatter disease.*

HIGH-YIELD FACTS

Musculoskeletal Emergencies

TABLE 14-5. Salter–Harris Fractures

CLASS	DEFINITION	TREATMENT
I	▪ Fracture through epiphyseal plate	Closed reduction
II	▪ Fracture of metaphysis with extension into epiphyseal plate ▪ Most common type of Salter–Harris fracture	Closed reduction
III	▪ Intra-articular fracture of epiphysis with extension into epiphyseal plate	Open reduction
IV	▪ Intra-articular fracture of epiphysis, metaphysis, and epiphyseal plate	Open reduction
V	▪ Crush injury of epiphyseal plate	Open reduction

CLINICAL FEATURES

- Deformity and tenderness.
- Joint effusion on x-ray.

TREATMENT

Surgery needed if displacement, angulation, or neurovascular deficit.

▶ **ORTHOPEDIC COMPLICATIONS**

General Complications of Orthopedic Trauma

- Compartment syndrome (see below)
- Rhabdomyolysis
- Malunion
- Nonunion
- AVN (see below)
- Fat embolism (especially with fracture of long bone)
- Hemorrhage (especially with pelvic fracture)
- Neurovascular injury

Compartment Syndrome

DEFINITION

Compartment syndrome is most common in the lower extremity.

- Compartment syndrome results when the pressure in a compartment exceeds the arterial perfusion pressure.
- This normally occurs at pressures > 20 mm Hg.
- It occurs at lower pressures when the arterial pressure is lower than normal, such as in prolonged systemic shock.
- The excess compartment pressure causes muscle and nerve necrosis due to ischemia.

ANATOMY

Major compartments include:
- Hand: Associated with crush injury.
- Forearm: Associated with supracondylar fracture of the humerus.
- Thigh: Associated with crush injury.
- Leg: Associated with tibial fracture.
- Foot: Associated with calcaneus fracture.

RISK FACTORS

- Crush injuries.
- Circumferential burns.
- Constrictive devices (military antishock trousers suit, casts, clothing).
- Hemorrhage.
- Edema.
- Patients with altered mental status who cannot report compartment pain.

SIGNS AND SYMPTOMS

Earlier Findings
- Pain out of proportion to the injury.
- Pain with passive flexion.
- Decreased two-point sensory discrimination.
- Paresthesia or hypesthesia.
- Tenseness of compartment.

Late Findings
- Pallor of skin
- Absence of pulses
- Cold extremity

DIAGNOSIS

- Maintain high index of suspicion in those with high-risk injuries.
- Made by measuring compartment pressure, which can be done with a commercial device (the Stryker) or with an 18G needle connected to a manometer and a water piston via a three-way stopcock.

TREATMENT

- Remove any constricting devices.
- Fasciotomy for pressures > 30 mm Hg.

AVN of the Hip

ETIOLOGY

- The medial and lateral circumflex arteries supply the femoral head and then circle closely around the head of the femur, rendering them vulnerable.
- These arteries may become occluded as in sickle cell disease or during immobilization.
- AVN is also frequently a complication following fractures of the neck of the femur or dislocation of the head.
- It may occur at any time postop up to 20 years later.

6 Ps *of compartment syndrome:*
Pain
Paresthesia (most reliable)
Paralysis
Pallor
Pulselessness
Poikilothermia

The presence of pulses does *not* rule out compartment syndrome.

SIGNS AND SYMPTOMS

- Aching of joint early on.
- Difficulty sitting for prolonged periods.
- Weakness of hip.
- Limp.

DIAGNOSIS

- Radiographs may not show signs of the disease until it is more advanced so that the femoral head has started to flatten and become irregularly shaped.
- Later films show evidence of OA.

TREATMENT

- In less severe cases, physical therapy can provide a strengthening and mobility program and assistive devices for protective weight bearing.
- More severe cases require total hip replacement followed by physical therapy.
- There is a worse prognosis in older patients compared to the young who are still growing.

Endocrine Emergencies

DEFINITION

Acute life-threatening emergency that occurs secondary to cortisol and aldosterone insufficiency.

ETIOLOGY

- Autoimmune
- Infections (tuberculosis [TB], fungal)
- Metastatic cancer
- Adrenal hemorrhage (trauma, burns, sepsis, coagulopathy)
- Drugs
- Withdrawal of steroid therapy

> The most common cause of adrenal crisis is abrupt withdrawal of steroid therapy.

SIGNS AND SYMPTOMS

- Shock
- Anorexia
- Nausea, vomiting
- Abdominal pain
- Fatigue
- Confusion, coma
- Fever
- Hyperpigmentation (seen in long-standing primary adrenal insufficiency)

> Adrenal insufficiency is low serum cortisol, diagnosed by inadequate response to ACTH stimulation test, or 24-hour urine cortisol level.

DIAGNOSIS

- Blood draw for electrolytes, cortisol, adrenocorticotropic hormone (ACTH).
- Low serum cortisol level and inadequate serum cortisol response 30 or 60 minutes after ACTH stimulation test (cosyntropin stimulation test).
- Electrolyte abnormalities: Hyperkalemia and hyponatremia.
- Computed tomographic (CT) scan of the abdomen may show hemorrhage in the adrenals, calcification of the adrenals (seen with TB), or metastasis.
- Electrocardiogram (ECG) may show elevated peaked T waves indicating hyperkalemia.

TREATMENT

- Aggressive rehydration with 5% dextrose in normal saline (D$_5$NS).
- Steroid replacement with dexamethasone.
- Identification and treatment of precipitating cause.
- Long-term glucocorticoid and mineralocorticoid replacement.
- An ACTH stimulation test should be performed whenever a diagnosis of adrenal insufficiency is considered. Diagnostic tests should not delay timely treatment of patients in adrenal crisis.

DEFINITION

- Most common endocrine disorder in the world (up to 3% of population).
- Type 1: Autoimmune pancreatic beta-cell destruction with resulting insulin deficiency.

- Type 2: Genetically influenced disease characterized by impairment in insulin secretion and/or action.
  - Fasting plasma glucose > 126 mg/dL on two occasions in the presence of symptoms of hyperglycemia.
  - Absolute or relative deficiency of insulin.

PATHOPHYSIOLOGY

- Insulin is a major anabolic hormone with inhibitory effects on ketogenesis, glycogenolysis, lipolysis, and proteolysis.
- Inhibits ketogenesis, glycogenolysis, lipolysis, gluconeogenesis.

SIGNS AND SYMPTOMS

- Usually presents with symptomatic hyperglycemia or diabetic ketoacidosis (DKA) (discussed below).
- Polyuria.
- Polydipsia.
- Weight loss.
- Dehydration.
- Blurred vision.
- Fatigue.

TREATMENT

- Insulin for injections for Type 1 DM.
- Oral hypoglycemic agents alone or in combination with insulin for Type 2 DM.

Dawn Phenomenon

Early morning hyperglycemia from decreasing insulin concentration and increasing insulin requirement from surge in counterregulatory hormones (e.g., growth hormone [GH]).

Somogyi Effect

- Characterized by nighttime hypoglycemia followed by a dramatic increase in fasting glucose levels and increased plasma ketones
- If Somogyi phenomenon is suspected, patients should get their blood glucose checked around 3 A.M. Hypoglycemia at this time confirms diagnosis.
- The morning hyperglycemia is a rebound effect.
- Intermediate-acting insulin administration at bedtime (rather than earlier in the evening) can prevent this effect (try to avoid peaking of insulin effect in the middle of the night).

> A patient presents with persistent morning hyperglycemia, despite steadily increasing his nighttime NPH insulin dose. Further, he complains of frequent nightmares. His wife brings him now because she witnessed him having a seizure in the middle of the night. *Think: Somogyi effect.*

▶ DIABETIC KETOACIDOSIS (DKA)

DEFINITION

- State of absolute or relative insulin deficiency and counterregulatory excess resulting in hyperglycemia, dehydration, acidosis, and ketosis.
- More common in Type 1 diabetics.

ETIOLOGY

Most common causes:
- Infection
- Discontinuation of insulin replacement
- New-onset DM

PATHOPHYSIOLOGY

- Insulin deficiency and counterregulatory hormones cause severe hyperglycemia.
- Hyperglycemia leads to osmotic diuresis and depletion of Na, K^+, PO_4, water.
- Counterregulatory hormones enhance lipolysis and free fatty acid generation. Beta oxidation of free fatty acid produces ketones.
- Ketosis and anion gap metabolic acidosis.
- Acetone, a volatile ketone, produces the fruity odor typical of DKA.

SIGNS AND SYMPTOMS

- Symptomatic hyperglycemia (polyuria, polydipsia, nocturia).
- Weakness, nausea, vomiting.
- Confusion, coma.
- Signs of dehydration are present and patients may be hypotensive and tachycardic.
- Kussmaul's respirations (slow deep breaths) may be present.
- Acetone (fruity) odor may be present on the patient's breath.
- Fever suggests underlying infection.

DIAGNOSIS

- *Hyperglycemia.*
- *Hyperketonemia.*
- *Anion gap metabolic acidosis* (ketones are unmeasured ions).
- Usually, the diagnosis can be presumed at the bedside if patient's urine is strongly positive for ketones and the fingerstick glucose is high.
- Glucose is usually between 400 and 800 mg/dL.
- Initially, potassium is high due to acidosis but drops with treatment, so it is important to replace it.
- Blood urea nitrogen (BUN) may be increased because of prerenal azotemia.

TREATMENT

- Rapid administration of intravenous (IV) fluids (initially NS) should begin promptly (usual deficit is 3 to 6 L).
- Insulin infusion to control hyperglycemia and reverse ketosis: Start with bolus 0.1 U/kg IV, then infuse at 0.1 U/kg/hr.
- Continue insulin infusion until glucose is < 250 *and* ketoacidosis (anion gap) is resolved (change intravenous (IV) fluid to $^1/_2$ NS when glucose is < 250).
- Bicarbonate (to reverse acidosis) is controversial and not routinely recommended:
    - Side effects include lowering intracellular pH, hypokalemia, and shifting O_2 dissociation curve.
    - Use judiciously in selected patients with pH < 7.1, severe hyperkalemia, or refractory hypotension.

A 26-year-old man presents with vomiting. He reports several days of malaise and fatigue. He appears pale and diaphoretic. He is afebrile but tachycardic to 120 with a blood pressure (BP) of 105/70. His blood sugar is 550. *Think: DKA as a first presentation of diabetes mellitus.*

DKA treatment goals:
- Replacement of fluid losses
- Correction of metabolic derangements
- Reversal of ketosis
- Treatment of precipitating causes
- Restoration of normal diabetic regimen

Dehydration and acidosis (exchange of H for K) may cause serum K to be falsely normal.

- Frequent monitoring and replacement of serum potassium is essential. Add potassium to IV fluids when $K^+ < 4.5$.
- Replace other electrolytes (e.g., phosphate) as needed.
- Identify and treat precipitating cause aggressively.

► NONKETOTIC HYPEROSMOLAR COMA (NKHC)

DEFINITION

- Syndrome of marked hyperglycemia without ketoacidosis.
- Insulin action is inaedequate to prevent hyperglycemia. Small amounts of insulin are present that are enough to protect against lipolysis and subsequent ketoacid generation.
- Absence of ketosis leads to dramatic hyperglycemia (glucose > 800 to 1,000).
- More common in Type 2 diabetics (who produce small amounts of insulin) and the elderly.

CAUSES

- Infection
- Myocardial infarction (MI)
- Stroke
- Gastrointestinal (GI) bleed
- Pancreatitis
- Uremia
- Drugs (steroids, thiazide diuretics)

SIGNS AND SYMPTOMS

- Symptomatic hyperglycemia and pronounced osmotic diuresis.
- Seizures, coma.
- Decreased skin turgor.
- Hypertonicity of serum.
- More pronounced K, Mg, PO_4 losses than DKA.
- Abdominal pain is usually not a presenting symptom.

TREATMENT

- Replacement of fluid losses (usually 8 to 10 L initially) with NS solution.
- Switch to $1/2$ NS with potassium when $K^+ < 4.5$.
- Insulin requirements usually less than in DKA (0.1 U/kg/hr).
- Identify and treat precipitating factor.

► HYPOGLYCEMIA

DEFINITION

Low plasma glucose (< 60 mg/dL).

CAUSES

Multiple.

A 73-year-old woman who is a known diabetic is brought in due to altered mental status. The home health aide states the patient ran out of her medicines 4 days ago. Her fingerstick glucose is > 1,000. *Think: NKHC.*

Rapid correction of hyperosmolar state may lead to cerebral edema (high mortality).

Aggressive fluid resuscitation in patients with heart failure may require invasive monitoring.

Hypoglycemia due to certain oral hypoglycemic agents lasts approximately 24 hours, so it cannot be corrected quickly. Patients with long-acting oral hypoglycemic agent overdose should be admitted to the hospital for 24-hour monitoring and continuous glucose administration.

SIGNS AND SYMPTOMS

- Low plasma glucose.
- Neuroglycopenic symptoms (confusion, irritability).
- Sympathetic activation symptoms (sweating, palpitations, anxiety).

DIAGNOSIS

- Low blood sugar.
- Symptoms of hypoglycemia.
- Reversal of symptoms with restoration of blood glucose.
- Insulin levels.
- C-peptide levels.
- Sulfonylurea assay.

If etiology is unclear to differentiate between exogenous insulin, endogenous insulin, or drug effect.

TREATMENT

- Oral replenishment with fast-acting carbohydrate (glucose tablet, candy, sweetened juice) is appropriate for patients with intact mental status.
- Elderly or obtunded patients should receive IV dextrose (25 to 50 g of 50% dextrose).
- Patients with adrenal insufficiency may require hydrocortisone in addition to dextrose.
- Patients who require prolonged monitoring of blood sugar (overdose of long-acting insulin or oral hypoglycemics) or are unable to maintain adequate oral glucose intake should be admitted.
- Intramuscular (IM) glucagon can reverse hypoglycemia in 10 to 15 minutes. Useful when IV access is a problem in obtunded patients.

▶ **PARATHYROID DISORDERS**

Parathyroid Physiology

- Parathyroid hormone (PTH), calcitonin (from thyroid), and vitamin D work in concert to regulate calcium.
- PTH increases serum calcium by three mechanisms:

1. Increasing resorption of calcium (and phosphate) from bone.
2. Decreasing renal excretion of calcium (and increasing phosphate excretion).
3. Stimulating kidneys to produce calcitriol, a potent vitamin D metabolite that enhances intestinal absorption of calcium.

Hyperparathyroidism

CAUSES

- Primary hyperparathyroidism is most common: Parathyroid adenoma, hyperplasia, carcinoma.

- Multiple endocrine neoplasia types I and IIA both feature parathyroid neoplasms as part of the syndrome.
- Milk-alkali syndrome.
- Granulomatous.

SIGNS AND SYMPTOMS

- Kidney stones
- Bone pain
- Fatigue, confusion, stupor
- Depression
- Abdominal pain (ulcers, pancreatitis)
- Can also be asymptomatic

Signs of hyperparathyroidism: "Stones, bones, abdominal groans, and psychic moans"

TREATMENT

- Volume expansion with saline followed by loop diuretics to induce urinary Ca^{2+} loss.
- Identify and treat underlying cause of hypercalcemia.

Hypoparathyroidism

DEFINITION

Syndrome of decreased calcium, increased phosphate, or decreased PTH.

CAUSES

- Most commonly seen as a complication of thyroid/parathyroid surgery (inadvertent excision).
- Less common causes: Autoimmune, congenital (DiGeorge's), infiltrative (Wilson's hemochromatosis).

SIGNS AND SYMPTOMS

- Perioral and digital paresthesias.
- Decreased myocardial contractility.
- Chvostek's and Trousseau's signs.

TREATMENT

Treat hypocalcemia.

Pheochromocytoma

DEFINITION

Catecholamine-secreting tumor of neural crest cells, most often found in adrenal medulla.

EPIDEMIOLOGY

- Equal incidence in men and women.
- Tumors in women are three times as likely to be malignant.

CAUSES

Episodes precipitated by abdominal movement, trauma, drugs, or idiopathic.

SIGNS AND SYMPTOMS

Clinical presentation is of catecholamine excess:
- Hypertensive crisis with headaches, chest pain, palpitations, shortness of breath, sweating.
- Sequelae may include arrhythmias, MI, renal failure, lactic acidosis, CVA, and death.
- Hallmark of disease is marked hypertension (sustained or paroxysmal).

5 Hs:
- Headache
- Hypertension
- Hot (diaphoretic)
- Heart (palpitations)
- Hyperhidrosis
- 6th H: Hyperglycemia

DIAGNOSIS

- Family history in familial disease.
- Elevated urinary catecholamine excretion (vanillylmandelic acid and metanephrines).
- CT or magnetic resonance imaging (MRI) scan of abdomen/pelvis.
- Metaiodobenzylguanidine (MIBG) scan to detect tumors not seen on CT/MRI.

TREATMENT

- Control of hypertension with alpha blockade (phenoxybenzamine).
- Tachycardia may be controlled with beta blockers (propranolol, esmolol) after alpha blockade.
- Avoid use of beta blockers alone (unopposed alpha activity may lead to paradoxical increase in BP).
- Acute hypertensive crisis can be controlled by nitroprusside or phentolamine.

▶ **PITUITARY DISORDERS**

Pituitary Physiology

- Pituitary gland sits in sella turcica (near optic chiasm and cavernous sinus).
- Anterior pituitary produces thyroid-stimulating hormone (TSH), adrenocorticotropic hormone (ACTH), GH, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and prolactin.
- Posterior pituitary stores and releases oxytocin and vasopressin (antidiuretic hormone).
- Tumors of pituitary gland (most commonly prolactinoma) may present with visual field defects (most commonly bitemporal hemianopsia) or cranial nerve palsies (III, IV, V, VI) from local compression.

Pituitary Apoplexy

- Sudden hemorrhage into the pituitary, often into a preexisting adenoma.

- Sudden, severe headache; diplopia; and hypopituitarism.
- May cause life-threatening hypotension.
- Prompt neurosurgical decompression is required.

Sheehan's Syndrome

- Rare cause of hypopituitarism.
- Postpartum pituitary necrosis.
- Failure to lactate and menstruate after delivery.

Panhypopituitarism

Hormone loss follows typical sequence:
GH (first) → LH/FSH → TSH → ACTH → prolactin (last)

Thyroid Physiology

- Thyroid function controlled by hypothalamus (thyrotropin-releasing hormone) → pituitary (TSH) → thyroid (T_3, T_4).
- Thyroxine (T_4) is converted to triiodothyronine (T_3) (more potent) in peripheral tissues.

Causes of Hyperthyroidism

- Graves' disease (autoimmune stimulation of TSH receptors)
- Toxic multinodular goiter
- Toxic adenoma
- Thyroiditis (autoimmune or viral)

Thyroid Storm

DEFINITION

Life-threatening hypermetabolic state resulting from hyperthyroidism.

EPIDEMIOLOGY

Mortality is high (20 to 50%) even with the correct treatment. Incidence has decreased with the advent of a preoperative preparation before thyroid surgery.

ETIOLOGY

- Infection.
- Trauma and major surgical procedures.
- DKA.
- MI, stroke, pulmonary embolism.
- Withdrawal of antihyperthyroid medications, iodine administration, thyroid hormone ingestion.
- Idiopathic.

A 41-year-old woman with known hyperthyroidism is brought in by her family, who state that she has had days of diarrhea and has now started acting "crazy" with labile mood. She is febrile to 102°F, has a pulse of 140, and has rales on auscultation. *Think: Thyroid storm.*

SIGNS AND SYMPTOMS

Overactivated sympathetic nervous system causes most of the signs and symptoms of this syndrome:

- Fever > 101°F.
- Tachycardia (out of proportion to fever) with a wide pulse pressure.
- High-output congestive heart failure and volume depletion.
- Exhaustion.
- GI manifestations: Diarrhea, abdominal pain.
- Continuum of central nervous system (CNS) alterations (from agitation to confusion when moderate, to stupor or coma with or without seizures).
- Jaundice is a late and ominous manifestation.

DIAGNOSIS

- This is a clinical diagnosis, and since most patients present in need of emergent stabilization, treatment is initiated empirically.
- Patients may have untreated hyperthyroidism.
- May also occur in the setting of unintentional or intentional toxic ingestion of synthetic thyroid hormone.

TREATMENT

Primary stabilization:
- Airway protection.
- Oxygenation.
- Assess circulation (pulse/BP) and continuous cardiac monitoring.
- IV hydration.

Definitive treatment:
- Beta-blocker therapy (e.g., propranolol) to block adrenergic effects.
- Treat fever with acetaminophen (not aspirin, which would displace T_4 from thyroid-binding protein).
- Propylthiouracil (PTU) or methimazole to block synthesis of new thyroid hormone.
- Iodine to decrease release of preformed thyroid hormone. Do not give iodine until the PTU has taken effect (1.5 hours).
- Treat any possible precipitating factors that may be present.
- Radioiodine administration to destroy thyroid gland once patient is stable and euthyroid.

In initial stabilization of thyroid storm, cooling blankets can be applied to treat hyperpyrexia, if present.

Never send any thyroid storm patient for a procedure involving iodine contrast before giving PTU.

Methimazole does not block conversion of T_3 to T_4.

Causes of Hypothyroidism

- Hashimoto's thyroiditis (most common cause).
- Iodine deficiency or excess.
- Radiation therapy to neck (from other malignancy).
- Medications (lithium is most common).
- Secondary causes include pituitary tumor, tuberculosis, and Sheehan's syndrome.

Myxedema Coma

DEFINITION

Life-threatening complication of hypothyroidism, with profound lethargy or coma usually accompanied by hypothermia: Mortality is 20 to 50% even if treated early.

- Sepsis.
- Prolonged exposure to cold weather.
- CNS depressants (sedatives, narcotics).
- Trauma or surgery.

SIGNS AND SYMPTOMS

- Profound lethargy or coma is obvious.
- Hypothermia: Rectal temperature < 35°C (95°F).
- Bradycardia or circulatory collapse.
- Delayed relaxation phase of deep tendon reflexes, areflexia if severe (this can be a very important clue).
- Hyponatremia.
- Hypoglycemia.
- Hypoventilation.

DIAGNOSIS

- History of hypothyroidism.
- Exclude other causes of coma.
- Complete blood count with differential.
- Blood and urine cultures.
- Serum electrolytes, BUN and creatinine, blood glucose.
- Urine toxicology screen.
- Serum transaminases and lactic dehydrogenase.
- Arterial blood gas to rule out hypoxemia and CO_2 retention.
- Cortisol level.
- Carboxyhemoglobin.
- Thyroid function testing.

CT scan and chest radiograph are also commonly ordered because myxedema coma is often a diagnosis of exclusion.

TREATMENT

- Airway management with mechanical ventilation is often necessary.
- Prevent further heat loss but do not initiate active rewarming unless cardiac dysrhythmias are present, as peripheral vasodilation can lead to hypotension.
- Monitor patient in an intensive care setting.
- Do not let lab results delay therapy.
- Pharmacologic therapy:
  - IV levothyroxine (oral absorption may be impaired).
  - Glucocorticoids (until coexisting adrenal insufficiency is excluded).
- IV hydration (avoid hypotonic fluids).
- Rule out and treat any precipitating causes (antibiotics for suspected infection).

Hypothermia is often missed by regular thermometers. Use a rectal probe if profound hypothermia is present.

Differential diagnosis of myxedema coma:
- Severe depression or primary psychosis
- Drug overdose or toxic exposure
- CVA
- Liver failure
- Hypoglycemia
- CO_2 narcosis
- CNS infection

HIGH-YIELD FACTS

Endocrine Emergencies

Dermatologic Emergencies

Shapes

- Annular: Ring-shaped
- Arcuate: Arc-shaped
- Confluent: Coalescence of lesions
- Discoid: Coin- or disc-shaped
- Linear: Line-shaped
- Serpiginous: Wavy linear lesions
- Target: Concentric rings (like a target sign)

Definitions

Flat Lesions

- **Macule:** Nonpalpable, discrete area of change in color. Generally < 10 mm diameter (Figure 16-1).
- **Erythema:** Nonpalpable, diffuse redness.
- **Patch:** Nonpalpable, discrete area of change in color > 10 mm diameter.
- **Telangiectasia:** Blanchable, visibly dilated blood vessel at skin surface.
- **Petechiae:** Nonblanching purple spots < 2 mm.
- **Purpura:** Nonblanching purple spots > 2 mm (can be palpable in some conditions).

Raised, Solid Lesions

- **Papule:** Solid, raised lesion < 5 mm diameter (Figure 16-2).
- **Nodule:** Solid, raised lesion > 5 mm diameter (Figure 16-3).
- **Tumor:** Solid, palpable lesion > 10 mm.
- **Induration:** Raised "hardening" of the skin.
- **Wheal:** Transient localized skin edema (Figure 16-4).
- **Scale:** Flake of keratinized epidermal cells on top of skin surface (Figure 16-5).
- **Plaque:** Flat-topped lesion of induration (Figure 16-6).
- **Crust:** Dried serous/serosanguinous exudate (Figure 16-7).
- **Hyperkeratosis:** Thickened stratum corneum.
- **Lichenification:** Indurated and thickened skin caused by excessive scratching and chronic inflammation.
- **Filiform/Warty:** Flesh-colored, circumscribed hypertrophy of epidermal papillae.

Raised, Fluid-Filled Lesions

- **Vesicle:** Blister < 5 mm diameter (Figure 16-8).
- **Bulla:** Blister > 5 mm diameter.
- **Pustule:** Visible pus under the skin (yellow, white, or green in color) (Figure 16-9).
- **Comedone:** Collection of sebum and keratin in a blocked, dilated sebaceous duct.
- **Cyst:** Sack of fluid-containing material (Figure 16-10).
- **Abscess:** Tender, fluctuant pocket of pus with surrounding inflammation deep to skin.

Depressed Lesions

- **Erosion:** Localized epidermal loss (Figure 16-11).
- **Atrophy:** Thinning of skin from layer loss (Figure 16-12).

HIGH-YIELD FACTS

Dermatologic Emergencies
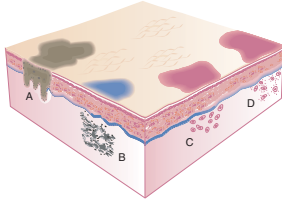
FIGURE 16-1. Macule.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
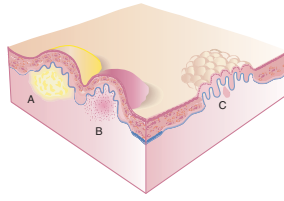
FIGURE 16-2. Papule.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
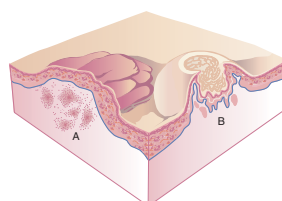
FIGURE 16-3. Nodule.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
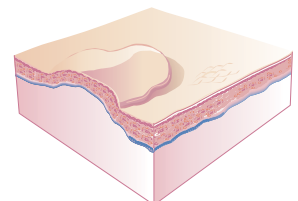
FIGURE 16-4. Wheal.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
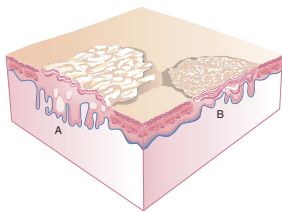
FIGURE 16-5. Scale.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
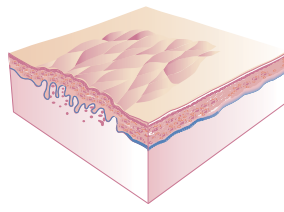
FIGURE 16-6. Plaque.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
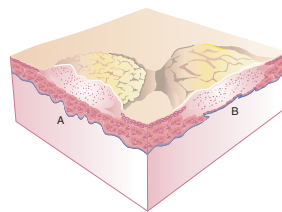
FIGURE 16-7. Crust.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
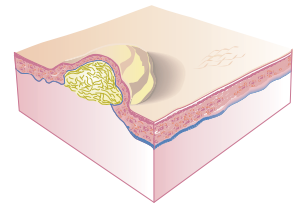
FIGURE 16-8. Vesicle.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
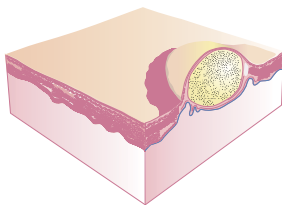
FIGURE 16-9. Pustule.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
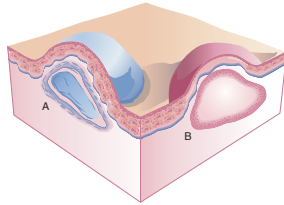
FIGURE 16-10. Cyst.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
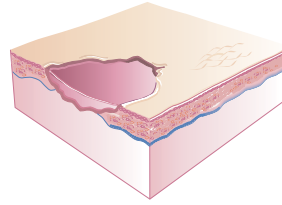
FIGURE 16-11. Erosion.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)
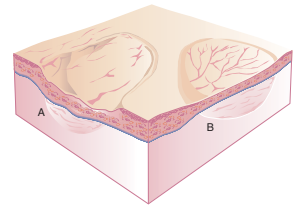
FIGURE 16-12. Atrophy.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)

- **Excoriation:** Abraded or scratched skin.
- **Ulcer:** Localized dermal and epidermal loss (can be deeper) (Figure 16-13).
- **Fissure:** Cleft-shaped ulcer.

Color

Black, white, yellow, red, flesh, brown, hyper- or hypopigmented, blanchable, or nonblanchable.

Distribution

Flexor/extensor surfaces, sun-exposed, dermatomal, clothing-covered, intertriginous, discrete/scattered/grouped.

Size (Does Matter)

- Planar dimensions: Circular—average diameter, oblong—length and width.
- Height/depth: If raised or depth discernible, or if biopsied.
- Large lesions: Estimate body surface area involved (see Environmental Emergencies chapter).

FIGURE 16-13. Ulcer.

(Reproduced, with permission, from Rycroft RJG, Robertson SL. *A Color Handbook of Dermatology.* London, England: Manson Publishing Ltd., 1999: 9.)

Use the "Rule of 9s" to estimate body surface area.

► **GENERAL DIAGNOSIS**

History of Present Illness

- Signs and symptoms (painless, itching, burning, etc.).
- How long present?
- Where are lesions?
- Evolutionary changes?
- Systemic complaints?
- Exposures (chemicals, foods, animals, plants, medications, etc.)?
- Allergies?
- Ever have this before?
- Partially treated?

Examination

See above.

Diagnostic Procedures

See Table 16-1.

Top Dermatologic Problems in Emergency Medicine

- Decubitus ulcer
- Abscess
- Cellulitis
- Erysipelas

Dermatologic physical exam: Look everywhere.

These are the problems you'll most likely be tested on, not necessarily the ones you'll see most frequently in the emergency department.

	DEFINITIONS
Diascopy	Pressing of a glass slide firmly against a red lesion will determine if it is due to capillary dilatation (blanchable) or to extravasation of blood (nonblanchable).
KOH preparation	Used to identify fungus and yeast. Scrape scales from skin, hair, or nails. Treat with a 10% KOH solution to dissolve tissue material. Septated hyphae are revealed in fungal infections, and pseudohyphae and budding spores are revealed in yeast infections.
Tzanck preparation	Used to identify vesicular viral eruptions. Scrape the base of a vesicle and smear cells on a glass slide. Multinucleated giant cells will be identified in herpes simplex, herpes zoster, and varicella infections.
Scabies preparation	Scrape skin of a burrow between fingers, side of hands, axilla, or groin. Mites, eggs, or feces will be identified in scabies infection.
Wood's lamp	Certain conditions will fluoresce when examined under a long-wave ultraviolet light ("black" lamp). Tinea capitis will fluoresce green or yellow on hair shaft.
Patch testing	Detects type IV delayed hypersensitivity reactions (allergic contact dermatitis). Nonirritating concentrations of suspected allergen are applied under occlusion to the back. Development of erythema, edema, and vesicles at site of contact 48 hours later indicates an allergy to offending agent.
Biopsy	Type of biopsy performed depends on the site of lesion, the type of tissue removed, and the desired cosmetic result. Shave biopsy is used for superficial lesions. Punch biopsy (3 to 5 mm diameter) can remove all or part of a lesion and provides tissue sample for pathology. Elliptical excisions provide more tissue than a punch biopsy and are used for deeper lesions or when the entire lesion needs to be sent to pathology.

- Necrotizing infection
- Herpes zoster
- Erythema multiforme (EM)
- Henoch–Schönlein purpura (HSP)
- Purpura
- Urticaria
- Pemphigus
- Staphylococcal scalded syndrome
- Stevens–Johnson syndrome (SJS)
- Toxic epidermal necrolysis (TEN)

Top Causes of Rash with Fever

- Rubella
- Measles
- Staphylococcal scalded skin syndrome
- Toxic shock syndrome
- Scarlet fever
- Meningococcemia

Rashes that can be seen on palms and soles: **Mrs. HEP**

- **M**eningococcemia
- **R**MSF
- **S**yphilis
- **H**and, foot, and mouth disease
- **E**M
- **P**soriasis

- Disseminated gonococcal infection
- Bacterial endocarditis
- Rocky Mountain spotted fever (RMSF)
- Kawasaki's disease
- Erythema nodosum
- Hypersensitivity vasculitis

Initial Modalities

- Astringents (drying agents): Domeboro solution.
- Emollients (moisturizers): Eucerin cream, lotion.

Antihistamines

For pruritic (itchy) disorders:
- Diphenhydramine: Adult, 25 to 50 mg PO q6h; child, 4 to 6 mg/kg/24 hr ÷ q6 to 8 (max 200 mg). Also available as a topical (lotion).
- Hydroxyzine: Adult, 25 to 100 mg PO q8h; child, 2 to 4 mg/kg/24 hr ÷ q8 to 12 (max 200 mg).
- Cetirizine/loratadine/fexofenadine.

Antibacterials (Topical)

- Mupirocin: Used for impetigo.
- Bacitracin: Used for burns and cuts.
- Neomycin/polymixin B: Used for cuts.
- Silver sulfadiazine: Used for burns.

Antifungals (Topical)

- Polyenes: Nystatin, amphotericin B 3%.
- Imidazoles: Ketoconazole 1%, clotrimazole 1%, miconazole 2%, econazole 1%.

Antiparasitics (Topical)

- Lindane (for age > 1 year):
  - Lice: 1% shampoo 30 mL for 4 minutes, then rinse thoroughly; again after 5 days.
  - Scabies: Lotion 30 to 60 mL and wash after 8 hours.
- Permethrin (for age > 2 months):
  - Lice: 1% rinse—wash after 10 minutes, again after 5 days for next generation.
  - Scabies: 5% cream all over and wash thoroughly after 8 to 12 hours.

Antivirals

Topical
- Acyclovir: 5% ointment q3h × 7 days.
- Used for herpes (varicella, zoster, simplex, and genitalis).

- If it's wet . . . dry it.
- If it's dry . . . wet it.
- If present, remove offending agent.

Absorption of topical antihistamines is unpredictable.

Warn patients about drowsiness associated with antihistamines.

Lindane can induce seizure if overused.

Oral

- Acyclovir:
  - Genitalis: 400 mg PO tid × 10 days
  - Zoster: 800 mg PO 5/day × 7 to 10 days
  - Varicella: 20 mg/kg PO qid × 5 days—max 800 mg
- Famciclovir:
  - Genitalis: 250 mg PO tid × 10 days
  - Zoster: 500 mg PO tid × 7 days
- Valacyclovir:
  - Genitalis: 1 g PO bid × 10 days
  - Zoster: 1 g PO tid × 7 days

Corticosteroids (Topical)

- Potency graded on ability to vasoconstrict: High potency, group I; low potency, group VII.
- Avoid using groups I, II, III, and IV in pregnancy, infancy, face, genitalia, flexure creases, and intertriginous areas.
- Bid–tid therapy for 1 to 2 weeks (potent), or 2 to 4 weeks (less potent).

Acne

Topical
- Benzoyl peroxide: Many preparations
- Clindamycin: 10 mg/mL bid
- Tretinoin: Many preparations
- Erythromycin: 1.5 to 2% bid

Oral
- Tetracycline: 250 mg PO qid
- Doxycycline: 100 mg PO bid
- Isotretinoin: 0.5 mg/kg PO bid

▶ **CUTANEOUS BACTERIAL INFECTIONS**

Abscess

- Location: Back, buttocks, axillae, groin, and anywhere pus can accumulate (acne, wounds).
- Definition: Pocket of pus from skin flora.
- Signs and symptoms: Red, hot, swollen, and tender.
- Treatment: Incise, drain, pack, wound check (see Procedures chapter).

Cellulitis

- Location: Commonly lower extremities (diabetics and peripheral vascular disease), but can be anywhere.
- Definition: Superficial skin and subcutaneous tissue infection from skin flora *(Staphylococcus aureus, Streptococcus pyogenes)*.
- Signs and symptoms: Red, hot, swollen, and tender.
- Treatment: Outpatient oral antibiotics in the young and healthy (first-generation cephalosporin) or inpatient intravenous (IV) antibiotics in the frail/elderly.

The thicker the skin, the more potent steroid needed.

Topicals are first-line agents for treatment of acne.

Tretinoin and isotretinoin are teratogenic.

Exposure to sun while on tetracycline or doxycycline can cause a rash.

A 55-year-old diabetic man presents with a right lower extremity that is red, warm, and tender to the touch. The rash has poorly demarcated borders and has been spreading over the last day. He is febrile to 101°F. *Think: Cellulitis.*

Erysipelas, erysipeloid, and necrotizing fasciitis are variants of cellulitis.

Impetigo

- Location: Face (usually), can be anywhere (Figure 16-14).
- Definition: Bacterial superinfection of epidermis from broken skin. Bullous *(S. aureus)* and nonbullous (group A beta-hemolytic strep [GAS]).
- Signs and symptoms:
  - Initial lesion is a transient erythematous papule or thin-roofed vesicle that ruptures easily and forms a "honey-colored" crust.
  - Lesions can be discrete and scattered or become confluent, forming a superficial crusted plaque.
- Treatment:
  - Remove crusts by soaking in warm water.
  - Antibacterial washes (benzoyl peroxide).
  - Topical antibiotic if disease is limited (mupirocin).
  - Oral antibiotics (e.g., first-generation cephalosporin) for less mild cases.

Erysipelas

- Location: Face (most often), extremities (Figure 16-15).
- Definition: An acute onset of superficial spreading cellulitis, arising in inconspicuous breaks in skin; involves dermis and epidermis. Pathogens include *S. aureus*, GAS, and occasionally *Haemophilus influenzae*.
- Signs and symptoms: An erythematous, shiny area of warm and tender skin with a well-demarcated and indurated advancing border.
- Less edematous than cellulitis, but margins are more sharply demarcated and elevated.
- Face is most commonly involved, but can affect any area, especially sites of chronic edema.
- Treatment: Same as for cellulitis of the face (IV antibiotics), with care taken if orbital involvement to consult ophthalmologist.

Lyme Disease

- Location: Usually affects the trunk, proximal extremities, axilla, and inguinal area.

Complications of GAS impetigo include acute glomerulonephritis and guttate psoriasis. If untreated, patients can develop cellulitis, lymphangitis, and septicemia.

A 67-year-old woman presents with an erythematous, shiny area of warm and tender skin on her face with a well-demarcated and indurated advancing border. *Think: Erysipelas.*

F I G U R E   1 6 - 1 4 .   Impetigo.

(Reproduced, with permission, from Pantell R, et al. *The Common Symptom Guide*, 4th ed. New York: McGraw-Hill, 1996.)

HIGH-YIELD FACTS

Dermatologic Emergencies

F I G U R E   1 6 - 1 5 .   Erysipelas.

(Reproduced, with permission, from Fauci AS, et al. *Harrison's Principles of Internal Medicine,* 14th ed. New York: McGraw-Hill, 1997.)

- Definition: Chronic multisystem infection with spirochete *Borrelia burgdorferi* transmitted through *Ixodes* ticks in Atlantic and northeast states. Rash is erythema chronicum migrans (ECM), a spreading, annular, macular erythema seen 2 to 20 days from site of tick bite.
- Signs and symptoms: Mild–moderate itch, mild burn. If untreated, progresses with systemic illness (arthralgias, fever, adenopathy, flulike symptoms, facial nerve palsies, cranial neuritis, myocarditis, cardiac conduction abnormalities, encephalopathy, polyneuropathy, more rash).
- Treatment: Doxycycline 100 mg PO bid × 21 days (or clarithromycin, amoxicillin, cefuroxime).

Rocky Mountain Spotted Fever (RMSF)

- Location: Wrists and ankles (acral rash), then spreads to trunk (Figure 16-16).
- Definition:
  - A potentially life-threatening disease due to a tick bite, with highest incidence in children aged 5 to 10 years old.
  - Ninety-five percent of cases occur from April through September.
  - Occurs only in the western hemisphere, primarily in southeastern states and most often in Oklahoma, North and South Carolina, and Tennessee.
  - Rarely occurs in the Rocky Mountains.
  - Only 60% of patients report a history of a tick bite.
- Signs and symptoms:
  - *Rickettsia rickettsii* through tick bite invades bloodstream and causes blanching, maculopapular lesions that become petechial and can coalesce and become ecchymotic or gangrenous.
  - Sudden onset of high **fever,** myalgia, severe headache, rigors, nausea, and photophobia within first 2 days of tick bite.
  - Fifty percent develop rash within 3 days. Another 30% develop the rash within 6 days.
  - Rash consists of 2- to 6-mm pink **blanchable macules** that first appear peripherally on wrists, forearms, ankles, palms, and soles.
  - Within 6 to 18 hours the exanthem spreads centrally to trunk, proximal extremities, and face.

F I G U R E   1 6 - 1 6 .   Late Manifestation of RMSF.

Note disseminated macules and papules. Initial lesions were noted on the palms and soles, wrists, and ankle and extended centripetally (Reproduced, with permission, from Fitzpatrick TB, et al. *Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases,* 4th ed. New York: McGraw-Hill, 2001: 807.)

- Within 1 to 3 days, the macules evolve to deep red papules, and within 2 to 4 days, the exanthem is hemorrhagic and no longer blanchable.
- Up to 15% have no rash.
- Treatment: Doxycycline 100 mg PO bid × 14 days. Use chloramphenicol for pregnant patients and children under age 8.

Scarlet Fever

- Location: Diffuse.
- Definition: A toxin-mediated disease caused by GAS.
- Signs and symptoms:
  - Pain precedes rash and follows fever.
  - A finely punctate pink-scarlet exanthem first appears on upper trunk 12 to 48 hours after onset of fever.
  - As the exanthem spreads to extremities, it becomes confluent and feels like sandpaper and fades within 4 to 5 days, followed by desquamation.
  - Linear petechiae evident in body folds (Pastia's sign).
  - Pharynx is beefy red and tongue is initially white, but within 4 to 5 days, the white coating sloughs off and tongue becomes bright red ("strawberry tongue").
- Treatment: Oral penicillin or erythromycin, acetaminophen.

The sandpaper rash is typical of scarlet fever.

Complications of untreated scarlet fever include:
- Acute rheumatic fever
- Acute glomerulonephritis
- Erythema nodosum

A 20-year-old college student has a low-grade fever, chills, and migratory polyarthralgias accompanied by a tender rash. The rash initially consisted of erythematous macules that have now evolved into hemorrhagic pustules. *Think: Disseminated gonococcal infection.*

Complications of meningococcemia include meningitis and Waterhouse–Friderichsen syndrome (fulminant meningococcemia with adrenal hemorrhage).

Gonococcemia

- Location: Anywhere.
- Definition: Emboli of disseminated *Neisseria gonorrhoeae*, usually in menstruating or peripartum females. Looks like multiple papular, vesicular, and pustular petechial lesions with erythematous base that become hemorrhagic; associated fever and arthralgias.
- Signs and symptoms: Painful.
- Treatment: IV ceftriaxone or ciprofloxacin.
- Risk factors: Third-trimester pregnancy, postpartum period, 7 days within menses onset.

Meningococcemia

- Location: Extremities and trunk (anywhere) (Figure 16-17).
- Definition: Infectious vasculitis from emboli of disseminated *Neisseria meningitidis*, usually in age < 20, sometimes in epidemics.
- Signs and symptoms:
  - Petechia, urticaria, hemorrhagic vesicles, macules, and papules with surrounding erythema.
  - Associated with fever, altered mental status and vitals, headache, arthralgias, and stiff neck.
- Treatment: IV ceftriaxone, add vancomycin for cephalosporin-resistant pneumococcus. Admit to the hospital.

Toxic Shock Syndrome

- Location: Diffuse or just extremities/trunk.
- Definition: Severe, life-threatening, multisystem syndrome arising because of *S. aureus* toxic shock syndrome toxin (TSST-1) in menstruat-

F I G U R E   1 6 - 1 7.   Meningococcemia.

(Reproduced, with permission, from Knoop KJ, Stack LB, Storrow AB. *Atlas of Emergency Medicine.* New York: McGraw-Hill, 1997: 404.)

ing women using tampons, or enterotoxins B and C also from *Staphylococcus* but unrelated to tampon use.
- Signs and symptoms:
    - Nonpruritic, tender erythroderma.
    - **Fever, hypotension,** diffuse tender erythroderma, followed by desquamation, mucosal hyperemia (three of four must be present).
    - Erythema may resolve in 3 to 5 days with subsequent desquamation of hands and feet in 5 to 14 days.
- Treatment: Hospital admission, aggressive IV fluid resuscitation, IV oxacillin or cefazolin, vancomycin if penicillin allergic.
- In addition to major criteria, there must be evidence of multisystem involvement such as altered mental status, heart failure, adult respiratory distress syndrome (ARDS), diarrhea, renal insufficiency, thrombocytopenia, or arthralgias.

► **FUNGAL INFECTIONS**

Candida

- Location: Can be on mucous membranes (palate, pharynx, tongue, vagina) or can be cutaneous (intertriginous, groin, under fat pannus).
- Definition: *Candida albicans*, normally a nonpathogenic colonizer of moist skin and mucosa, causes painful, raised, whitish plaques that detach and leave red erosions.
- Signs and symptoms: Painful, pruritic in vagina.
- Treatment: Oral nystatin swish and swallow 5 mL tid for oral lesions; topical nystatin or clotrimazole cream for cutaneous and vaginal types. Fluconazole 150 mg PO single dose for both oral and vaginal types.

F I G U R E 1 6 - 1 8 . Tinea corporis.

(Reproduced, with permission, from Pantell R, et al. *The Common Symptom Guide*, 4th ed. New York: McGraw-Hill, 1996.)

■ Risk factors: Obesity, systemic antibiotics, corticosteroid or chemotherapy, urinary or fecal incontinence, and immunocompromised states.

Tinea Infection

> All tinea infections except tinea capitis are treated with a topical antifungal cream.

Tinea are fungal infections caused by *Trichophyton* and *Microsporum* species and are named according to the part of the body they are on:
- Tinea cruris: Groin, gluteal cleft.
- Tinea pedis: Feet, in between toes (athlete's foot).
- Tinea versicolor: On trunk, multiple-colored lesions that do not tan with surrounding skin in sunlight.
- Tinea corporis: See Figure 16-18.
- Tinea capitis: Invade hair shafts and surrounding skin; causes red, circular patches with raised edges, sometimes swollen, boggy, and crusted, with loss of hair:
    - Treatment: Griseofulvin 7.5 mg/kg PO bid × 6 weeks.

▶ **PARASITIC INFECTIONS**

Pediculosis (Lice)

- Location: *Phthirus capitis* on scalp and neck. *Phthirus pubis* (crabs) in pubic hair.
- Definition: *P. capitis* mite lives on scalp and lays eggs (nits) on hair shafts; lives on human blood.
- Signs and symptoms: Severe itch.
- Treatment: Permethrin, then lindane, fine-toothed comb to manually remove nits.

Scabies

- Location: Flexural creases, hands, feet.
- Definition: *Sarcoptes scabiei,* the "itch mite," burrows into the skin and lays its eggs.

F I G U R E 1 6 - 1 9 . Scabies.

Notice the papulovesicular nature of the rash, which tends to occur in places where mites can burrow, such as the web spaces of the digits and the axilla, as shown here. (Reproduced, with permission, from Rudolph A, et al. *Rudolph's Pediatrics*, 20th ed. Stamford, CT: Appleton & Lange, 1997.)

- Signs and symptoms: Intense itch and mild burning; excoriations and pruritic red papules (Figure 16-19).
- Treatment: Lindane or permethrin.

Varicella Zoster: Shingles

- Location: Commonly thoracic and facial dermatomes.
- Definition: An acute dermatomal viral infection caused by reactivation of latent varicella-zoster virus that has remained dormant in a sensory root ganglion. The virus travels down the sensory nerve, resulting initially in dermatomal pain, followed by skin lesions.
- Risk factors: Age, malignancy, immunosuppression, and radiation.
- Signs and symptoms:
  - Prodrome of pain, burning, itching, and paresthesia in affected dermatome precedes eruption by 3 to 5 days.
  - Accompanied by fever, headache, and malaise, and heightened sensitivity to stimuli (allodynia).
  - Grouped vesicles on an erythematous base distributed unilaterally along a dermatome (Figure 16-20).
  - Crust formation within 5 to 10 days.
  - Some vesicles may occur outside of involved dermatome.
- Treatment:
  - Moist and cool compresses to affected dermatome.
  - Oral acyclovir, valacyclovir, or famciclovir (accelerate healing of lesions and decrease duration of pain if started within 3 days of infection).
  - Analgesics.

FIGURE 16-20. Varicella-zoster infection.

Note dermatomal distribution (T8–10) to rash. (Reproduced, with permission, from Fitzpatrick TB, et al. *Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases*, 4th ed. New York: McGraw-Hill, 2001: 807.)

A 27-year-old human immunodeficiency virus (HIV)-positive patient presents due to an intensely painful erythematous rash that is over his right flank in a dermatomal distribution. *Think: Varicella-zoster.*

Patients with zoster can infect nonimmune contacts with chickenpox. Exposed nonimmune contacts should be treated with varicella-zoster immune globulin.

Varicella-zoster infection of cranial nerve VIII is called Ramsay Hunt syndrome and results in hearing loss, vertigo, and tinnitus.

Varicella-zoster infection of V_1 (ophthalmic branch of trigeminal nerve) can be vision threatening. Ophthalmic consultation should be obtained for these cases.

F I G U R E 1 6 - 2 1 . **Molluscum contagiosum.**

(Reproduced, with permission, from Seltzer V, Pearse WH. *Women's Primary Health Care: Office Practice and Procedures*, 2nd ed. New York: McGraw-Hill, 2000.)

Molluscum Contagiosum

- Location: Anywhere, typically head and neck.
- Definition: A self-limited contagious poxvirus infection transmitted by direct contact and characterized by an umbilicated "pearly" papule; commonly seen with HIV.
- Signs and symptoms:
  - Mild pruritus.
  - Shiny, umbilicated, slightly translucent skin or flesh-colored papules (Figure 16-21); slow growing, < 10 mm diameter, sometimes grouped.
- Treatment: Cryotherapy, surgical excision, or wait it out; resolves in 12 to 18 months.

Verruca (Warts)

There is an increased incidence of verrucae in atopic and immunocompromised patients.

- Location: Anywhere, typically hands.
- Definition: Human papillomavirus (common warts—hard, rough, skin-colored papules) and others (plantar warts—bottom of foot; plane warts, also called flat warts; mosaic warts) caused by different viruses.
- Signs and symptoms:
  - Initial lesion is skin colored with a smooth surface.
  - As lesion enlarges with time, the surface becomes roughened and papillomatous.
  - Several types of warts exist and are named according to their location.
- Treatment:
  - Cryotherapy with liquid nitrogen or carbon dioxide (requires multiple treatments every 2 to 3 weeks and is painful).
  - Topical application of keratolytic agents (salicylic acid and lactic acid) and destructive agents (podophyllin or cantharidin).
  - Curettage and desiccation.
  - Topical imiquimod (an immune response modifier that stimulates the immune system to fight the virus).
  - Or wait it out; resolves in 2 to 3 years.

Angioedema

- Location: Face (tongue, lips, larynx, more), anywhere.
- Definition:
  - Immunologic (associated with food, cold, insect venom, pollen) mechanism is immunoglobulin E (IgE)–antigen complex triggered massive histamine release.
  - Nonimmunologic (associated with angiotensin-converting enzyme [ACE] inhibitors, contrast dye) mechanism not well understood.
- Signs and symptoms: Warm; itchy; difficulty breathing, talking, and swallowing due to airway edema.
- Treatment:
  - ABCs.
  - For stridor, wheezing, or low SaO_2:
    - Subcutaneous (SQ) epinephrine.
    - Albuterol nebulizer.
    - Consider early intubation (patient's airway can be rapidly lost due to significant airway edema).
  - IV methylprednisolone.
  - IV diphenhydramine (H_1 blocker).
  - Some people give IV H_2 blocker (thought to provide some cross-reactive antihistamine benefit).
  - Admit for observation.

Urticaria

- Location: Anywhere.
- Definition: An immunologic reaction that results in mast-cell degranulation of histamine, causing localized capillary and postcapillary venule leak of proteinaceous fluid that is gradually resorbed. Histamine also causes vasodilation, giving localized erythema and classic wheal appearance. Types include immune type I (IgE) and type III immune-complex IgG and IgM (drugs, pollen, dust, animal dander) and non–immune mediated (cold, pressure, heat, cholinergic, dermatographism, strawberries).
- Signs and symptoms:
  - Characterized by **wheals:** An abrupt development of transient, edematous, pink papules and plaques that may be localized or generalized and are usually pruritic.
  - Wheals may develop after exposure to circulating antigens (drugs, food, insect venom, animal dander, pollen), hot and cold temperatures, exercise, and pressure or rubbing (dermatographism).
  - Wheals usually last < 24 hours and may recur on future exposure to the antigen.
- Treatment:
  - Antihistamines (H_1 and H_2 blockers).
  - SQ epinephrine if anaphylactic/impending associated airway compromise.
  - PO or IV corticosteroids if severe.
  - Observation.
  - Supportive therapy.

Bullous Pemphigoid

- Location: Anywhere (skin and mucosa—usually oral).
- Definition: Autoimmune disorder with immunoglobulin G (IgG) antibodies to the dermoepidermal junction giving vesicles and bullae that lyse and yield erosions.
- Signs and symptoms:
  - Occasional pruritus and tenderness.
  - Large, erythematous urticarial plaques may precede bullae by months.
  - Multiple, intact, tense bullae become crusted after rupturing.
  - Bullae can be localized or generalized, primarily distributed on flexural areas of axilla, groin, medial thighs, forearms, and lower legs (Figure 16-22).
  - Only one third of patients have oral involvement.
- Treatment: Oral/IV steroids; consult dermatologist for management.

Eczema

- Location: Extremities (usually).
- Definition:
  - Also called dry skin; from loss of epidermal lipids by excessive washing or decrease in production (elderly), causing flaking and cracking.
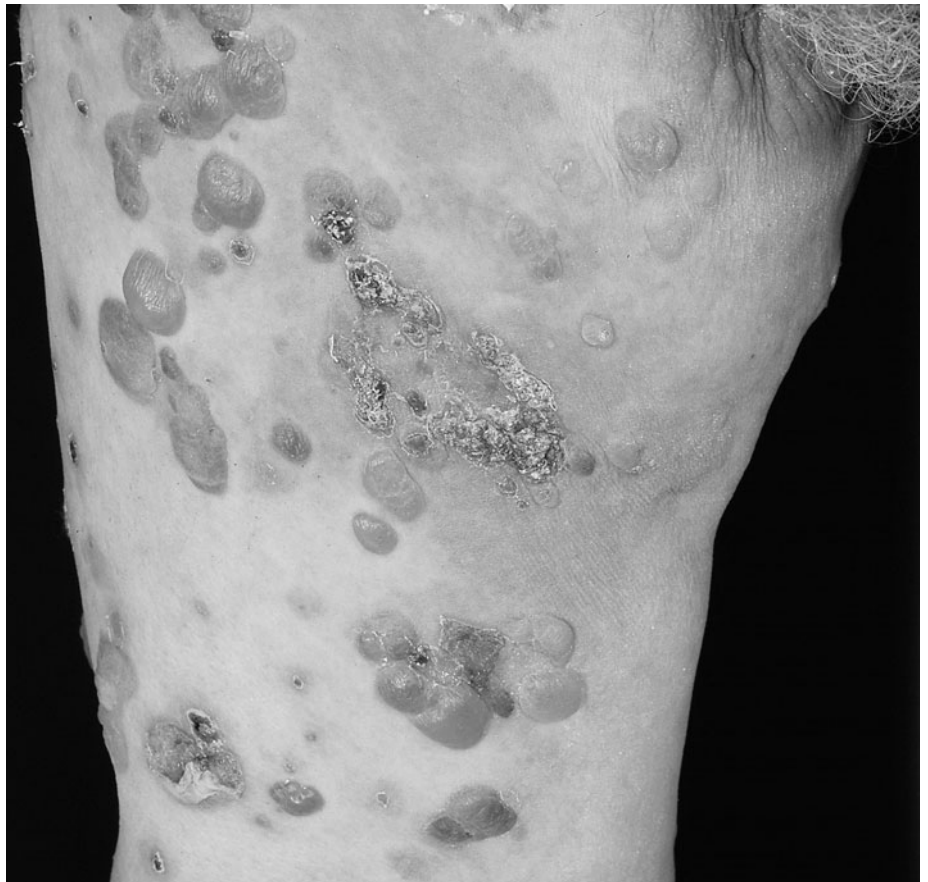
F I G U R E   1 6 - 2 2 .   Bullous pemphigoid.

(Reproduced, with permission, from Fitzpatrick TB, et al. *Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases*, 4th ed. New York: McGraw-Hill, 2001: 100.)

- *Eczema* is a broad term used to describe several inflammatory skin reactions and is used synonymously with dermatitis. Eczema is an inherited skin condition with a discrete classification system (atopic, contact, allergic, stasis, or seborrheic).
- Signs and symptoms:
    - Lesions can be described as acute or chronic.
    - Acute lesions are red, blistery, and oozy.
    - Chronic lesions are thickened, lichenified, and pigmented.
- Treatment: Decrease frequency of washing or use moisturizer after each washing.

Areas involved in eczema:
FACE
Flexor surfaces = **A**dults
Children = **E**xtensor surfaces

Psoriasis

- Location: Elbows, knees, scalp, gluteal cleft, nails, palms, soles.
- Definition: Inherited disorder in which the keratinocyte life cycle is shortened (i.e., rapid cell turnover) looking like erythema, scaly silvery plaques, fissures, and nail plate separation from nail bed with pitting of nails. Variant exists with pustules on palms and soles, minimal scale.
- Signs and symptoms:
    - Well-demarcated, thick, "salmon-pink" **plaques** with an adherent silver-white scale (Figure 16-23).
    - Distributed bilaterally over **extensor surface** of extremities, often on elbows, knees, trunk, and scalp.
    - Nails are commonly involved: Pitting of nails, oil spots (yellow-brown spots under nail plate), onycholysis (separation of distal nail plate from nail bed), subungual hyperkeratosis (thickening).
    - Can occur at site of injury (Koebner phenomenon).
    - Pinpoint capillary bleeding occurs if scale is removed (Auspitz sign).
- Treatment: Tar emulsion (1 tsp in quart of water) applied bid followed by group I topical steroid cream, moisturizer creams.

Psoriasis is worse in winter.

A 35-year-old man has salmon-colored papules covered with silvery white scales on his scalp, elbows, and knees. *Think: Psoriasis.*

Erythema Multiforme (EM)

- Location: Palms, soles, extremities, anywhere.
- Definition: Immune complex–mediated (IgM, C3) vasculitis of blood vessels at dermo-epidermal junction that give rise to multiple pink-red, target-shaped bullae and papules of varying sizes; most commonly due to drugs, infections, x-ray therapy, malignancy, rheumatologic disease, pregnancy, and unknown etiology.

Fifty percent of cases are idiopathic, but herpes simplex virus accounts for most cases of recurrent EM.
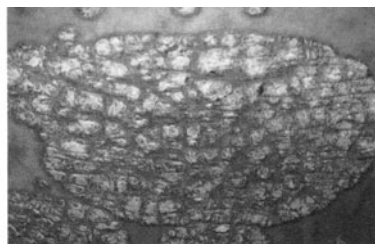
FIGURE 16-23. Psoriasis.

(Reproduced, with permission, from Rudolph A, et al. *Rudolph's Pediatrics*, 20th ed. Stamford, CT: Appleton & Lange, 1997.)

- Most commonly seen in 20- to 40-year-old age group.
- Signs and symptoms:
  - Viral-like prodrome may precede eruption.
  - Lesions itch and burn, may lyse yielding erosions.
  - Although characterized by target lesions, multiforme refers to the wide variety of lesions that may be present, including papules, vesicles, and bullae (Figure 16-24).
  - Ocular involvement in 10% of cases.
- Treatment:
  - Cessation of medication (if possible).
  - Antihistamines for itch.
  - Wet-to-dry dressings with topical bacitracin for erosions.
  - Look for underlying cause.
  - Supportive care.

Stevens–Johnson Syndrome (SJS)

- Location: Mucous membranes, conjunctiva, respiratory tract, various areas of skin.

FIGURE 16-24. Erythema multiforme.

Note the many different-sized lesions. (Courtesy of Michael Redman, PA-C. Reproduced, with permission, from Knoop KJ, Stack LB, and Storrow AB. *Atlas of Emergency Medicine*. New York: McGraw-Hill, 1997.)

- Definition: Bullous variant of EM most often secondary to medication (sulfonamides, barbiturates, phenytoin, carbamazepine, thiazide diuretics, penicillins) or infection (upper respiratory infections, gastroenteritis, mycoplasma, herpes simplex virus).
- Signs and symptoms:
  - Viral-like prodrome precedes skin and mucosal lesions, which are itchy, burning, red-pink, target-shaped bullae, lysing to give erosions (Figure 16-25).
  - Bullous target lesions often less than 10% of epidermis.
  - High morbidity and mortality.
  - Ocular involvement present in 75% of cases.
- Treatment:
  - Hospital admission may be required.
  - Antihistamines for the itch.
  - Corticosteroid (IV/oral) use is controversial, with most favoring its use.
  - Removal of offending medication if possible.
  - Soft/liquid diet.
  - Eroded lesions treated with wet-to-dry dressings and topical bacitracin.

Toxic Epidermal Necrolysis (TEN)

- Location: Everywhere, with mucosal involvement.
- Definition: EM variant that is a true emergency from lysis of 30 to 100% of epidermis at dermal junction caused by similar things that cause EM/SJS; mortality high.

> SJS and toxic epidermal necrolysis are severe variants of EM that are potentially life threatening.

> *Nikolsky's sign:* Sloughing of epidermis with light pressure over lesion.

F I G U R E   1 6 - 2 5 .   Stevens–Johnson syndrome.

Note involvement of oral mucous membranes. (Reproduced, with permission, from Knoop KJ, Stack LB, Storrow AB. *Atlas of Emergency Medicine.* New York: McGraw-Hill, 1997: 343.)

- Signs and symptoms:
  - Prodrome of fever and influenza-like symptoms.
  - Pruritus, pain, tenderness, and burning.
  - Classic target-like lesions symmetrically distributed on dorsum of hands, palms, soles, face, and knees.
  - Initial **target lesions** can become confluent, erythematous, and tender, with bullous formation and subsequent loss of epidermis.
  - Epidermal sloughing may be generalized, resembling a second-degree burn, and is more pronounced over pressure points (Figure 16-26).
  - Positive Nikolsky's sign.
  - Ninety percent of cases have mucosal lesions—painful, erythematous erosions on lips, buccal mucosa, conjunctiva, and anogenital region.
- Treatment:
  - Hospital admission (usually to burn unit).
  - Wounds treated as second-degree burns.
  - Avoid steroid use.
  - Studies suggest that plasmapheresis or exchange, hyperbaric O_2, and cyclosporine can decrease extent of disease and facilitate healing.
- Prognosis:
  - Mortality rate as high as 30%.
  - Typical causes of death include hypovolemia, infection, and electrolyte disturbances (as would be expected for widespread burns).

Erythema Nodosum

- Location: Shins, lower extremities (Figure 16-27).
- Definition: Hypersensitivity vasculitis of venules in subcutaneous tissue from drugs (sulfonamides, oral contraceptives), infections (tuberculosis,

F I G U R E   1 6 - 2 6 .   Toxic epidermal necrolysis.

Note the generalized macular eruption and large denuded erosive area. (Reproduced, with permission, from Fitzpatrick TB, et al. *Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases*, 4th ed. New York: McGraw-Hill, 2001: 141.)
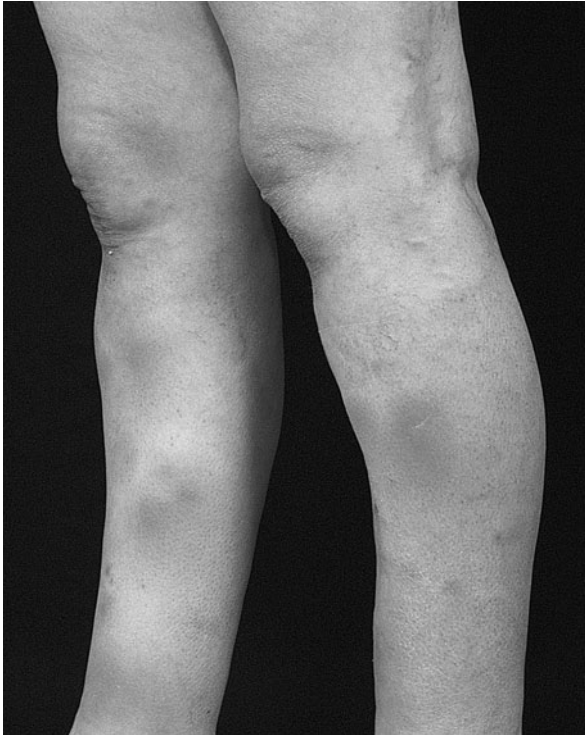
F I G U R E   1 6 - 2 7 .   Erythema nodosum.

Note indurated, very tender inflammatory nodules mostly over pretibial region. Palpable as deep nodules. (Reproduced, with permission, from Fitzpatrick TB, et al. *Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases*, 4th ed. New York: McGraw-Hill, 2001: 145.)

Streptococcus spp., coccidioidomycosis), or systemic disease (sarcoidosis, inflammatory bowel disease, lymphoma, leukemia) and look like red subcutaneous nodules with surrounding erythema; can last 6 weeks.

- Signs and symptoms:
  - Painful and tender lesions accompanied by fever, malaise, and arthralgias.
  - +/– Regional adenopathy.
- Treatment: Cessation of medication (if possible), nonsteroidal anti-inflammatory drugs for pain, look for underlying etiology.

Henoch-Schönlein Purpura (HSP)

- Location: Lower legs and buttocks.
- Definition: IgA immune complex vasculitis involving arterioles and capillaries caused by drugs, infections, foods, immunizations, and insect bites; usually a childhood disorder.
- Signs and symptoms:
  - Purplish raised papules and "palpable purpura."
  - Arthralgias.
  - Gastrointestinal complaints (nausea, vomiting, diarrhea, abdominal pain—70%).
  - Renal involvement (hematuria, red blood cell [RBC] casts—50%).

A 5-year-old child presents with a palpable purpura over his buttocks and back of his legs and also complains of joint pains and nausea. *Think: HSP.*

359
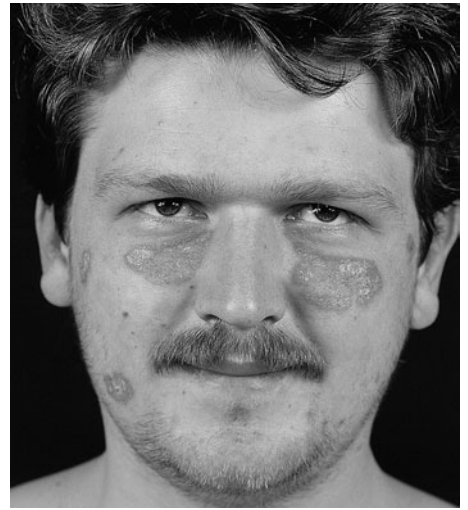
A   **B**

F I G U R E   1 6 - 2 8 .   Malar (A) and discoid (B) rashes of systemic lupus erythematosus.

(Reproduced, with permission, from Fitzpatrick TB, et al. *Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases*, 4th ed. New York: McGraw-Hill, 2001: 363, 364.)

- Treatment:
  - Admit for IV steroids if renal involvement.
  - Otherwise, discharge home on PO prednisone 1 mg/kg/day, and remove the offending agent if possible.

Systemic Lupus Erythematosus (SLE)

- Location: Face (malar rash), widespread (discoid) (Figure 16-28).
- Definition: Multisystem anti–double-stranded DNA autoantibody-mediated inflammatory disorder.
- Signs and symptoms: Systemic symptoms include fever, arthralgia, pneumonitis, nephritis, pericarditis, and vasculitis.
- Treatment: Systemic steroids and immunosuppressive therapy for flare-ups.

▶ **MISCELLANEOUS RASHES**

Pityriasis Rosea

- Location: Chest or back or both.
- Definition: A common self-limiting eruption of a single herald patch followed by a generalized secondary eruption within 2 weeks; ages 10 to 35 commonly.
- Signs and symptoms:
  - A 2- to 10-cm solitary, oval, erythematous "herald patch" with a collarette of scale precedes the generalized eruption in 80% of patients.
  - Within days, multiple, smaller, pink, oval, scaly patches appear over trunk and upper extremities.
  - Secondary eruption occurs in a Christmas tree distribution, oriented parallel to the ribs (Figure 16-29).
- Treatment: Antihistamines for itch.

A 21-year-old male presents with a pruritic, spotted rash on the trunk that began as one solitary larger patch. *Think: Pityriasis rosea.*
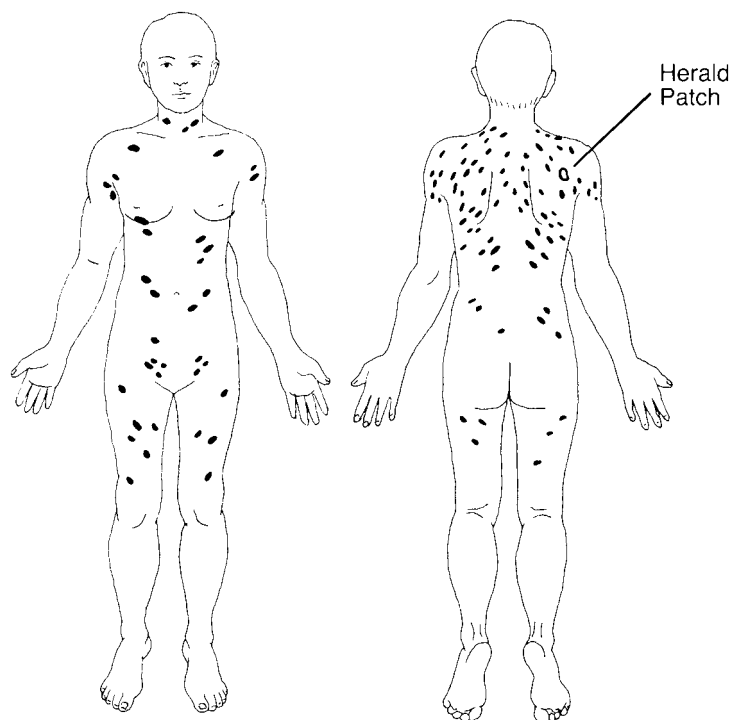
FIGURE 16-29. Distribution of pityriasis rosea.

Note "Christmas tree" pattern of rash and location of herald patch. (Reproduced, with permission, from Fitzpatrick TB, et al. *Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases*, 4th ed. New York: McGraw-Hill, 2001: 107.)

Seborrheic Dermatitis

- Location: Skin folds and hair-bearing areas of face, scalp, chest, groin.
- Definition: Waxy, erythematous scale possibly related to yeast *(Pityrosporum ovale)*; "cradle cap" in infancy.
- Signs and symptoms: Mild itch.
- Treatment: Zinc pyrethrin (Head and Shoulders), selenium sulfide (Selsun Blue), or tar (Neutrogena T-Gel) shampoo—lathered for 10 minutes, then rinsed, 3 times a week; 1 to 2.5% hydrocortisone cream for face.

Decubitus Ulcers

- Location: Over bony prominences (sacrum, ischial tuberosities, iliac crests, greater trochanters, heels, elbows, knees, occiput).
- Definition: Any pressure-induced ulcer that occurs secondary to external compression of the skin, resulting in ischemic tissue necrosis (i.e., bedsore, pressure ulcer). Early ulcers have irregular, ragged borders, but chronic ulcers have smooth, well-demarcated borders. Infection is usually polymicrobial: *S. aureus, Streptococcus, Pseudomonas, Enterococcus, Proteus, Clostridium,* and *Bacteroides.*
- Signs and symptoms: Painful, ulcerated.
- Treatment:
  - *Prophylaxis*:
    - Mobilize patients as soon as possible.
    - Reposition patients every 2 hours.

Risk factors for decubitus ulcers:
- Immobility, fracture
- Malnutrition
- Age > 70
- Hypoalbuminemia
- Spinal cord injury
- Fecal incontinence
- Diabetes mellitus
- Inadequate nursing care
- Decreased level of consciousness

- Pressure-reducing devices (foam, air, or liquid mattresses).
- Correction of nutritional status.
  - *Local wound care:*
    - Proper cleansing with mild agents.
    - Moisturize to maintain hydration and promote healing.
    - Polyurethane, hydrocolloid, or absorptive dressings, and topical antibiotics for wound.
    - Necrotic tissue may require surgical debridement, flaps, and skin grafts.
    - Appropriate antibiotic therapy for infected ulcer.

► MALIGNANT GROWTHS

Basal Cell Carcinoma (Rodent Ulcer)

- Location: Sun-exposed areas (forehead, nose).
- Definition: Slow-growing proliferation of basal keratinocytes.
- Signs and symptoms:
  - Asymptomatic, rarely painful.
  - Flesh-colored or hyperpigmented nodule with surface telangiectasia that expands outward leaving central ulcer and "rolled" raised edge (Figure 16-30).
- Treatment: Surgical excision.

Kaposi's Sarcoma

- Location: Anywhere (skin and mucosa).
- Definition: Multisystem vascular neoplasm characterized by mucocutaneous violaceous lesions, commonly seen in acquired immune deficiency syndrome patients.
- Signs and symptoms: Cutaneous, nonblanching, reddish-purple macules, plaques, and nodules made of vasoformative tissue (spindle cells, vascular spaces, hemosiderin-stained macrophages, extravasated RBCs) (Figure 16-31).

F I G U R E 1 6 - 3 0 . Basal cell carcinoma.

Note the translucent nature of the lesion. (Reproduced, with permission, from Seltzer V, Pearse WH. *Women's Primary Health Care: Office Practice and Procedures,* 2nd ed. New York: McGraw-Hill, 2000.)

FIGURE 16-31. Kaposi's sarcoma.

Note multiple purplish confluent papules, often mistaken for bruising. (Reproduced, with permission, from Fitzpatrick TB, et al. *Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases*, 4th ed. New York: McGraw-Hill, 2001: 527.)

- Treatment: Radiation therapy (if limited disease), chemotherapy, and radiation (if disseminated—palliative).

Melanoma

- Location: Anywhere.
- Definition: A malignant proliferation of melanocytes (> 10 mm diameter, crusting or inflammation, change in size, color, contour, texture, or sensation) (Figure 16-32).
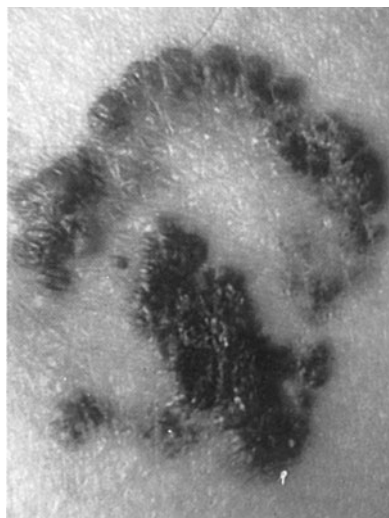
FIGURE 16-32. Melanoma.

(Reproduced, with permission, from Pantell R, et al. *The Common Symptom Guide*, 4th ed. New York: McGraw-Hill, 1996.)

F I G U R E 1 6 - 3 3 . Squamous cell cancer.

(Reproduced, with permission, from Pantell R, et al. *The Common Symptom Guide*, 4th ed. New York: McGraw-Hill, 1996.)

- Signs and symptoms: May itch or lose sensation.
- Treatment: Wide surgical excision.

Squamous Cell Carcinoma

- Location: Sun-exposed areas (face, neck, forearms).
- Definition: Malignant proliferation of epidermal keratinocytes, sometimes locally invasive; expanding nodular plaque, with indurated base and central ulcer with crust/scale (Figure 16-33).
- Signs and symptoms: A cut that won't heal, bleeds easily.
- Treatment: Surgical excision.

Procedures

HIGH-YIELD FACTS

Procedures

DEFINITION

Tube thoracostomy, commonly called a chest tube, is used to remove air or fluid from the pleural space.

INDICATIONS

- Pneumothorax
- Hemothorax
- Hemopneumothorax
- Open pneumothorax (sucking chest wound)
- Drainage of recurrent pleural effusion
- Empyema
- Chylothorax

RELATIVE CONTRAINDICATIONS

- Multiple adhesions.
- Need for thoracotomy.
- Recurrent pneumothorax requiring definitive treatment.
- Severe coagulopathy.

PROCEDURE (MIDAXILLARY LINE PLACEMENT)

1. Elevate the head of the bed at least 30 degrees to reduce the chances of injury to abdominal organs.
2. Identify the fourth intercostal space in the midaxillary line.
3. Prep and sterilize the area (Figure 17-1A).
4. Anesthetize the skin, muscle, periosteum, and parietal pleura through which the tube will pass by utilizing a local anesthetic such as lidocaine. If time permits, do intercostal blocks above and below to provide better anesthesia.
5. Estimate the distance from incision to apex of the lung on the chest tube, ensuring that the distance is enough to allow the last drainage

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

> When the clamp enters the pleura, a rush of air or fluid should be obtained.
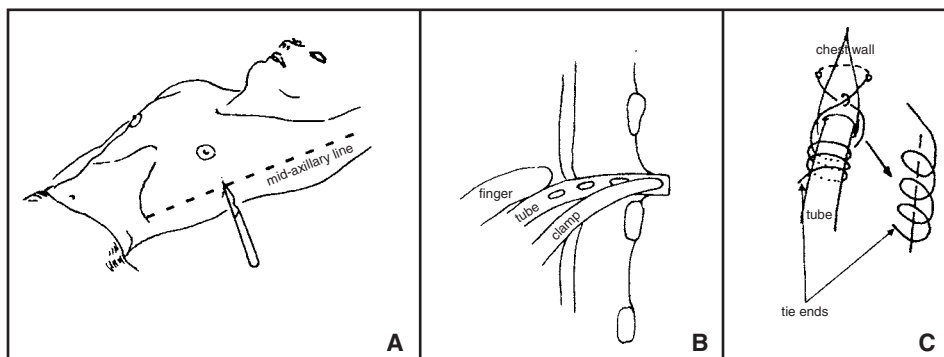
FIGURE 17-1. Procedure for tube thoracostomy.

A. An incision is made in the fourth or fifth intercostal space in the midaxillary line. **B.** Following finger exploration to confirm space, the tube is advanced, guided by the curved clamp. **C.** The tube is secured in place. (Modified, with permission, from Scaletta TA, Schaider JJ. *Emergent Management of Trauma.* New York: McGraw-Hill, 1996: 359–361.)

hole of the chest tube to fit inside the pleura. Place a clamp at this point of the chest tube.

6. Make a 2- to 4-cm skin incision over the rib below the one the tube will pass over. The incision should be big enough for the tube and one finger to fit through at the same time. Use blunt dissection to penetrate down to the fascia overlying the intercostal muscles.

7. Insert a closed, heavy clamp over the rib and push through the muscles and parietal pleura. Spread the tips of the clamp to enlarge the opening.

8. Close the clamps and insert one finger next to the clamp into the pleural space. Sweep the finger around to ensure that you are in the pleural space and there are no adhesions. While leaving your finger in, remove the clamp and insert the chest tube by clamping the tip with a curved clamp and following the path of your finger (Figure 17-1B).

9. Remove the clamp and guide the chest tube in a superior and posterior direction.

10. Insert the tube until your previously placed marker clamp is against the skin.

11. Attach the tube to a water seal.

12. Secure the tube by using suture material to close the skin and then wrapping it around the chest tube tightly enough to prevent slipping (the two ends of the suture are wrapped in opposite directions [Figure 17-1C]). A purse string stitch also works nicely.

13. Place an occlusive dressing over the area.

14. Chest tube placement should be confirmed by chest x-ray.

COMPLICATIONS

- Subcutaneous (SQ) (versus intrathoracic) placement
- Bleeding from intercostal vessels
- Injury to intercostal nerves
- Infection
- Lung laceration
- Diaphragm injury
- Liver injury

► PERICARDIOCENTESIS

DEFINITION

The drainage of fluid from the pericardium, which relieves tamponade (Figure 17-2).

PROCEDURE

1. If possible, electrocardiographic (ECG) monitoring should be utilized by clamping one of the precordial leads to the needle.

2. After prepping the area, insert a 16 or 18G needle at a 30-degree angle into the left xiphocostal angle about 0.5 cm below the costal margin.

3. Advance the needle to the inner aspect of the rib cage.

4. Depress the needle to get under the ribs and point it toward the left shoulder.

5. Advance the needle as you aspirate until fluid is reached; this should not be much more than 1 cm.

Size of chest tube to use:
For adult large hemothorax: 36–40 French
For adult pneumothorax: 24 French
For children: Four times the size of appropriate endotracheal tube

Pericardiocentesis is usually performed with ultrasound guidance or under fluoroscopy. Blind pericardiocentesis should be performed only in the unstable patient or as part of cardiac arrest protocols when other modalities are not available. In this instance, the subxiphoid approach is recommended.
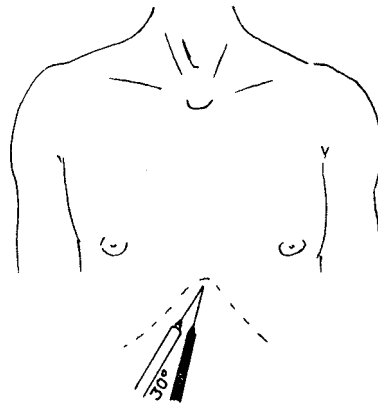
FIGURE 17-2. Pericardiocentesis via subxiphoid approach.

COMPLICATIONS

- Dry tap
- Dysrhythmias
- Air embolism
- Cardiac, vessel, or lung injury

Places you should not use epinephrine: **SPF-10**
Scrotum
Penis
Fingers

Toes
Ears
Nose

► **LOCAL ANESTHESIA**

- Local anesthesia is done by local infiltration into and around the wound or by regional block.
- Anesthetic is slowly injected adjacent to the wound edge in a sequential fashion or directly into the dermis and SQ tissue through the open wound edge using a small-gauge (25G) needle.
- Epinephrine may be added to provide vasoconstriction and prolong the action of the anesthetic.
- Commonly used local anesthetic agents are listed in Table 17-1.

TABLE 17-1. Commonly Used Local Anesthetic Agents

AGENT	CONCENTRATION	ONSET OF ACTION	MAXIMUM DOSAGE
Lidocaine (Xylocaine)	1% or 2%	Immediate: 4–10 min Duration: 1–2 hr	4.5 mg/kg = 30 mL of 1% solution
Mepivacaine (Carbocaine)	1% or 2%	Immediate: 6–10 min Duration: 1.5–2.5 hr	5 mg/kg = 35 mL of 1% solution
Bupivacaine (Marcaine)	0.25%	Slower: 8–12 min Duration: 6–8 hr	3 mg/kg = 80 mL of 0.25% solution

- Indications:
  - Diagnose acute, painful nontraumatic or traumatic joint disease by synovial fluid analysis.
  - Therapeutic intervention to drain an effusion or hemarthrosis.
- Contraindications: Infection (i.e., cellulitis or abscess) overlying affected joint.

PROCEDURE

- Under sterile conditions, use povidone–iodine solution to prep skin, then wipe off with alcohol to prevent introduction of iodine into the joint space.
- Apply sterile drape and anesthetize skin and overlying SQ tissue down to the joint capsule.
- When joint space is entered, there will be an abrupt decrease in resistance.
- Remove anesthetizing needle and syringe and follow same track using an 18G needle, or catheter for large joints, with a 30-mL syringe so as to completely drain the joint space.
- Once in joint space, gently aspirate. Then send fluid for analysis, which should include: Gram stain and culture, microscopy for crystals, complete blood count with differential, glucose, and protein.
- Cover area with sterile dressing.

JOINT FLUID ANALYSIS

Septic	White blood cell (WBC) > 50,000
	Polymorphonuclear neutrophil (PMN) > 85%
	Glucose < 50 mg/dL
	Gram stain positive in 65%
	Culture positive
Gout/pseudogout	WBC 2,500–50,000
	PMN 40–90%
	Urate crystals (gout)
	Ca^{2+} pyrophosphate crystals (pseudogout)
Inflammatory	WBC 10,000–50,000
	PMN 65–85%
Degenerative joint disease	WBC < 5,000
	PMN < 25%
Traumatic	Bloody
	WBC < 1,000
	Fat droplets (with fracture)

ASPIRATION SITES

Shoulder	Patient sits upright with arm held in neutral position. Enter joint space anteriorly and inferiorly to the coracoid process.
Elbow	Place elbow at 90 degrees of flexion with hand prone. Locate the radial head, lateral epicondyle, and lateral aspect of the olecranon tip (anconeus triangle). Needle enters at center of triangle, perpendicular to radius (Figure 17-3).
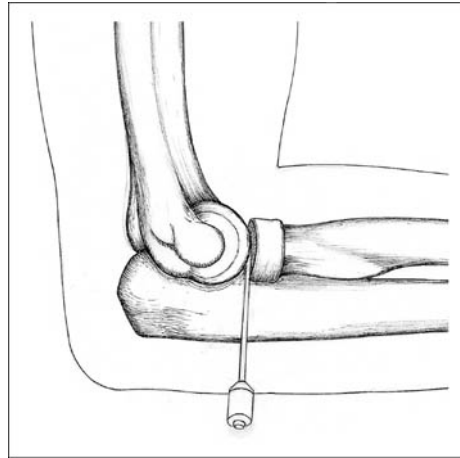
F I G U R E   1 7 - 3 .   Elbow arthrocentesis.

(Reproduced, with permission, from Wilson FC, Lin PP. *General Orthopaedics*. New York: McGraw-Hill, 1997: 121.)

Knee	With knee held in extension and slight flexion, enter joint space medially and inferior to the patella at its midpoint (Figure 17-4).
Ankle	With the foot in plantar flexion, place needle just medial to the anterior tibial tendon at the anterior edge of the medial malleolus.
Fingers/toes	With the digit flexed 15 to 20 degrees and traction applied, enter joint from the dorsal aspect medially or laterally to extensor tendon.
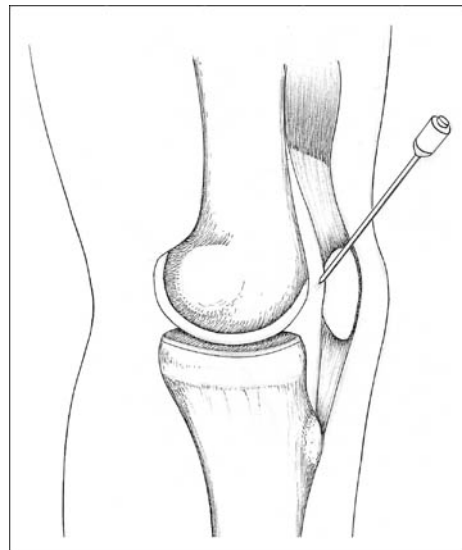
F I G U R E   1 7 - 4 .   Knee arthrocentesis.

Viewing the patella as the face of a clock, the needle is inserted just behind the patella at either 10 or 2 o'clock (medially or laterally). (Reproduced, with permission, from Wilson FC, Lin PP. *General Orthopaedics*. New York: McGraw-Hill, 1997: 123.)

| Wrist | With the wrist held at 20 to 30 degrees of flexion and traction applied, place needle dorsal and ulnar to the extensor pollicis longus tendon. |
| Thumb | With the thumb opposed, place the needle at the base of the first metacarpal on the palmar side of the abductor pollicis longus. |

INDICATIONS

- When vascular access cannot be obtained through other means.
- In children up to age 6 with difficult peripheral or central access. After age 6, red marrow is replaced by yellow marrow, making infusion more difficult.

SITES

- Proximal tibia (most common in children) puncture site is 1 to 3 cm below the tibial tuberosity, midline on the medial flat surface of the anterior tibia. Direct needle 15 degrees off the perpendicular away from the epiphysis (Figure 17-5).
- Distal tibia (common in adults) puncture site is the medial surface of the of ankle proximal to the medial malleolus.
- Distal femur puncture site is on the dorsal surface where the condyle meets the shaft of bone.
- Sternum (high complication rate).

PROCEDURE

- Using sterile technique, a bone marrow aspiration needle, intraosseous infusion needle, or spinal needle is grasped in the palm of the hand and
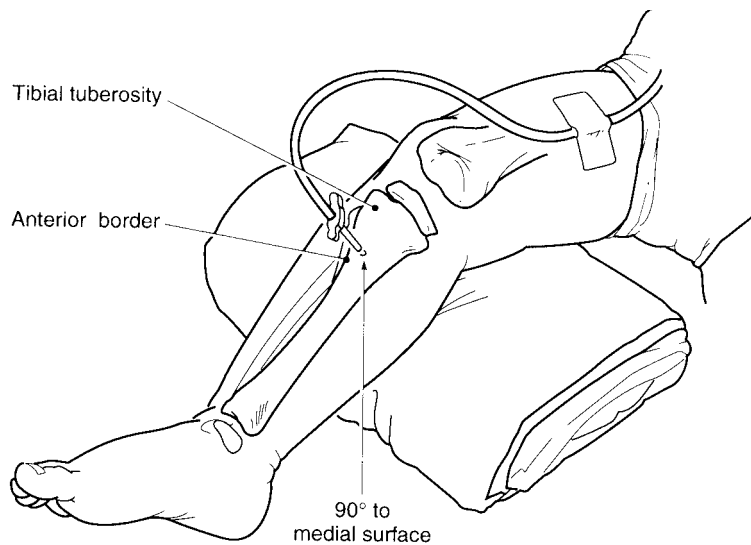
FIGURE 17-5. Intraosseous access.

(Reproduced, with permission, from *Textbook of Pediatric Life Support,* ©1997 American Heart Association.)

using a twisting motion is bored into the periosteum until the resistance decreases.
- Proper placement is confirmed by bone marrow aspiration or successful infusion of several milliliters of normal saline (NS).
- Local anesthesia is optional.

COMPLICATIONS

- Cellulitis
- Osteomyelitis (< 1%)
- Fracture
- Fat embolism (rare)
- Growth plate injury

▶ CONSCIOUS SEDATION

INDICATIONS

- Painful procedures done in the emergency department (ED) (incision and drainage [I&D], reduction of bone).
- Anxious patient.
- Uncooperative/anxious child who needs procedure or diagnostic test performed.

GOALS

- To provide an adequate state of sedation and analgesia while allowing patient to maintain an independent airway, reflexes, and response to verbal stimulation.
- To allow patient to be discharged quickly and safely.

PATIENT SELECTION

- Healthy individuals with mild or no systemic disease.
- No history of neurologic impairment.
- Fasting at least 4 hours from solid food or 2 hours from liquids (non-emergent cases only).

MONITORING

- Cardiac monitor and pulse oximetry.
- O_2, intubation equipment, and medical reversal agents should be readily available.
- Peripheral access should be obtained.
- Initial set of vitals, vitals 10 minutes after administration, and every 15 minutes thereafter until patient is alert and oriented × 3 and can sit up (if previously able to sit up).

MEDICATIONS

- Two agents are usually used, one for sedation (benzodiazepine) and another for analgesia (opiate).
- Although medications may be given IV, IM, PO, or PR, the IV route is preferred because of ease of titration of medications and quicker action.

- **Midazolam** (Versed):
  - Benzodiazepine.
  - 0.05 to 0.1 mg/kg IV, incremental dose at 2-minute intervals to desired effect.
  - Duration 30 to 45 minutes.
- **Diazepam** (Valium):
  - Benzodiazepine.
  - 0.1 to 0.2 mg/kg IV (max 10 mg).
  - Duration 2 to 6 hours, do not give IM (erratic absorption).
- **Fentanyl:**
  - Opioid
  - 50 to 400 μg IV, incremental dose at 2-minute intervals to desired effect.
  - Onset 1 to 3 minutes.
  - Duration 30 to 60 minutes.
- **Ketamine** (Ketalar):
  - Dissociative hypnotic.
  - 1 to 3 mg/kg.
  - Onset 30 to 60 seconds.
  - Duration 15 minutes.
  - Contraindications: Age < 3 months, increased intracranial pressure, seizure, glaucoma, psychosis, thyroid disorder, porphyria.
  - May see emergence reaction after age 10.
- **Pentobarbital:**
  - Barbiturate.
  - 4 to 6 mg/kg IV.
  - Onset 30 to 60 seconds.
  - Duration 2 to 4 hours.
- **Nitrous Oxide** (N_2O):
  - 50% N_2O/50% O_2 via inhalation.
  - Onset 2 to 5 minutes.
  - Duration 2 to 5 minutes.
  - Must administer continuously.
- **Flumazenil:**
  - Benzodiazepine antagonist.
  - 0.2 mg q1min to max of 1 mg in 5 minutes.
  - Onset 30 to 60 seconds.
  - Duration 20 minutes.
- **Naloxone:**
  - Opiate receptor antagonist.
  - 0.2 to 2.0 mg IV.
  - Onset 30 to 90 seconds.
  - Duration 2 to 3 hours.

An emergence reaction is the occurrence of hallucinations and nightmares during the wearing off of ketamine. Occurs in about half of all adults and in about 10% of children. Allowing the patient to recover in a dark, quiet room minimizes this phenomenon.

Naloxone is an opiate antagonist. Flumazenil is a benzodiazepine antagonist. Have these on hand when doing conscious sedation in case quick reversal becomes necessary.

▶ **SPLINTING**

INDICATIONS

- Fractures to be seen by orthopedics at a later date.
- Dislocations that have been reduced.

MATERIALS

- Fiberglass or plaster.

Plaster

- Measure the length of the affected extremity with the plaster, then place the plaster roll on a flat surface and unroll the plaster back and forth on itself to a total of 12 layers.
- Measure several layers of padding to be both longer and wider than the plaster.
- Submerge the plaster in water and hold until the bubbling stops.
- Strip excess water from plaster by holding plaster up in one hand and stressing plaster between thumb and index finger with the other from top to bottom.
- Place plaster on flat surface and massage layers together.
- Place padding on top, then apply to extremity with padding against skin and mold with palms, not fingers, to avoid creating pressure points.
- Wrap with Ace bandage (over gauze) for compression.

Fiberglass

- Cut material to desired length.
- Do not submerge in water. Put one strip of water down center of splint then curl up splint in a towel to remove excess moisture.
- Stretch outer padding over the ends of the fiberglass to avoid sharp edges and apply to extremity and Ace wrap.
- Fiberglass will harden within 10 minutes, much more quickly than plaster.

Upper Extremity Splints

- Reverse sugar tong (Figure 17-6):
  - Indications: Forearm or Colles' fracture.
  - Use 3″ to 4″ adult; cut splint in center, leaving small piece to overlie thumb.
- Boxer splint (Figure 17-7):
  - Indications: Fourth and fifth metacarpal fracture.
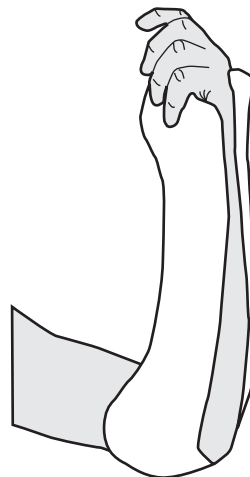
FIGURE 17-6. Reverse sugar tong splint.
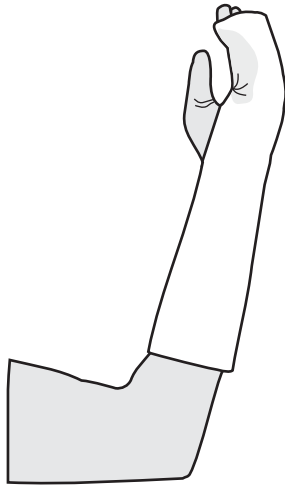
FIGURE 17-7. Boxer splint.

- Long arm ulnar gutter (elbow) splint (Figure 17-8):
  - Indications: Supracondylar fracture, elbow sprain, radial head fracture.
  - Elbow is held at 90 degrees. Splint extends from metacarpal heads to upper arm below axillary crease along ulnar surface.
- Cock-up splint:
  - Indications: Wrist sprain, carpal tunnel syndrome.
  - Wrist is in extension, splint extends from midforearm to metacarpophalangeal (MCP) on volar surface of hand.
- Thumb spica (Figure 17-9):
  - Indications: Navicular or scaphoid fracture, thumb dislocation, ulnar collateral ligament sprain, thumb proximal phalanx fracture, MCP fracture.
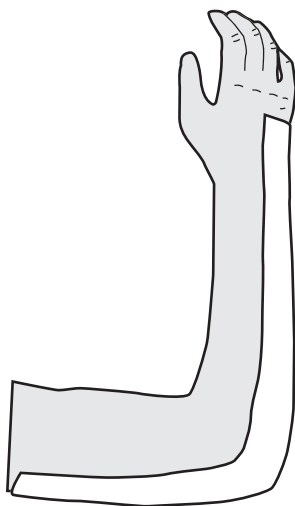
FIGURE 17-8. Elbow splint.
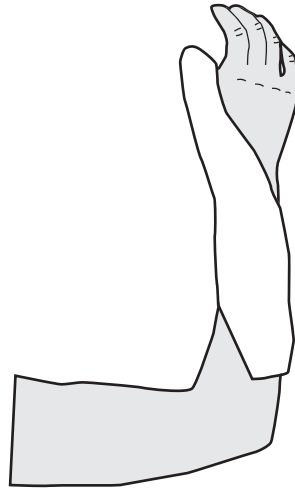
FIGURE 17-9. Thumb spica splint.

- The wrist is in neutral position and the thumb in abduction; the splint extends from the ulnar aspect of forearm and comes radially over dorsum of wrist and hand to encompass thumb.

Lower Extremity Splints

- Posterior leg (ankle) splint (Figure 17-10):
  - Indications: Distal tibia and fibula fracture, ankle sprain, Achilles' tendon tear, metatarsal fracture.
  - The ankle is in neutral position, except for Achilles' tears in which the patient should be immobilized in plantar flexion. The splint extends from 2″ posterior to knee to metatarsophalangeal heads.
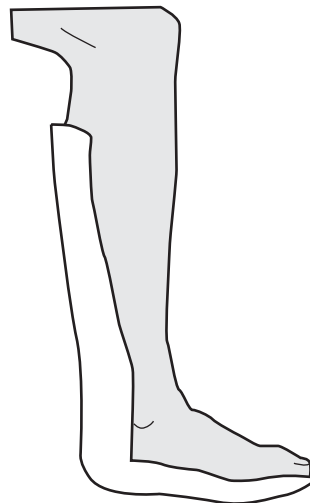
FIGURE 17-10. Posterior ankle splint.

FIGURE 17-11. Ankle stirrup splint.

- Ankle stirrup (Figure 17-11):
  - Indications: Ankle strain/sprain, shin splint, hairline fracture.
  - The splint extends from medial to lateral aspect of lower leg at mid-calf to encompass the calcaneus. This prevents inversion/eversion.
- Long leg splint:
  - Indications: Femoral fracture.
  - Apply like posterior leg splint, but superior aspect of splint extends to 3″ below buttock.
- Knee immobilizer:
  - Indications: Knee sprain, postop knee surgery.
  - Usually ready-made device that wraps around posterior and sides of the lower extremities from upper thigh to lower leg above the ankle. It is held in place by anterior Velcro straps.

▶ SUTURES

Types of Closure

- Primary:
  - Closure within 6 to 8 hours of any wound on body.
  - Face and scalp may be closed primarily up to 24 hours because of good vascular supply.
- Secondary: Wound heals by granulation alone ("2-degree intention").
- Delayed primary closure: Closure of a wound 3 to 5 days following injury.

Tetanus

- If unknown history or fewer than three doses, give Td (tetanus and diptheria) toxoid.
- If three doses given and within 7 years, no immunization necessary.
- If tetanus-prone wound (> 6 hours, avulsion, crush, > 1 cm depth, abrasion, contusion, contamination, devitalized tissue, or frostbite) and unknown tetanus history, give Td and tetanus immune globulin.

Give tetanus prophylaxis to anything that is **CUT:**
Contaminated wounds
Unknown tetanus history
Tetanus status expired

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Prevent infection with **PSI:**
Pressure 8 to 10 psi
Sterile saline
Irrigate with 500 to 1,000
mL

Prevent Infection

- Irrigation is the most important procedure required to clean a wound.
- Use NS, an 8G needle, and 30-cc syringe to irrigate wound with 8 to 10 psi.
- Antibiotics are not proven to be of prophylactic benefit. Do give for grossly contaminated wounds.

Suture Equipment

- Needles:
    - Cutting: Has two cutting edges for shallow bites.
    - Reverse cutting: Has three cutting edges for deeper bites.
- Needle holder:
    - Place at 90-degree angle.
    - One third from swage.
    - Needle at tip of needle holder.
- Dissecting forceps: To gently evert skin edges.
- Skin hooks.
- Scissors.
- Local anesthetic.
- Suture material: See Tables 17-2 and 17-3. See Table 17-4 for closure recommendations by wound site.

Suturing Techniques

- Simple interrupted (Figure 17-12):
    - 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.

TABLE 17-2. **Suture Materials: Absorbable**

MATERIAL	HALF-LIFE	TYPE	COMMENTS
Gut	5–7 days	Natural	Stiff, rapidly absorbed, used for mucosal closure only
Chromic gut	10–14 days	Natural	Used for intraoral lacerations
Polyglycolic acid (Dexon)	10–15 days	Synthetic multifilament	Used for SQ sutures, difficult to tie
Polyglactin 910 (Vicryl)	14–21 days	Synthetic multifilament	Use clear variety on face, easy workability, SQ
Polydioxanone (PDS II)	28 days	Synthetic monofilament	Very strong, low reactivity, large knot mass

TABLE 17-3. Suture Materials: Nonabsorbable

MATERIAL	TYPE	COMMENTS
Silk	Natural multifilament	Easiest to handle but poses greatest risk of infection, not used on face
Nylon (Ethilon and Dermalon)	Synthetic mono- or multifilament	Low tissue reactivity, most often used for cutaneous closure
Polypropylene (Prolene)	Synthetic monofilament	Stiffest sutures, requires five to six knots, tends to untie
Polyester (Mersilene)	Synthetic multifilament	Easy handling with excellent security, often used for vascular or facial wounds
Polybutester (Norafil)	Synthetic monofilament	Slight elasticity allows wound swelling

TABLE 17-4. Closure Recommendations by Wound Sites

LOCATION	MATERIAL	TECHNIQUE
Scalp	3-0 or 4-0 nylon or polypropylene	Interrupted in galea, single tight layer in scalp, horizontal mattress if bleeding not well controlled
Pinna	5-0 Vicryl/Dexon in perichondrium	Close perichondrium with interrupted Vicryl and close skin with interrupted nylon
Eyebrow	4-0 or 5-0 Vicryl (SQ) 6-0 nylon	Layered closure
Eyelid	6-0 nylon	Single-layer horizontal mattress or simple interrupted
Lip	4-0 Vicryl (mucosa) 5-0 Vicryl (SQ or muscle)	If wound through lip, close three layers (mucosa, muscle, skin); otherwise do two-layer closure
	6-0 nylon (skin)	
Oral cavity	4-0 Vicryl	Simple interrupted or horizontal mattress if muscularis of tongue involved
Face	6-0 nylon (skin) 5-0 Vicryl (SQ)	Simple interrupted for single layer, layered closure for full-thickness laceration
Trunk	4-0 Vicryl (SQ, fat) 4-0 or 5-0 nylon (skin)	Single or layered closure
Extremity	3-0 or 4-0 Vicryl (SQ, fat, muscle) 4-0 or 5-0 nylon (skin)	Single-layer interrupted or vertical mattress; apply splint if over a joint
Hands and feet	4-0 or 5-0 nylon	Single-layer closure with simple interrupted or horizontal mattress; apply splint if over a joint
Nail bed	5-0 Vicryl	Meticulous placement to obtain even edges, allow to dissolve

HIGH-YIELD FACTS

Procedures
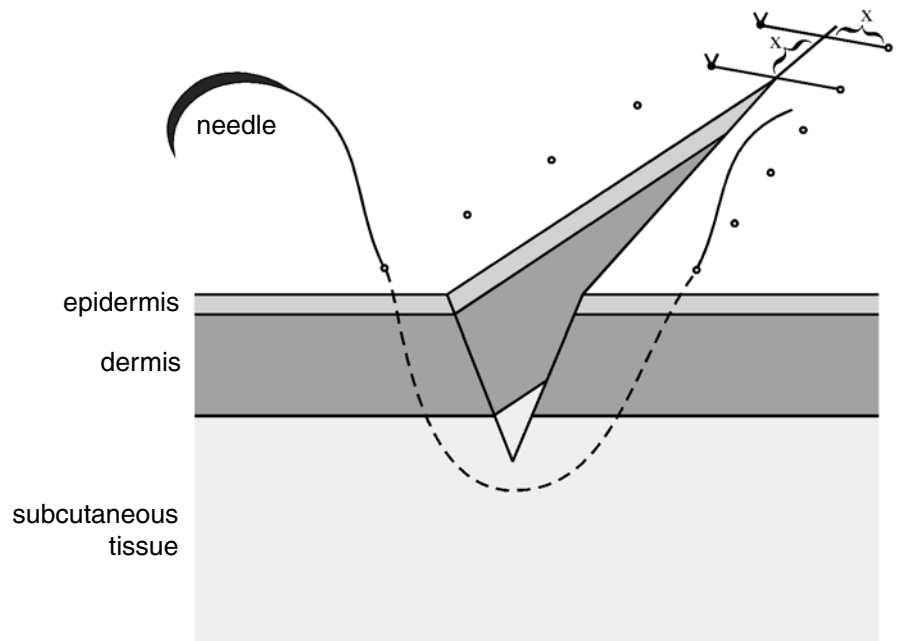
FIGURE 17-12. Simple interrupted suture.

- Running suture (Figure 17-13):
  - 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.
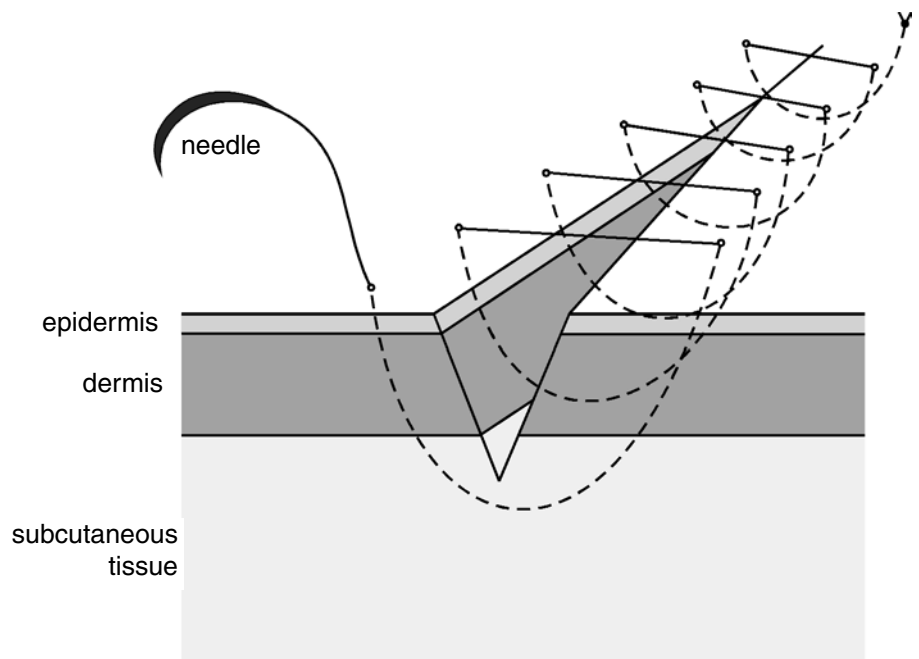
FIGURE 17-13. Running suture.
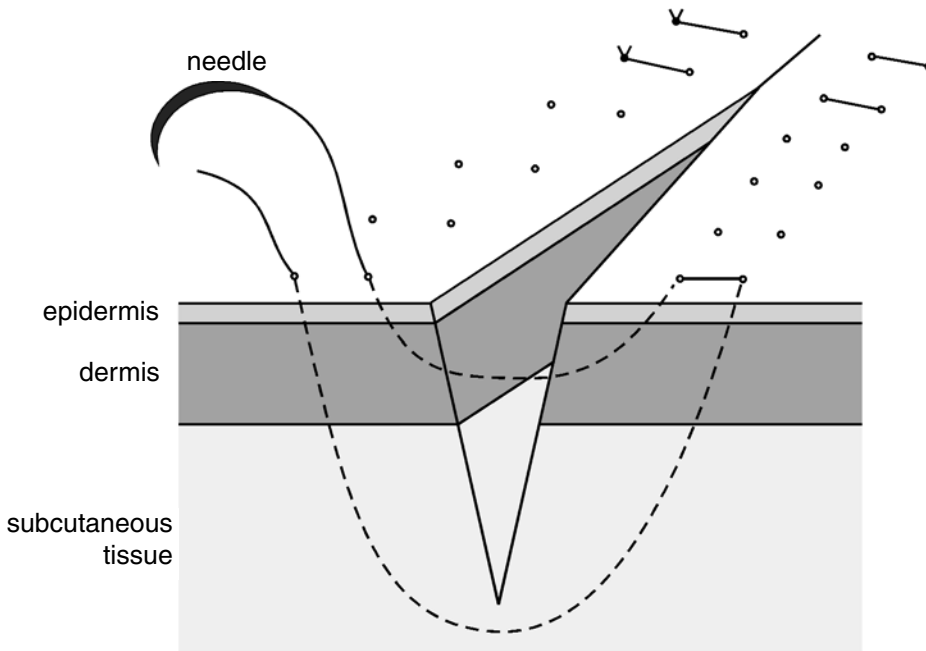
F I G U R E   1 7 - 1 4 .   Vertical mattress suture.

- Vertical mattress (Figure 17-14):
  - 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 farther 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.
- Horizontal mattress (Figure 17-15):
  - 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.
- Deep sutures (absorbable):
  - Used for multilayered closure.
  - Deep sutures are absorbable because you will not be removing them.
  - Use your forceps (pickups) to hold the skin from the inside of the wound.
  - The first stitch is placed deep inside wound and exits superficially in dermal layer on same side of wound.
  - Then it enters in the superficial dermal layer on the opposite side and exits deep.
  - Tie a square knot and cut the tail of the suture close to the knot, which is called a *buried knot*.
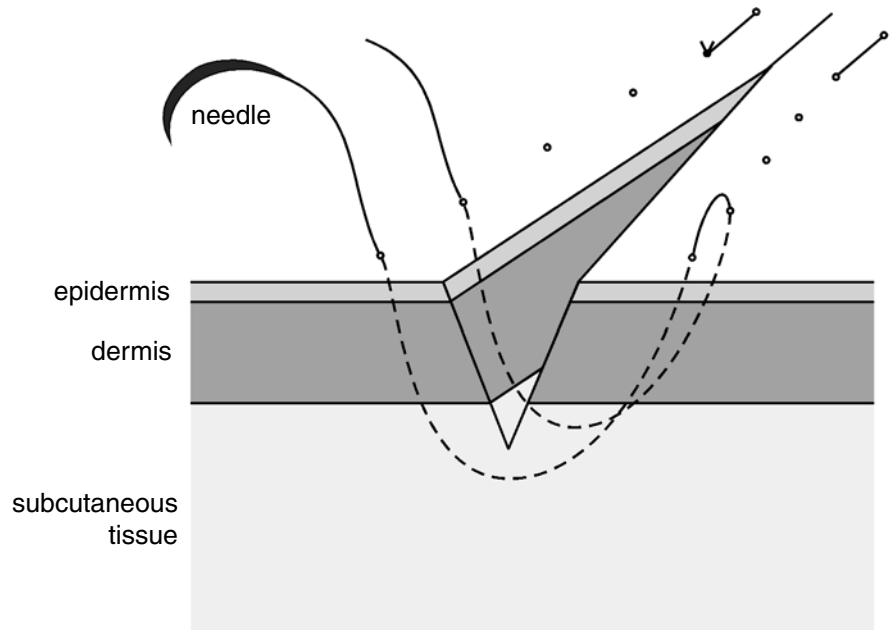
FIGURE 17-15. Horizontal mattress suture.

- Now proceed with your superficial closure of the skin with nonabsorbable sutures.
- Corner stitch (Figure 17-16):
  - This is used to repair stellate lacerations and help to preserve the blood supply to the tips of the skin.
  - The needle enters the epidermis of the nonflap or nontip portion of the wound.
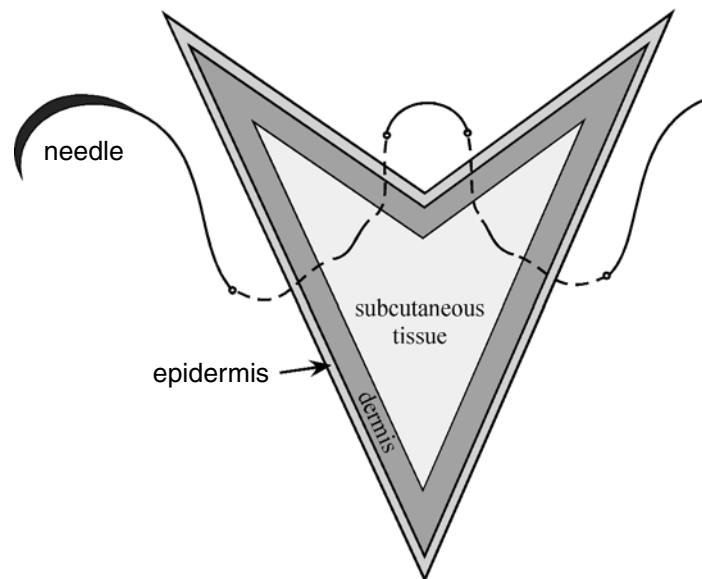
FIGURE 17-16. Corner stitch.

TABLE 17-5. Suture Removal Times

SITE	DAYS
Face, eyelid, ear, nose	3–5
Neck	5–7
Scalp, trunk	7–12
Arm, hand	8–12
Leg, foot, extensor surface of joints	10–14

- It then enters the dermal layer of the skin tip on one side and proceeds through the dermal layer to exit the dermis on the other side of the tip (this portion will be buried).
- It then enters and exits the other side of the stellate wound. It will appear as a simple interrupted suture.

Dressing

- Bacitracin ointment may be used over the repair.
- Cover the laceration with a single layer of nonadherent dressing, then cover that with gauze.
- For an extremity, wrap with gauze bandage (Kerlix) and take care not to tape circumferentially.
- The patient should come back for a wound check in 2 to 3 days for contaminated or deep wounds.
- The dressing should be changed every day and replaced with a Band-Aid or gauze.
- Keep area dry and look for signs of infection: Increased warmth, swelling, increasing erythema, streaking, dehiscence, more-than-normal discharge from wound, and fever.

See Table 17-5 for when to remove sutures.

► CENTRAL VENOUS ACCESS

Relative Indications

- Patient undergoing cardiopulmonary resuscitation: Internal jugular (IJ), SC subclavian, femoral.
- Patient unable to lie flat: SC subclavian.
- Coagulopathy: Femoral because more readily compressible.

Relative Contraindications

General (applies to all routes)
- Distorted anatomy
- Overlying cellulitis or severe dermatitis
- Prior scarring of vein
- Significant coagulopathy

Subclavian vein cannulation can be infraclavicular (IC) or supraclavicular (SC).

Subclavian
- Contralateral pneumothorax
- Chest wall deformity
- Chronic obstructive pulmonary disease

IJ
- Carotid artery stenosis (dislodging of plaque may occur due to inadvertent carotid artery cannulation).

Femoral
- Ambulatory patient
- Groin trauma

Procedure of Central Venous Cannulation

1. Use aseptic technique.
2. Place patient in Trendelenburg position.
3. Local anesthesia at point of needle entry.
4. Insert and aim needle on syringe as appropriate for approach with gentle negative pressure to the syringe.
5. Nonpulsatile free flow flashback of blood indicates good position. Pulsatile flow may indicate inappropriate arterial placement.
6. Once within the vein, advancing of the guidewire depends on the commercial set you use. Some models will allow catheter insertion through the needle; others are over-the-needle.
7. Use scalpel to make small stab incision adjacent to guidewire (enlarges opening for catheter).
8. Pass catheter over guidewire. In most sets, removing the guidewire from the distal end of the catheter requires removal of the brown port.
9. NEVER let go of the guidewire.
10. Catheter should pass easily, without any forcing.
11. Once catheter is in to desired length, remove guidewire.
12. Check blood flow from catheter.
13. If not a triple lumen, connect catheter to IV tubing.
14. Suture catheter in place.
15. Place occlusive dressing over site.
16. Obtain chest x-ray for neck lines to confirm placement and to be certain no pneumothorax was caused by the procedure.

Landmarks for IC Subclavian

- Insert needle at junction of middle and proximal thirds of clavicle (Figure 17-17).
- Aim needle toward suprasternal notch.
- Vein entry at 4 cm.

Landmarks for SC Subclavian

- Insert needle behind the clavicle, lateral to the clavicular head of the sternocleidomastoid (SCM) (Figure 17-18).
- Aim needle toward contralateral nipple.
- Vein entry at 3 cm.

The Trendelenburg position is thought to prevent air embolism.

The right subclavian is preferred over the left in order to avoid thoracic duct (on left) or lung injury (dome of right lung is lower).

FIGURE 17-17. IC approach to subclavian vein cannulation.

(Reproduced, with permission, from Scaletta TA, Schaider JJ. *Emergent Management of Trauma*, 2nd ed. New York: McGraw-Hill, 2001: 441.)

Landmarks for IJ Cannulation, Central Approach

- Insert needle at junction of the two heads of the SCM.
- Aim needle toward ipsilateral nipple.
- Maintain needle at 30- to 45-degree angle.
- Vein entry at 1 to 1.5 cm.

Landmarks for IJ Cannulation, Anterior Approach

- Insert needle at medial edge of sternal head of SCM halfway up (Figure 17-19).
- Maintain needle at 30- to 45-degree angle.
- Aim needle toward ipsilateral nipple.
- Vein entry at 1.5 cm.

Landmarks for IJ Cannulation, Posterior Approach

- Insert needle at lateral edge of clavicular head of SCM, a third of the way up between the clavicle and the mastoid (Figure 17-20).
- Aim needle toward sternal notch.
- Vein entry at 5 cm.
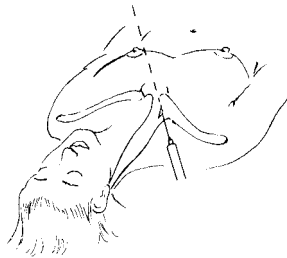
FIGURE 17-18. Supraclavicular (SC) approach to subclavian vein cannulation.

(Reproduced, with permission, from Scaletta TA, Schaider JJ. *Emergent Management of Trauma*, 2nd ed. New York: McGraw-Hill, 2001: 442.)

F I G U R E 1 7 - 1 9 . Anterior IJ cannulation.

Remember **NAVEL** *from lateral to medial:* **N**erve, **A**rtery, **V**ein, **E**mpty space, **L**ymphatics

Landmarks for Femoral Vein Cannulation

- Insert needle medial to pulsation of femoral artery.
- Maintain needle at 45-degree angle.
- Aim needle toward the head.
- Vein entry at 3 cm.
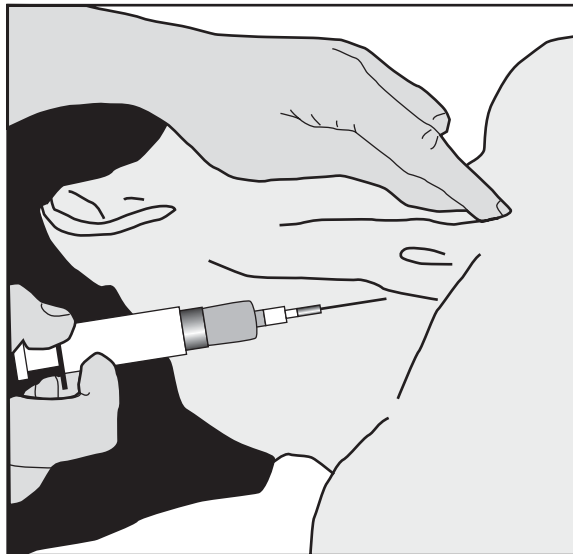
F I G U R E 1 7 - 2 0 . Posterior IJ cannulation.

Complications

- Infection
- Thrombosis
- Pneumothorax (not with femoral)
- SQ emphysema

The I&D of abscesses is a very common procedure performed in the ED. Most skin abscesses are uncomplicated and can be drained easily with local anesthetic. Larger abscesses or ones in exquisitely sensitive areas may require conscious sedation or, occasionally, drainage in the operating room under general anesthesia.

Most Common Sites

- Axilla—25%
- Buttock/perirectal—25%
- Head/neck—20%
- Extremities—18%
- Inguinal area—15%

Pathogens

Variety of aerobic and anaerobic. Most common:
- *Staphylococcus aureus*
- *Bacteroides fragilis*
- *Streptococcus viridans*

Equipment Needed

- Scalpel
- Hemostats
- Scissors
- Iodine solution
- Gauze
- Packing material—most commonly 1″ or 2″ plain or iodoform
- Dressing material
- Chucks
- Personal protective equipment
- Gloves
- Gown
- Eye protection
- Mask

Procedure

After explaining the procedure and placing the patient in a comfortable position with adequate exposure and lighting with chucks to minimize the mess:

1. Clean the area and prepare it with iodine solution.
2. Anesthetize the skin with the lidocaine preparation.

Immunocompromise is associated with recurrent abscesses (i.e., diabetes, human immunodeficiency virus).

It is very difficult to completely anesthetize the abscess locally due to the acidic environment of abscesses. Use a regional block if possible.

While this is not a sterile procedure, it should be a clean procedure. Care should be taken not to infect areas not involved with the abscess.

HIGH-YIELD FACTS

Procedures

Antibiotics in healthy individuals are unnecessary. Consider antibiotics in immunocompromised individuals and in patients with valvular disease to prevent seeding due to transient bacteremia.

Perirectal abscesses can be life threatening.

3. Make an incision large enough to ensure adequate exposure.
4. Care must be taken with the incision in the face and breast due to cosmetic considerations.
5. In these areas, cut along the natural wrinkle or crease lines to minimize scarring.
6. Explore the cavity with the hemostats to break up any loculations and express remaining pus.
7. If the abscess is large enough, pack it as much as possible, leaving some packing outside of the cavity.
8. Dress the area appropriately.
9. Patients should return in 24 hours for packing removal and wound check.

Special Considerations

- Perirectal abscesses require careful evaluation because they can extend deep into the perineum.
- Sebaceous cyst/abscesses can be excised with the shell intact, thus preventing recurrences.
- Cultures are generally unnecessary in first-time abscesses.

▶ DORSAL SLIT

INDICATIONS

Symptomatic phimosis where foreskin cannot be retracted.

RELATIVE CONTRAINDICATIONS

Coagulopathy.

PROCEDURE (SEE FIGURE 17-21)

1. Cleanse and drape the penis.
2. Infiltrate 1% lidocaine *without epinephrine* in the foreskin on the dorsal midline along the line of incision to raise a wheal.
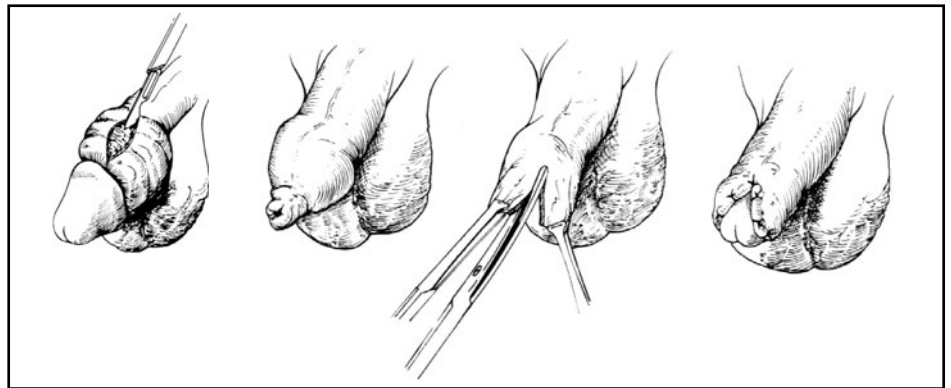
FIGURE 17-21. **Dorsal slit procedure for foreskin phimosis.**

(Reproduced, with permission, from Stone CK, Humphries RL. *Current Emergency Diagnosis & Treatment,* 5th ed. New York: McGraw-Hill, 2004.)

3. Using a straight hemostat to remove adhesions between the inner surface of the foreskin and the glans to create a tract until just proximal to the coronal sulcus.
4. Clamp the foreskin up to the coronal sulcus on the dorsal midline tract created using the hemostat for 3 to 5 minutes to reduce bleeding when incised.
5. Incise along the line and retract the foreskin.

COMPLICATIONS

- Urethral injury
- Infection
- Bleeding

▶ NASOGASTRIC TUBE PLACEMENT

INDICATIONS

- Therapeutic:
    1. Gastric decompression prior to surgery or in trauma
    2. Recurrent vomiting as with intestinal obstruction or paralytic ileus
    3. Administration of medication/feeds
- Diagnostic:
    1. Lab analysis of gastric contents
    2. Determination of presence, volume of an upper GI hemorrhage
    3. Instillation of air to enhance visualization of free air under the diaphragm on an erect chest film in gut perforation

CONTRAINDICATIONS

- Facial fractures or cribriform plate injury (absolute)
- Esophageal strictures
- Esophageal weakening (e.g., recent alkali exposure)
- Coagulopathy

PROCEDURE

1. Position the patient sitting upright against the bed/backrest.
2. After selecting tube type and size, estimate insertion depth by adding the distance from the tip of the nose to the earlobe plus the earlobe to the xiphoid.
3. Check patency of the nostrils by occluding each nostril separately and asking the patient to inhale.
4. Anesthetize nares and oropharynx using a topical anesthetic spray.
5. Lubricate the distal 6 mm of the nasogastric tube.
6. Insert at a 90-degree angle to the patient's face until it reaches the posterior nasal pharynx, where resistance will be encountered.
7. Gentle pressure helps turn the tube and advance it caudally. Have the patient sip water simultaneously to facilitate the passage of the tube until depth marked.
8. Secure using tape and confirm position a chest x-ray.

COMPLICATIONS

- Nasopharyngeal injury
- Pulmonary placement
- Pneumothorax, pneumomediastinum
- Oropharyngeal coiling

► CARDIOVERSION

INDICATIONS

1. Any patient with reentrant tachycardia (ventricular rate > 150) who is unstable/symptomatic (e.g., chest pain, pulmonary edema, light-headedness, hypotension) should be immediately treated with synchronized electrical cardioversion.
2. Cardioversion can also be used in hemodynamically stable patients to restore sinus rhythm in conditions like:
   - Atrial fibrillation
   - Atrial flutter
   - Atrial tachycardia
   - Supraventricular tachycardias

RELATIVE CONTRAINDICATIONS

- Known digitalis toxicity–associated tachydysrhythmia
- Sinus tachycardia caused by various clinical conditions

PROCEDURE

1. Sedation with IV midazolam (0.15 mg/kg) or other IV anesthetic agent.
2. Selection of synchronized mode on the cardioverter to synchronize the discharge and avoid cardioversion during repolarization. Synchronization should not be used in patients with ventricular fibrillation.
3. Apply conductive gel pads in the anteroposterior or anterolateral position as shown.
4. Energy is dialed up according to the indication and discharged until patient reverts to sinus rhythm.

ENERGY SETTINGS

Energy levels used depend on whether the defibrillator is monophasic or biphasic. Energy settings for common indications are given below:

	Monophasic (J or Watt-sec)
Atrial fibrillation	100, 200
Atrial tachycardia	50, 100
Ventricular tachycardia	100, 200

Settings on biphasic defibrillators depend on the manufacturer recommendations for different brands.

COMPLICATIONS

- Arrhythmias and conduction abnormalities
- Embolization
- Myocardial necrosis or dysfunction
- Transient hypotension
- Pulmonary edema
- Painful skin burns from electrode placement

► **OTHER PROCEDURES**

See Table 17-6 for procedures covered in other chapters.

TABLE 17-6. **Procedures Covered in Other Chapters**

PROCEDURE	CHAPTER
Intubation, cricothyroidotomy (needle and surgical), laryngeal mask airway, Heimlich maneuver	Resuscitation
Interpretation of ECGs and imaging studies	Diagnostics
Reading a C-spine film, focused assessment with sonography for trauma, diagnostic peritoneal lavage, retrograde urethrogram, and cystogram	Trauma
Lumbar puncture	Neurologic Emergencies
Nasal packing	Head and Neck Emergencies
Testicular detorsion	Renal and Genitourinary Emergencies
Vaginal wet prep	Gynecologic Emergencies
Gastric lavage	Emergency Toxicology

HIGH-YIELD FACTS

Procedures

Emergency Toxicology

Initial management of poisoned patient should emphasize supportive care:

- Stabilize ABCs and abnormal vital signs.
- Search to identify toxidromes.
- Perform a focused diagnostic workup.
- Decontamination, elimination, and antidotes as indicated.
- Continuous reassessment is critical (patients may deteriorate rapidly).

Clinical pictures of symptoms and physical findings that correlate with a specific toxin recognize that "classic" toxidromes may be obscured in the setting of a multidrug overdose (in which each toxin may cause competing signs/symptoms).

► TOXIDROMES

Cholinergic

- Commonly seen with organophosphates and carbamates.
- Symptoms are due to excessive stimulation of nicotinic/muscarinic acetylcholine (ACh) receptors.
- Muscarinic effects: Bronchorrhea, miosis, bradycardia (SLUDGE).
- Nicotinic effects: Fasciculations, cramping, hyperreflexia, hypertension, tachycardia.
- Use atropine +/– pralidoxime to reverse cholinergic excess.

Anticholinergic

- Many potential agents: Scopolamine, amanita muscaria, monoamine oxidase inhibitors (MAOIs).
- Think of the following four "anti" groups of drugs: Antidepressants, antihistamines, antipsychotics, antiparkinsonian.
- Clinical picture caused by antagonism/inhibition of ACh.
- Other findings: Seizures, dysrhythmias, hyperthermia.
- Treatment is supportive: Benzodiazepines, cooling measures.
- Physostigmine:
  - Binds to acetylcholinesterase, increases ACh.
  - Indicated only for unstable/refractory cases (seizures and dysrhythmias are common).
  - Avoid in tricyclic antidepressant (TCA) ingestions (asystole).

Sympathomimetic

- Agitation, mydriasis, tachycardia, hypertension, hyperthermia.
- Sympathomimetic toxidrome resembles anticholinergic except for diaphoresis (sympathetic-mediated ACh stimulation of sweat glands causes diaphoresis), and hypoactive bowel sounds (hyperactive in sympathomimetics).
- Multiple mechanisms of action of sympathomimetics:
  - Direct stimulation of alpha/beta-adrenergic receptors.
  - Amphetamines stimulate release of norepinephrine (NE) into synapse.

Cholinergic toxidrome:
BAD SLUDGE
Bradycardia
Anxiety
Delirium

Salivation
Lacrimation
Urination
Defecation
Gastrointestinal (GI) upset
Emesis

Anticholinergic toxidrome:
- Mad as a hatter: *Altered mental status*
- Blind as a bat: *Mydriasis*
- Red as a beet: *Flushed skin*
- Hot as a hare: *Hyperthermia (can't sweat)*
- Dry as a bone: *Dry mucous membranes*

Toxins associated with mydriasis: **AAAAS**
Antihistamines
Antidepressants
Anticholinergics
Atropine
Sympathomimetics

HIGH-YIELD FACTS

Emergency Toxocology

 ■ Cocaine/TCAs prevent reuptake of NE from synapse.
 ■ MAOIs inhibit breakdown of NE.
 ■ Treatment is supportive: Benzodiazepines, hydration, cooling.

Opioid

 ■ Heroin, morphine, propoxyphene, meperidine, codeine, fentanyl.
 ■ Hypothermia, bradycardia, hypotension, pulmonary edema.
 ■ Naloxone (Narcan): Competitive opiate receptor antagonist.
 ■ Dose depends on sensitivity of receptors to particular opiate.
 ■ Caution: May precipitate seizures (especially tramadol), agitation
 (withdrawal), noncardiogenic pulmonary edema.

Sedative–Hypnotic

 ■ Benzodiazepines and barbiturates both work by potentiating gamma-
 aminobutyric acid (GABA) (inhibitory neurotransmitter).
 ■ Dose-dependent central nervous system (CNS) and respiratory depres-
 sion (with barbiturates). Distinguish from ethanol (EtOH) intoxication
 by lack of vasodilation.
 ■ Coingestion of alcohol, tranquilizers, or other depressants may potenti-
 ate these effects and lead to coma and/or apnea.
 ■ Flumazenil: Competitive benzodiazepine receptor antagonist (cau-
 tion—withdrawal seizures are common).
 ■ Benzodiazepine overdoses usually low morbidity, flumazenil rarely used.

▶ **DIAGNOSTIC ADJUNCTS**

A variety of diagnostic studies and interventions are available to guide man-
agement.

"Coma Cocktail"

Serves as both a diagnostic and therapeutic intervention (hypoglycemia, hy-
poxia, opiates are common and easily reversible causes of CNS depression).

Acid–Base

 ■ Arterial blood gas (ABG) evaluation should be obtained if indicated
 clinically:
 ■ Respiratory acidosis in comatose patients suggests opiates/sedatives.
 ■ Respiratory alkalosis may be seen in sympathomimetic overdoses.
 ■ Respiratory alkalosis with metabolic acidosis is suggestive of acetylsal-
 icylic acid (ASA).
 ■ Serial ABG may be indicated in patients who require mechanical venti-
 lation.
 ■ Alkalinization of serum (TCA) and urine (ASA).

Electrolytes

 ■ Metabolic acidosis should be classified as anion gap versus nonanion
 gap using serum electrolytes (anion gap metabolic acidosis may be seen
 in certain toxic ingestions—CAT MUDPILES).

- STAT electrolytes may be sent with ABG for potassium (useful in management of digoxin overdoses).
- May be used in conjunction with serum osmolarity to detect toxic alcohol ingestion (with elevated osmole gap).

Spot Tests

- Classic example is ferric chloride test for salicylates (ferric chloride drops turn urine purple in presence of ASA).
- Spot tests also exist for acetaminophen, phenothiazines, barbiturates, ethanol, and certain types of mushrooms (most require high drug concentrations and have low sensitivity).

Immunoassays

- Most popular screening tool in emergency setting.
- Antibodies generated to representative antigen in each class (i.e., amitriptyline for TCA, morphine for opiates, etc.).
- Tests have varying sensitivities and low specificity (negative test does not rule out all types of TCA, opiates, etc.).

Quantitative Tests

Useful for determining concentrations of specific toxins:
- May be useful in guiding management (acetaminophen).
- Serial levels (assess adequacy of decontamination/elimination).
- Predicting clinical outcome.

Radiography

Certain toxins appear radiopaque on plain films.

▶ **METHODS OF DECONTAMINATION**

Forced Emesis

Rarely used anymore.
- Syrup of ipecac:
  - Plant derivative containing alkaloids (emetine and cephaeline).
  - Direct emetic effect on stomach.
  - Central effect on chemotactic trigger zone.
  - Produces emesis in over 90% of patients after single dose.
- Limit ipecac use to:
  - Prehospital (when access to emergency services may be significantly delayed).
  - Pediatric (where large-bore orogastric lavage may be contraindicated).
- Complications of ipecac include intractable vomiting, myocardial toxicity, and aspiration.

Gastric Lavage

- Technique for orogastric lavage:
  - Use large-bore tubes for intact pills (size 36 to 40 in adults, 22 to 24 in children).
  - Place patient in left lateral decubitus position (to minimize aspiration risk).
  - Have suction ready, measure length of tube, insert, and confirm position in stomach.
  - Lavage with fluid until aspirate is clear.
- Usually must be performed within 1 hour to be of clinical utility except:
  - Toxic ingestions that delay gastric emptying (i.e., anticholinergics).
  - Drugs that form concretions (aspirin) take longer to clear stomach.
- Complications include aspiration, esophageal/gastric perforation, and hypoxia during procedure.

Activated Charcoal

- Directly binds to toxin in gut lumen.
- Decreases concentration of toxin in stomach, creating a gradient favoring flow of toxin from blood into stomach.
- Binds to toxin in bile (interrupts enterohepatic circulation).
- Multiple-dose charcoal:
  - May be beneficial with certain toxins (digoxin, phenytoin, etc.).
  - Given empirically for ingestions of sustained-release products and drugs that form concretions.
  - Avoid repeat doses of cathartics with charcoal.

Cathartics

- Limited role in toxic ingestions:
  - May relieve constipating effects of charcoal.
  - May prevent "desorption" of toxin from charcoal over time.
  - By decreasing transit time through gut.
- Concerns about cathartics:
  - May cause significant fluid and electrolyte shifts.
  - Decreased transit time gives charcoal less time to bind.
- Current recommendations are to routinely give single dose of cathartic with charcoal.

Whole Bowel Irrigation (WBI)

- WBI is a technique of flushing out the entire GI system with large volumes of fluid.
- Polyethylene glycol (Go-Lytely) is an isotonic fluid that does not cause significant bowel edema or fluid and electrolyte shifts.
- Concerns with WBI:
  - Vomiting, bloating, and rectal irritation are common.
  - Polyethylene glycol may occupy charcoal binding sites.
  - It is time consuming, labor intensive, and messy.
- Indications for WBI:
  - Toxins that do not bind to charcoal.
  - Ingestions of sustained-release products.
  - Body-packers (smuggling illicit drugs in GI tract).

Cathartics

- Sorbitol causes catharsis in < 2 hours; may cause abdominal cramping.
- Magnesium citrate causes catharsis in 4 to 6 hours; contraindicated in patients with renal failure.
- WBI minimally requires adults to drink 2 L/hr of polyethylene glycol (0.5 L/hr in children).
- Usually requires nasogastric tube and/or antiemetics to administer large volumes of fluid.
- End point of therapy is clear rectal effluent.
- Contraindications to cathartic use:
  - Patients with impaired gag
  - Intestinal obstruction
  - Caustics

Urinary Alkalinization

- If pH of urine is raised to 7.5 to 8.0, "ion trapping" mechanism eliminates certain toxins.
- Hypokalemia impairs alkalinization by dumping H^+ ions in urine in exchange for K^+ (supplemental K^+ should be given intravenously [IV] with bicarbonate for alkalinization).

Hemodialysis and Hemoperfusion

- Hemoperfusion: Charcoal filter in series with dialysis:
  - Most useful for theophylline.
- Toxins that can be dialyzed: STUMBLE.

Toxins that can be dialyzed:
I STUMBLE
Isopropyl
Salicylates
Theophylline
Uremia
Methanol
Barbiturates
Lithium
Ethylene glycol/**E**thanol

▶ OVER-THE-COUNTER MEDICATIONS

Acetaminophen

- Chemical name is *N*-acetyl-para-aminophenol (commonly abbreviated as APAP).
- Most commonly reported potentially toxic ingestion, accounts for one third of all emergency department (ED) visits in the United States.
- Frequent coingestant.

EXPOSURE

- Tylenol, Paracetamol.
- Cold and flu preparations.
- Acetaminophen/oxycodone (Percocet), acetaminophen/hydrocodone (Vicodin).

MECHANISM OF TOXICITY

- Metabolism of acetaminophen (therapeutic doses):
  - Over 90% is metabolized by liver into nontoxic sulfate and glucuronide conjugates.
  - Less than 5% is directly excreted in urine.
  - Less than 5% is processed by cytochrome P450 system in liver to form *N*-acetyl-para-benzoquinoneimine (NAPQI).

Toxins for which alkalinization may be helpful:
- Aspirin
- Phenobarbital
- Chlorpropamide

- Metabolism of acetaminophen (in overdoses):
  - Sulfation and glucuronidation pathways become saturated.
  - P450 processes more APAP, generating more NAPQI.
  - NAPQI is a toxic intermediate of APAP.
  - NAPQI depletes glutathione stores and starts to accumulate.
  - Glutathione reduces NAPQI into nontoxic mercaptate conjugate.
  - NAPQI binds nonspecifically to intracellular proteins, causing cell dysfunction.

CLINICAL SIGNS OF TOXICITY

- Acetaminophen (APAP) overdose is usually marked by a lack of clinical signs or symptoms in the first 24 hours.
- Toxic level of APAP at 4 hours is 150 µg/mL.
- Twenty-four to 48 hours, begin to have right upper quadrant pain with elevation of liver function tests and prothrombin time/international normalized ratio.
- Forty-eight to 96 hours, severe liver dysfunction with coagulopathy, renal failure, death.
- Survivors will recover hepatic function over next 2 weeks.

MANAGEMENT

- Decontamination:
  - Activated charcoal.
  - Avoid emesis (this delays *N*-acetylcysteine [NAC] administration).
  - Lavage only for coingestants.
- Administration of NAC:
  - Acts as glutathione precursor or substitute.
  - Acts as a sulfate precursor.
  - Directly reduces NAPQI back to APAP.
  - After 24 hours, acts as a hepatocellular protectant.
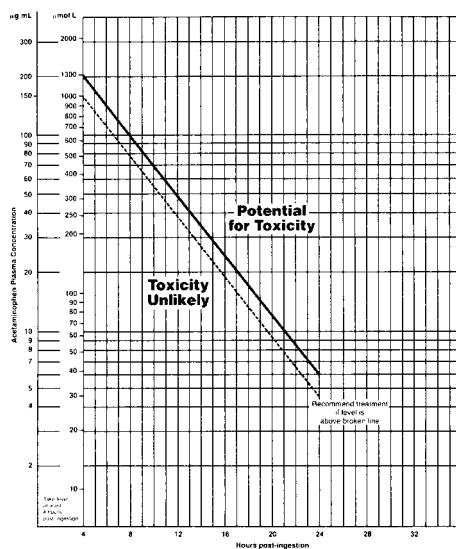
F I G U R E   1 8 - 1 .   Rumack–Matthew nomogram.

(Reproduced, with permission, from *Management of Acetaminophen Overdose.* McNeil Consumer Products Co., 1986.)

Aspirin

Acetylsalicylic acid (ASA).

EXPOSURE

- Present in over 200 oral and topical preparations (aspirin, Pepto-Bismol, Alka-Seltzer, Dristan, Ben-Gay, Tiger Balm, etc.).
- Present in Oil of Wintergreen.

MECHANISM OF TOXICITY

- Absorption:
  - Normally 1 to 2 hours.
  - 4 to 6 hours in overdose (delayed gastric emptying, concretion formation).
- Distribution:
  - Normally weak acid that remains ionized.
  - Acidosis in overdose makes it easier for ASA to penetrate tissues.
- Metabolism:
  - Conjugated in liver via first-order kinetics.
  - Liver enzymes saturated in overdose, zero-order kinetics.
- Elimination:
  - Small amount of free salicylate excreted in urine.
  - Maximizing urinary excretion may be beneficial in overdose. Consider for levels > 50 mg/dL.

CLINICAL SIGNS OF TOXICITY

- Respiratory:
  - Tachypnea/hyperpnea
  - Noncardiogenic pulmonary edema
- CNS:
  - Tinnitus
  - Headache
  - Cerebral edema/coma
- Other:
  - Platelet dysfunction
  - Hyperthermia

ACID–BASE DISTURBANCES

- Respiratory alkalosis: Direct stimulation of medulla (tachypnea/hyperpnea).
- Metabolic acidosis: Uncoupling of oxidative phosphorylation leads to anaerobic metabolism with lactic acidosis.
- Metabolic alkalosis: Vomiting, diaphoresis, and tachypnea cause dehydration and volume contraction.

MANAGEMENT

- Check for presence of ASA: Ferric chloride—spot test turns urine purple if ASA is present.
- Decontamination: Activated charcoal.
- Respiratory support: Intubate if necessary to maintain hyperventilation (respiratory alkalosis buffers metabolic acidosis).
- IV fluids: Correct dehydration with glucose-containing crystalloid fluid.

Causes of noncardiogenic pulmonary edema: **MOPS**
Meprobamate/**M**ountain sickness
Opioids
Phenobarbital
Salicylates

A 67-year-old woman presents with 6 days of headache and 2 days of "ringing in the ears" and fever. She is breathing deeply at a rate of 22/min. *Think: Aspirin toxicity.*

If you intubate a patient with metabolic acidosis and compensatory respiratory alkalosis, do not impede their ability to "blow off" acid. Use paralytics with extreme caution, and set the vent at a high minute ventilation.

"Normal" ASA level does not rule out toxicity— repeat every 1 to 2 hours until levels decline and clinical status improves.

- Urine alkalinization: Maintain urine pH around 8.0 to trap ionized ASA in urine. Alkalinize with bicarbonate drip.
- Extracorporeal removal: Hemoperfusion is better at removing ASA; hemodialysis is better for correcting acid–base and electrolytes (consider for ASA level > 100 mg/dL or as indicated clinically).

Iron

Iron is an essential component of human red blood cells (RBCs), hemoglobin (Hgb), myoglobin, and cytochromes.

EXPOSURE

Accidental or intentional ingestion of iron-containing tablets.

MECHANISM OF TOXICITY

- Less than 10% of ingested iron is bioavailable:
  - Iron absorbed by intestinal mucosa, stored as ferritin.
  - Transported throughout body, complexed with transferrin.
  - Elimination is primarily via sloughing of intestinal mucosa (ferritin).
- Overdose:
  - Ingested iron overwhelms protein carriers, enters via passive diffusion.
  - Iron is corrosive to GI mucosa, enters circulation directly.
- Free iron in circulation leads to toxicity:
  - Direct corrosive effect on GI tract.
  - Causes vasodilation and myocardial depression.
  - Disrupts oxidative phosphorylation, which leads to buildup of lactic acid (metabolic acidosis).
  - Delayed hepatotoxicity.

CLINICAL SIGNS OF TOXICITY

Stage I: 1 to 6 Hours
- GI symptoms:
  - Abdominal pain
  - Nausea, vomiting, diarrhea
  - Hematemesis

Stage II: 6 to 24 Hours
- Resolution of GI symptoms
- Early shock

Stage III: Variable Time Course
- Shock.
- Metabolic acidosis.
- Coagulopathy.
- Multiorgan failure may occur.

Stage IV: 2 to 5 Days
- Hepatic insufficiency, may progress to failure.

Stage V: 4 to 6 Weeks After Ingestion
- Gastric outlet obstruction.

Patients can die in any stage of iron toxicity.

Toxic dose of iron:
< 20 mg/kg nontoxic
> 60 mg/kg severe toxicity
Calculate on basis of amount of elemental iron ingested.
Toxic level of iron:
< 300 μg/dL nontoxic
> 500 μg/dL moderate toxicity
> 1,000 μg/dL severe toxicity

MANAGEMENT

- Obtain serum level.
- Supportive care.
- Decontamination:
  - WBI effective at clearing large GI loads.
  - No ipecac (iron already induces emesis).
  - Lavage usually ineffective (large iron pills).
  - Charcoal does not adsorb well to iron.
- Deferoxamine:
  - Chelates free iron to form ferrioxamine (water soluble, excreted in urine).
  - Ferrioxamine turns urine "vin rosé" (or rusty brown) color.
  - Dose: 5 to 15 mg/kg/hr (not given for acute toxicity treatment).

► CARDIOVASCULAR DRUGS

Beta Blockers

EXPOSURE

Commonly prescribed for hypertension, hyperthyroidism.

MECHANISM OF TOXICITY

- Stimulation of beta-adrenergic receptor causes an increase in intracellular cAMP → phosphorylation of calcium channels (opens channels).
- Increased calcium influx triggers release of intracellular calcium stores → excitation–contraction coupling.
- Pharmacologic differences among beta blockers:
  - Selectivity: Agents may have beta-1 or beta-2 selectivity, which is lost in overdose.
  - Solubility: More lipid-soluble agents are more likely to penetrate CNS.
  - Agents with intrinsic sympathomimetic activity may present atypically.
  - Membrane stabilizing: These agents may cause sodium channel blockade.

CLINICAL SIGNS OF TOXICITY

- Bradycardia and hypotension.
- Sinus node suppression.
- Slowed atrioventricular (AV) nodal conduction.
- QRS widening with agents that block Na channels.
- Decreased myocardial contractility.
- Decreased cardiac output.
- Smooth muscle relaxation, peripheral vasodilation.
- Lipophilic agents may cause sedation and/or seizures (penetrate CNS).
- Beta-2 receptor blockade may lead to bronchospasm.

MANAGEMENT

- Supportive treatment.
- Fluid resuscitation.
- Decontamination: Gastric lavage (if within 1 to 2 hours) and activated charcoal.

Beta-adrenergic receptors:

Beta-1:
- Myocardium ($\uparrow$ inotropy)
- Eye ($\uparrow$ aqueous humor)
- Kidney ($\uparrow$ plasma renin)

Beta-2:
- Smooth muscle relaxation
- Liver (glycogenolysis, gluconeogenesis)

Beta-3:
- Adipose tissue (lipolysis)

- Glucagon bypasses beta receptor to increase intracellular cyclic adenosine monophosphate (cAMP).
- Catecholamines (dopamine/NE) for pressor support.
- Phosphodiesterase inhibitors (amrinone) block cAMP breakdown.
- Transcutaneous/transvenous pacing, intra-aortic balloon pump (IABP), bypass as indicated.

Calcium Channel Blockers

EXPOSURE

Commonly prescribed for hypertension.

MECHANISM OF TOXICITY

- Blockade of L-type calcium channels in cell membranes.
- Decreased calcium influx disrupts excitation–contraction coupling.
- Different classes of calcium channel blockers:
  - Both phenylalkylamines (verapamil) and benzothiazepines (diltiazem) cause decreased myocardial contractility and conduction, as well as vasodilation.
  - Dihydropyridines (nifedipine, amlodipine) cause mostly peripheral vasodilation.

CLINICAL SIGNS OF TOXICITY

- Hypotension
- Bradycardia
- Decreased conduction/automaticity
- Hypoperfusion
- Lactic acidosis
- Insulin resistance, hyperglycemia/hyperkalemia

MANAGEMENT

- Supportive care (including endotracheal intubation as indicated).
- Fluid resuscitation.
- Decontamination with lavage (if early) and charcoal.
- Consider WBI for sustained release preparations.
- Correct acidosis.
- IV calcium:
  - Increases gradient across calcium channel.
  - Stabilizes membranes in presence of hyperkalemia.
- Glucagon acts to increase cAMP and phosphorylate calcium channels.
- Electrical pacing and/or pressors (dopamine) as indicated.
- Refractory cases: Consider amrinone (inhibits cAMP breakdown), insulin (inotrope/chronotrope), IABP, or dialysis.

Causes of bradycardia:
PACED
Propranolol (beta blockers)
Anticholinesterase drugs
Clonidine/calcium channel blockers
Ethanol, other alcohols
Digoxin/**D**arvon (opiates)

Toxicity from chronic digoxin use may be seen with normal levels.

Cardiac Glycosides (Digoxin)

Commonly used in the treatment of congestive heart failure (CHF) and supraventricular tachycardias.

EXPOSURE

- Digoxin
- Foxglove plant
- Oleander plant

- Inhibits Na^+-K^+ ATPase pump:
  - Increases intracellular Na^+, extracellular K^+.
  - Less Ca^{2+} is pumped out in exchange for Na^+ (increased intracellular $Ca^{2+} \rightarrow$ increased inotropy).
  - Membrane resting potential becomes less negative (as Na^+ and Ca^{2+} accumulate inside cell), leads to increased automaticity (tachydysrhythmias).
- Increases vagal tone (leads to bradydysrhythmias).
  - Decreases conduction through AV node.
  - Also leads to sinus bradycardia.

CLINICAL SIGNS OF TOXICITY

- Cardiac toxicity (wide range of rhythm disturbances):
  - Sinus bradycardia/block.
  - Atrial fibrillation/flutter.
  - AV node blocks (junctional rhythms).
  - Premature ventricular contractions, ventricular tachycardia/fibrillation.
- Hyperkalemia (inhibition of Na^+-K^+ pump).
- Nausea, vomiting, and headaches are common in acute overdose.
- Visual disturbances:
  - Amblyopia
  - Photophobia
  - Yellow-green halos around light

MANAGEMENT

- Decontamination with activated charcoal for acute overdose (avoid lavage, as it may increase vagal tone).
- Treat bradydysrhythmias—atropine and/or pacing.
- Treat tachydysrhythmias—lidocaine, phenytoin.
- Treat hyperkalemia—indicator for digoxin toxicity as above; severe hyperkalemia: Insulin/glucose and bicarbonate.
- Treat hypokalemia—may potentiate digoxin toxicity, replace as indicated (may see toxic effects with normal digoxin levels).
- Digibind: Digoxin-specific antibody fragments bind to digoxin in serum, eliminated by kidneys. Use for:
  - Ventricular dysrhythmias.
  - Hemodynamically significant bradydysrhythmias.
  - Hyperkalemia > 5.0 in setting of overdose.
  - Digoxin level > 4 ng/mL.
- Digibind dosing:
  - Unknown ingestion (empiric): 5 to 10 vials.
  - Known ingestion: 1.6 × amount ingested.
  - Known digoxin level: [wt(kg) × level (ng/mL)/100].

Hypoglycemics

GLUCOSE METABOLISM

- Serum levels maintained by balance between three mechanisms:
  - Gut absorption of ingested glucose.
  - Glycogenolysis—mobilization of liver stores.

Salvador Dali mustache: Scooped ST segments are seen with digoxin use at therapeutic levels *(not an indicator of toxicity).*

Pathognomic for digoxin toxicity: Bidirectional ventricular tachycardia.

Digoxin levels are meaningless once Digibind is administered.

Never use calcium as therapy for hyperkalemia in digoxin overdose (reports of myocardial tetany, i.e., "stone heart" have been reported).

Without Digibind therapy, mortality in digoxin overdose is:
- 100% for K > 5.5 mEq/L
- 50% for K = 5.0–5.5 mEq/L
- 0% for K < 5.0 mEq/L

- Gluconeogenesis—major mechanism for glucose control in hypoglycemic states.
- Physiologic response to hypoglycemia:
  - CNS: Confusion, lethargy, seizures, coma, focal neurologic deficits
  - Autonomic response.
  - Release of counterregulatory hormones (epinephrine, glucagon, etc.).
  - Diaphoresis, tremors, palpitations, anxiety.

Insulin

EXPOSURE

Immediate- (Lispro), short- (regular), intermediate- (NPH), and long- (Ultralente) acting formulations.

MECHANISM OF TOXICITY

- Secreted by beta-islet cells of pancreas.
- Stimulates uptake/utilization of glucose in body.
- Insulin absorption variable in overdose.

CLINICAL SIGNS OF TOXICITY

- Hypoglycemia.
- Hypothermia.
- Delirium, coma, seizure, or focal neurologic deficit.

MANAGEMENT

- IV dextrose.
- Glucagon will not help, because hepatic glycogen stores are already depleted.

Sulfonylureas

Oral hypoglycemic agents.

EXPOSURE

Commonly prescribed for type 2 diabetes.

MECHANISM OF TOXICITY

Increases endogenous insulin secretion and sensitivity to insulin in peripheral tissues.

CLINICAL SIGNS OF TOXICITY

- Long duration of hypoglycemia.
- Hypothermia, hyponatremia, and disulfiram-like reactions have been reported.
- Delirium, coma, seizure, or focal neurologic deficit.

MANAGEMENT

- IV dextrose.
- Decontamination with charcoal for overdose.

- Octreotide (somatostatin analog) inhibits glucose-stimulated insulin release.
- Diazoxide inhibits beta-islet cell insulin release (removed from U.S. market).
- Chlorpropamide: Alkalinizing urine speeds elimination.

Biguanides

Oral hypoglycemic agents.

EXPOSURE

- Phenformin (removed from U.S. market)
- Metformin (Glucophage)

MECHANISM OF TOXICITY

- Increases peripheral sensitivity to insulin.
- Suppresses gluconeogenesis.

CLINICAL SIGNS OF TOXICITY

- Hypoglycemia (rarely).
- Hypothermia.
- Delirium, coma, seizure, or focal neurologic deficit.
- Lactic acidosis.

MANAGEMENT

- IV dextrose.
- Decontaminate with charcoal for overdose.
- Consider bicarbonate for management of significant acidosis.
- Consider dialysis for correcting large fluid and electrolyte shifts.

All patients with hypoglycemia due to oral hypoglycemics should be admitted.
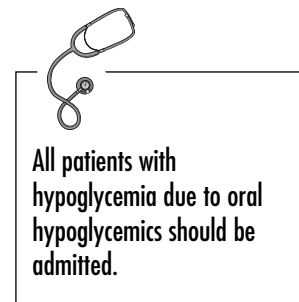
► **ANTIDEPRESSANTS**

Tricyclic Antidepressants (TCAs)

TCAs are responsible for more drug-related deaths than any other prescription medication.

EXPOSURE

Commonly prescribed for depression, chronic pain.

MECHANISM OF TOXICITY

- Blocks reuptake of dopamine, serotonin, NE.
- Binds to GABA receptor, lowering seizure threshold.
- Sodium channel blockade: Wide QRS.
- Alpha-adrenergic blockade: Orthostatic hypotension.
- Antihistamine effect: Sedation.
- Anticholinergic effect.

Action of sodium bicarbonate in heart in TCA overdose is due to the *sodium* component; it alters the interaction between the drug and sodium channels.

CLINICAL SIGNS OF TOXICITY

- Hyperthermia, tachycardia, lethargy/coma, seizures/myoclonus.
- Anticholinergic (dry skin/membranes, mydriasis, urinary retention, etc.).
- Abnormal electrocardiogram (ECG) (Figure 18-2):
    - Sinus tachycardia, right axis deviation (RAD), and prolongation of PR, QRS, and QT intervals.
    - QRS widening secondary to sodium channel blockade:
        - QRS < 100, no significant toxicity.
        - QRS > 100, seizures in one third of patients.
        - QRS > 160, ventricular dysrhythmias in one half of patients.
    - RAD is most apparent in aVR → terminal 40 msec positive R wave (this finding is sensitive but not specific for presence of TCA).

MANAGEMENT

- Decontamination:
    - Charcoal is effective at binding TCA.
    - Lavage only effective early in course.
    - Ipecac contraindicated.
- Treat hypotension:
    - Treat initially with IV crystalloid.
    - NE as pressor (if necessary).
    - Bicarbonate for refractory hypotension.
- Treat seizures:
    - Treat with benzodiazepines/barbiturates.
    - Consider general anesthesia and paralytics for refractory seizures.
    - Avoid phenytoin (risk of dysrhythmia).
- Treat dysrhythmias:
    - Sodium bicarbonate is first-line intervention.
    - Lidocaine/bretylium as indicated.
    - Cardioversion for unstable dysrhythmias.
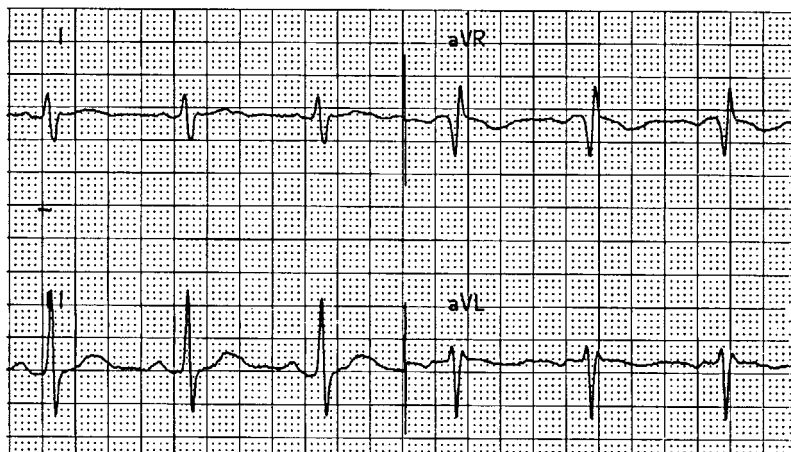
F I G U R E   1 8 - 2 .   ECG of TCA toxicity.

(Reproduced, with permission, from Goldfrank LR et al. *Goldfrank's Toxicology*, 7th ed. New York: McGraw-Hill, 2002: 123.)

- Sodium bicarbonate:
  - Shown to improve conduction/contractility and decrease ectopy.
  - Indications for sodium bicarbonate:
    - Refractory hypotension
    - QRS widening > 100 msec
    - Ventricular dysrhythmias
  - Goal of therapy is to maintain narrow QRS. Avoid excessive alkalemia (pH > 7.55).

Selective Serotonin Reuptake Inhibitors (SSRIs)

EXPOSURE

Commonly prescribed for depression, premenstrual syndrome.

MECHANISM OF TOXICITY

- Selectively inhibit reuptake of serotonin without affecting dopamine/NE.
- No direct effect on presynaptic/postsynaptic receptors (fewer side effects than TCAs).
- High toxic-to-therapeutic drug ratio (lower incidence of toxicity from overdose).
- Extrapyramidal symptoms (EPS): Dystonic reactions, parkinsonism, etc.
- Hyponatremia secondary to syndrome of inappropriate antidiuretic hormone.
- Seizures, QT prolongation: Rare, mostly with citalopram (Celexa).

MANAGEMENT

- Decontamination with activated charcoal.
- Benzodiazepines/barbiturates for seizures.
- Sodium bicarbonate for wide QRS.
- For serotonin syndrome:
  - Supportive care, cooling.
  - Consider cyproheptadine (antihistamine with antiserotonin properties).
  - Benzodiazepines, paralytics as needed.

Monoamine Oxidase Inhibitors (MAOIs)

EXPOSURE

Commonly prescribed for depression.

MECHANISM OF TOXICITY

- MAO: Enzyme found in nerve terminals; degrades epinephrine, norepinephrine (NE), dopamine, and serotonin.
- MAOIs: Form an irreversible covalent bond with MAO in nerve terminals.
- Increase the amount of biogenic amines available at nerve terminals:
  - Increase catecholamines.
  - Synergy with SSRIs may lead to serotonin syndrome.
  - Tyramine-containing foods may cause sympathomimetic crisis.
  - Other drugs (cocaine, amphetamines) may contribute to or cause sympathomimetic crisis.

Serotonin syndrome:
Usually results from combination of SSRI with:
- MAOI
- Cocaine
- Methylene dioxy metamphetamine (MDMA) (ecstasy)
- Lithium/tryptophan

Characterized by:
- Hyperthermia
- Tachycardia
- Rigidity
- Hyperreflexia
- Confusion/agitation

Do not use the following agents in patients on MAOIs (partial list—check all agents given to patients on MAOIs):
- Other MAOIs
- Amphetamines
- Dopamine
- Epinephrine, NE
- Meperidine
- Buspirone
- Dextromethorphan
- SSRIs
- Tyramine-containing foods
- Cocaine

CLINICAL SIGNS OF TOXICITY

- Tachycardia, hyperthermia, hypertension, mydriasis, agitation.
- Eventual catecholamine depletion can cause sympatholytic crisis (hypotension, bradycardia, CNS depression).
- Serum levels of MAOI correlate poorly with toxicity.
- Onset of toxicity may be delayed up to 24 hours.

MANAGEMENT

- Supportive care.
- Gastric lavage, if acute.
- Discontinue drugs that may interact with MAOI.
- Control hypertension (phentolamine, nitroprusside).
- Treat seizures, hyperthermia, and rigidity with benzodiazepines.
- Treat ventricular dysrhythmias with lidocaine or procainamide.
- Treat hypotension with fluids and NE.

Lithium

EXPOSURE

Commonly prescribed for bipolar disorder.

MECHANISM OF TOXICITY

- Overall, not well understood.
- Increases synthesis and turnover of serotonin.
- Downregulates number of adrenergic receptors (beta and alpha-2).
- Inhibits adenylate cyclase (decreases cAMP).
- Inhibits inositol monophosphatase.
- Deposits in bone and other tissues, forming a reservoir of lithium.
- Competes with other molecules of similar size.
- Ninety-five percent is excreted in urine (glomerular filtration rate dependent).

CLINICAL SIGNS OF TOXICITY

- Acute toxicity:
  - Nausea, vomiting, abdominal pain.
  - Acute ingestions can tolerate higher Li+ levels without toxicity.
- Chronic toxicity:
  - Resting tremor, hyperreflexia, seizure, coma, EPS.
- Acute or chronic:
  - Prolonged QT, flipped T waves (hypokalemia).
  - Nephrogenic diabetes insipidus.

MANAGEMENT

- Decontaminate with WBI for acute ingestions.
- Fluid resuscitation.
- Kayexalate is effective at binding lithium but requires massive doses so not done in practice.
- Hemodialysis for:
  - Lithium level > 4 (acute) or > 2.5 (chronic).
  - Significant CNS or cardiovascular toxicity.
  - Renal failure.
  - Heart failure.

Lithium does not bind to charcoal.
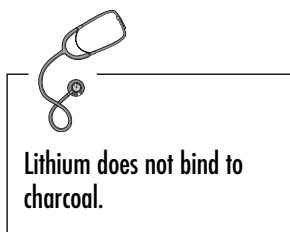
Antipsychotics

EXPOSURE

- Older "typical" agents:
  - Haloperidol, chlorpromazine.
  - More effective in controlling positive symptoms (hallucinations, delusions).
- Newer "atypical" agents:
  - Olanzapine, risperidone.
  - More effective at controlling negative symptoms (apathy, blunted affect).

MECHANISM OF TOXICITY

- Older agents block D_2 (dopaminergic) receptor, possess antihistamine, anticholinergic.
- Newer agents block $5\text{-}HT_2$ (serotonergic) receptor.
- Neuroleptic malignant syndrome (NMS) caused by central dopaminergic blockade.

CLINICAL SIGNS OF TOXICITY

- EPS.
- Orthostatic hypotension with tachycardia.
- Sedation.

MANAGEMENT

- Decontamination.
- Supportive care.
- Treat EPS symptoms with IV diphenhydramine and discontinue agent if possible.
- Treat NMS with discontinuation of agent, cooling, benzodiazepines, and dantrolene. Consider carbidopa/levodopa to increase dopamine activity.

▶ **ANTICONVULSANTS**

Phenytoin

- First-line agent useful for all types of seizures except absence.
- Blocks voltage-sensitive and frequency-dependent sodium channels in neurons.
- Suppresses ability of neurons to fire action potentials at high frequency.
- Fosphenytoin (Cerebyx):
  - Phenytoin prodrug, soluble in aqueous solution with pH ~8.8.
  - Converted to phenytoin in blood and peripheral tissues.
  - Well tolerated both IV and IM routes (fewer side effects—faster administration possible).

EXPOSURE

Commonly prescribed for seizure disorder.

MECHANISM OF TOXICITY

In overdose, kinetics change from first-order to zero-order.

Antipsychotics:
Toxicity can occur with overdose or therapeutic dose.

EPS:
- Dystonic reactions
- Akathisia (motor restlessness)

NMS:
- Hyperthermia
- Altered mental status
- Autonomic instability
- Muscular rigidity

Toxicologic causes of hyperthermia: **NASA**
NMS/nicotine
Antihistamines
Salicylates/ sympathomimetics
Anticholinergics/ antidepressants (MAOIs)

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> The cardiovascular side effects of IV phenytoin are due to the diluent.

CLINICAL SIGNS OF TOXICITY

- CNS toxicity: Nystagmus, lethargy, ataxia, seizures, coma.
- Local effects (IM): Crystallization, abscess, tissue necrosis.
- Hypersensitivity: Systemic lupus erythematosus, toxic epidermal necrolysis, Stevens–Johnson syndrome (1 to 6 weeks after initiating therapy).
- Gingival hyperplasia.
- Cardiovascular toxicity:
  - Almost always seen as infusion rate–related complication of IV therapy due to diluent.
  - Phenytoin diluent: Propylene glycol, ethanol solution (pH ~12).
  - Hypotension, bradycardia, AV blocks, asystole.
  - ECG: Prolonged PR and QRS, nonspecific ST-T wave changes.

MANAGEMENT

- Use multiple-dose charcoal for oral overdose.
- Hemodialysis and hemoperfusion are ineffective.
- Supportive care; discontinue infusion for signs of toxicity.

Carbamazepine (Tegretol)

- First-line agent useful for all types of seizures except absence.
- Also used in the management of trigeminal neuralgia and bipolar disorder.
- Available only in oral formulation (no parenteral forms).

CLINICAL SIGNS OF TOXICITY

- CNS: Nystagmus, ataxia, dystonia, seizures, coma.
- Cardiac: QRS widening, prolonged QT, AV blocks.

MANAGEMENT

- Decontamination with multiple-dose charcoal.
- Hemodialysis ineffective.
- Hemoperfusion with charcoal is effective.
- Bicarbonate for QRS widening > 100 msec.
- Benzodiazepines for seizures.

Valproic Acid

- Used for the treatment of absence, myoclonic, and tonic–clonic seizures.
- Also used as mood stabilizer for treatment of bipolar disorder.
- Metabolized extensively in liver, with several biologically active metabolites (2-n-valproic acid is active and accumulates in CNS and other tissues).

CLINICAL SIGNS OF TOXICITY

- Nausea/vomiting and abdominal pain.
- Cerebral edema from accumulation of metabolites.
- Respiratory depression, cardiac arrest.
- Metabolic derangements:
  - Hyperammonemia +/– hypocarnitinemia.
  - Metabolic acidosis.

- Hypernatremia.
- Hypocalcemia.
- Hepatotoxicity.

Management

- Supportive care (including intubation as required).
- Decontamination with multiple-dose activated charcoal.
- Hemodialysis improves clearance; reserve for most toxic patients.
- Carnitine supplementation in hyperammonemic patients.

▶ ALCOHOLS

General Principles

- Group of structurally similar molecules with common R–OH group.
- Level of inebriation after consumption is related to number of carbons in R group (methanol < ethanol < ethylene glycol < isopropyl alcohol).
- Calculated serum osmolarity: $2 (Na^+) + (BUN/2.8) + (Glucose/18)$.
- Osmol gap: Difference between measured and calculated serum osmolarity.
- Estimate toxic alcohol level as follows: Osmol gap = alcohol level/N (where N = molecular weight/10, i.e., N = 3.2 for methanol, 4.6 for ethanol, etc.).
- "Normal" osmol gap is between −14 and +10; baseline gap is usually unknown.
- Patients with "normal" gap may in fact be elevated from their baseline.
- Elevated gap of 10 corresponds to methanol level of 32, ethanol level 46, etc.
- Bottom line: Elevated gap is useful; normal gap does not rule out toxic ingestion.

Ethanol

Most commonly used and abused intoxicant in the United States.

Exposure

- Ethanol frequently consumed with other intoxicants (most common is cocaine).
- Ethanol + cocaine → cocaethylene (40 times more potent than regular cocaine).

Mechanism of Toxicity

- CNS depressant that cross-reacts with other depressants (benzodiazepines, barbiturates).
- Majority of ethanol is absorbed in proximal small bowel.
- Up to 10% is eliminated by lungs, urine, and sweat.
- Remainder is metabolized by liver as follows:
  - Catalyzed by alcohol dehydrogenase and aldehyde dehydrogenase (inhibited by disulfiram).
  - Microsomal alcohol oxidizing system.

BAL:
- One drink equals ~25 to 35 mg/dL BAL.
- Average person metabolizes 15 to 20 mg/dL/hr.
- Chronic drinkers metabolize ~30 mg/dL/hr.

- Elimination follows zero-order kinetics:
  - Approximately 15 to 20 mg/dL/hr in normal individuals.
  - Approximately 30 mg/dL/hr in chronic alcoholics.

CLINICAL SIGNS OF TOXICITY

- Slurred speech.
- Nystagmus.
- Disinhibition.
- CNS depression.
- Degree of intoxication clinically correlates poorly with blood alcohol level (BAL).

MANAGEMENT

Management of acute intoxication is supportive:
- Thiamine, folate, IV fluids.
- "Banana bag" consists of 1 L of D_5NS with 100 mg thiamine, 1 mg folate, and 1 amp of multivitamin (which turns bag yellow).

ALCOHOLIC KETOACIDOSIS (AKA)

- Anion gap acidosis in heavy alcohol user who has temporarily stopped drinking and eating.
- Acid–base: Frequently metabolic acidosis with respiratory alkalosis (compensatory) and metabolic alkalosis (vomiting). pH may be normal.
- Treat with IV fluid replacement, IV glucose, and thiamine.

Methanol

EXPOSURE

Product of wood distillation, found in:
- Antifreeze
- Windshield wiper fluids
- Paint thinners

MECHANISM OF TOXICITY

Toxicity is secondary to formic acid (Figure 18-3):
- Formic acid accumulation produces high-anion-gap metabolic acidosis.
- Formaldehyde accumulates in retina causing "snowfield vision."
- Onset of symptoms is usually delayed ~12 to 18 hours until metabolites form (delay is even longer if ethanol is coingested).
- Folate is a required cofactor to degrade formic acid to carbon dioxide and water.

CLINICAL SIGNS OF TOXICITY

- CNS depression.
- Visual changes (funduscopic examination demonstrates optic papillitis and edema).
- Abdominal pain (direct GI mucosal irritation).
- High anion gap metabolic acidosis.
- Severity of acidosis is better predictor of outcome than methanol level.
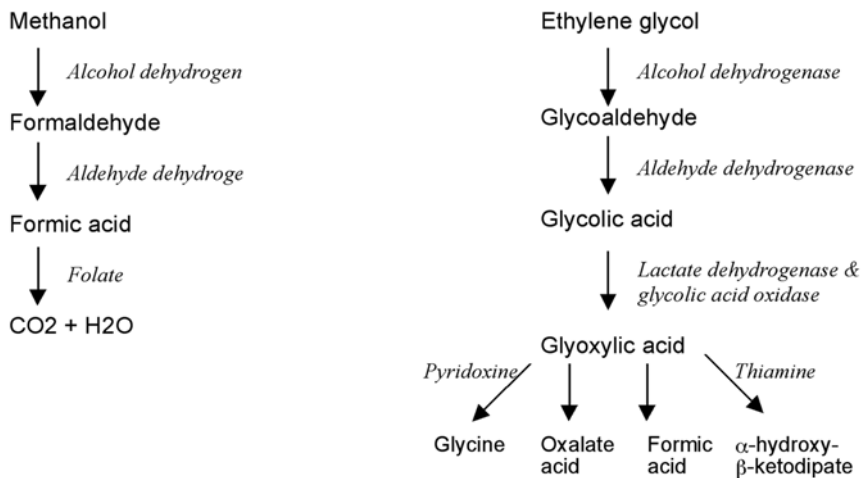
FIGURE 18-3. Metabolism of methanol and ethylene glycol.

Methanol
↓ *Alcohol dehydrogen*
Formaldehyde
↓ *Aldehyde dehydroge*
Formic acid
↓ *Folate*
$CO_2 + H_2O$

Ethylene glycol
↓ *Alcohol dehydrogenase*
Glycoaldehyde
↓ *Aldehyde dehydrogenase*
Glycolic acid
↓ *Lactate dehydrogenase & glycolic acid oxidase*
Glyoxylic acid

Pyridoxine / *Thiamine*

Glycine   Oxalate acid   Formic acid   α-hydroxy-β-ketodipate

MANAGEMENT

- Charcoal:
  - Binds poorly to all alcohols.
  - Rapid GI absorption of alcohols limits utility of charcoal.
- Folate: Hastens degradation of formic acid. Dose is 50 mg IV q4h; the first dose is given as activated folate (leucovorin).
- Fomepizole:
  - Competitive inhibitor of alcohol dehydrogenase.
  - Blocks metabolism of methanol to toxic metabolites.
  - Affinity for alcohol dehydrogenase 8,000 times greater than methanol.
  - Dose is 15 mg/kg IV, then four doses at 10 mg/kg each 12 hours apart.
- Ethanol:
  - Affinity for alcohol dehydrogenase 20 times greater than methanol.
  - BAL of ethanol should be maintained ~100 to 150 mg/dL.
  - Methanol is cleared renally (slow) while on ethanol drip.
  - Dose is 8 g/kg IV load, then continuous infusion at 11 g/hr (average drinker) or 15 g/hr (heavy drinker).
- Dialysis:
  - Indicated for large ingestions or with severe acidosis.
  - Indicated when methanol level > 25 mg/dL.

Ethylene Glycol

Colorless, odorless, and sweet-tasting liquid.
EXPOSURE

- Coolant/antifreeze
- Commercial solvents
- Detergents
- Polishes
- De-icers

Blood glucose levels need to be monitored during ethanol therapy.

Ethylene glycol is often consumed as an ethanol substitute or in suicide attempts.

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Funduscopy in ethylene glycol poisoning is normal; this distinguishes it from methanol poisoning.

Hallmark of ethylene glycol toxicity: Calcium oxalate crystals in urine (50% present).

How to calculate osmolarity: $2 \times Na + BUN/2 + Glucose/18 + ETOH/4.6$ If there is a difference between the measured (lab) and calculated osmolarity, consider toxic alcohols.

MECHANISM OF TOXICITY

Toxicity is secondary to toxic metabolites:
- Ethylene glycol $\rightarrow$ glycoaldehyde causes lactate formation.
- Glyoxylic acid broken down to glycine and ketoadipate (nontoxic).
- When above pathways are saturated, formic acid and oxalic acid are formed.
- Formic acid contributes to metabolic acidosis as with methanol.
- Oxalic acid crystallizes (calcium oxalate) causing renal stones and hypocalcemia.

CLINICAL SIGNS OF TOXICITY

- Early phase (1 to 12 hours): CNS depression, slurred speech, ataxia.
- Cardiopulmonary phase (12 to 24 hours): Tachycardia, tachypnea, CHF, adult respiratory distress syndrome (ARDS).
- Nephrotoxic phase (24 to 72 hours): Oliguric renal failure, acute tubular necrosis, hypocalcemia.

MANAGEMENT

- Quickly absorbed by gut and only 50% adsorbed to charcoal; charcoal has limited benefit.
- Obtain blood levels of ethanol, methanol, and ethylene glycol.
- Ethanol infusion or fomepizole to competitively inhibit toxic pathways.
- Calcium supplementation as indicated for hypocalcemia.
- Pyridoxine and thiamine supplementation to preserve nontoxic pathways.
- Dialysis as indicated clinically or for ethylene glycol level > 25 mg/dL.
- Asymptomatic patients are admitted for observation due to concern for delayed manifestation of toxicity.

Isopropanol

Clear liquid with bitter burning taste and characteristic odor.

EXPOSURE

- Rubbing alcohol
- Disinfectants
- Skin and hair products

MECHANISM OF TOXICITY

- Twice as potent as ethanol in causing CNS depression, with longer half-life.
- Metabolism of isopropanol follows first-order (concentration dependent) kinetics.

CLINICAL SIGNS OF TOXICITY

The hallmark of isopropanol ingestion is ketosis and elevated osmolal gap without significant acidosis.

- Hallmark of isopropanol ingestion is ketosis without significant acidosis:
  - Acetic acid and formic acid formation contribute to mild acidosis.
  - Acetone formation causes ketonemia/ketonuria in absence of hyperglycemia.
- Marked CNS depression (greater than ethanol).
- Hypotension secondary to peripheral vasodilatation.
- Hemorrhagic gastritis from direct mucosal irritation.

MANAGEMENT

- Rapidly absorbed and binds poorly to charcoal.
- Supportive treatment for coma/respiratory depression.
- IV fluids (pressors if necessary) for hypotension.
- H$_2$ blockers, nasogastric tube, and transfusion as indicated for hemorrhagic gastritis.
- Dialysis for refractory hypotension or peak level > 400 mg/dL.
- Asymptomatic patients can be discharged after 6 to 8 hours.

▶ DRUGS OF ABUSE

Cocaine

Naturally occurring alkaloid extract of *Erythroxylon coca* (South American plant).

EXPOSURE

- Cocaine hydrochloride: Absorbed across all membranes (usually snorted or injected).
- Cocaine alkaloid (crack): Stable to pyrolysis (may be inhaled), rapid onset/short duration.

MECHANISM OF TOXICITY

- Mechanisms of action:
  - Blocks presynaptic reuptake of biogenic amine transmitters: Dopamine, serotonin, NE.
  - Local anesthetic effect by blocking fast sodium channels.
- Initial euphoria secondary to release of biogenic amines, subsequent dysphoria secondary to depletion of neurotransmitters (dopamine).

CLINICAL SIGNS OF TOXICITY

- Euphoria followed by dysphoria.
- Hypertension.
- Tachycardia, dysrhythmias.
- Chest pain (coronary vasoconstriction).
- Seizures, infarction, hemorrhage (cerebral vasoconstriction).
- Cocaine psychosis.
- Rhabdomyolysis.
- Hyperthermia.
- QRS widening (sodium channel blockade) and QT prolongation (potassium channel blockade).
- Adulterants and direct toxicity cause pulmonary edema, hemorrhage, and barotrauma (patients try Valsalva to increase drug effect).
- Mesenteric vasospasm (common in body-stuffers).
- Uterine vasospasm causes abortions, abruption, prematurity, intrauterine growth retardation.
- Cocaine wash-out syndrome.

MANAGEMENT

- Initial supportive therapy includes sedation and cooling measures.
- Decontamination (charcoal and/or WBI) for ingestions (body-packers) (endoscopy contraindicated due to high incidence of bags rupturing).

Causes of toxin-induced seizures:

OTIS CAMPBELL
Organophosphates/**O**ral hypoglycemics
Tricyclic antidepressants
INH/**I**nsulin
Sympathomimetics

Camphor/**C**ocaine
Amphetamines
Methylxanthines
PCP/**P**henol/**P**ropranolol
Benzodiazepine withdrawal
Ethanol withdrawal
Lithium/**L**indane
Lidocaine/**L**ead

Beta blockers are contraindicated in cocaine toxicity; unopposed alpha stimulation may increase blood pressure.

- Benzodiazepines are effective in controlling tachycardia, hypertension, and seizures.
- Aggressive fluid resuscitation to maintain urine output.
- Aspirin/nitrates/morphine for myocardial ischemia.
- Bicarbonate for patients with widened QRS or rhabdomyolysis.
- Nitroprusside or phentolamine for control of severe hypertension (beta blockers contraindicated, unopposed alpha stimulation may increase blood pressure).

Opioids

EXPOSURE

Naturally occurring or synthetic derivatives of poppy plant:
- Morphine
- Codeine
- Fentanyl
- Heroin
- Methadone
- Others

MECHANISM OF TOXICITY

- There are three main opioid receptors: OP1 (formerly δ), OP2 (formerly κ), and OP3 (formerly μ).
- Stimulation of OP3 receptors produces analgesia, cough suppression, euphoria, and repiration depression.

CLINICAL SIGNS OF TOXICITY

- CNS depression.
- Hypothermia, bradycardia, hypotension.
- Miosis (not for every drug in the class).
- Histamine release may contribute to hypotension.
- Respiratory depression.
- Noncardiogenic pulmonary edema.
- Decreased GI motility—obstipation/constipation.

MANAGEMENT

- Respiratory support using bag–valve mask or endotracheal intubation (respiratory depression is the major cause of mortality with opiates).
- Naloxone in incremental doses (titrate to response) (naloxone is pure antagonist at all three opiate receptors).
- Charcoal and WBI for body-packers.
- Patients given naloxone may not leave until effects of naloxone have worn off (so that they do not pass out again in the street).
- Giving naloxone: Dilute 0.4 mg naloxone in 10 cc saline, then administer IV 1 cc at a time to full spontaneous respirations. Do not give more than needed, or you will have an angry, combative patient.
- Obtain acetaminophen level for any combined opioid preparations.

Causes of miosis: **COPS**
Cholinergics/clonidine
Opiates/organophosphates
Phenothiazines/pilocarpine/
pontine bleed
Sedative–hypnotics

Meperidine:
- Can cause seizures
- Is renally excreted
- Can precipitate serotonin syndrome in combination with MAOIs.

Amphetamines

EXPOSURE

- Long history of use and abuse as stimulants and nasal decongestants.
- Currently used in the management of narcolepsy, attention deficit–hyperactivity disorder, and short-term weight reduction.
- Methamphetamine (crystal, ice): High-potency stimulant effect.
- MDMA (ecstasy, X, Adam): Serotonin effects, intensifies emotions.
- Ephedrine: Amphetamine-like structure, used to ward off drowsiness.

MECHANISM OF TOXICITY

- Release of catecholamines (dopamine and NE) from presynaptic nerve terminals.
- Blocks reuptake of catecholamines (presynaptic).
- At higher doses, causes release of serotonin.

CLINICAL SIGNS OF TOXICITY

- Hyperadrenergic: Tachycardia, hypertension, myocardial infarction, dysrhythmias.
- CNS effects: Agitation, seizures, coma, ischemia/hemorrhage, psychosis (hallucinations, etc.) with serotonergic amphetamines.
- Increased metabolism: Hyperthermia, dehydration, rhabdomyolysis.

MANAGEMENT

- Decontamination using activated charcoal for oral ingestions.
- Benzodiazepines for sedation and anticonvulsant.
- External cooling and aggressive rehydration for hyperthermia.
- Phentolamine or nitrates for control of severe hypertension.

Toxic causes of
hypertension: **CT SCAN**
Cocaine
Theophylline

Sympathomimetics
Caffeine
Amphetamines/
    Anticholinergics
Nicotine

Sedative–Hypnotics

A diverse group of drugs that cause sedation and hypnosis, used for:
- Insomnia
- Anxiety
- Seizures
- Alcohol withdrawal
- Anesthesia

EXPOSURE

- Benzodiazepines
- Barbiturates
- GHB (gamma-hydroxybutyrate)
- Others

Causes of hypothermia:
COOLS
CO
Opiates
Oral hypoglycemics
Liquor (alcohols)
Sedative–hypnotics

MECHANISM OF TOXICITY

- Benzodiazepines/barbiturates both work by potentiating GABA:
  - GABA is the primary inhibitory neurotransmitter in the CNS.
  - GABA$_A$ receptor in cell membrane controls chloride ion flow.
  - Receptor has separate binding sites for benzodiazepines and barbiturates.

- GHB:
  - GHB is an endogenous metabolite of GABA.
  - Available by prescription, used to treat cataplexy associated with narcolepsy.
  - Used as a "date-rape drug" secondary to euphoria, aphrodisiac, and amnesia.

CLINICAL SIGNS OF TOXICITY

- Sleepiness and sedation.
- Muscle relaxation.
- May induce general anesthesia.
- May be associated with respiratory depression.
- Tolerance may develop rapidly.
- Benzodiazepine overdose:
  - Isolated benzodiazepine overdose is rarely associated with death.
  - May potentiate other CNS depressants (ethanol, opioids, etc.).
  - Cardiorespiratory depression usually only seen with parenteral administration.
- Barbiturate overdose:
  - Barbiturate overdose has a significant incidence of morbidity/mortality.
  - Confusion/lethargy progresses to coma with hypothermia, cardiovascular collapse, and respiratory arrest.

MANAGEMENT

- Control airway, breathing, and circulation (ABCs).
- Volume replacement and pressors as required for hemodynamic stability.
- Consider lavage (agents cause decreased gut motility) and charcoal.
- Alkalinization of urine promotes elimination of phenobarbital.
- Hemodialysis/hemoperfusion has limited utility in removing drug.
- Antidote:
  - Flumazenil is competitive antagonist at benzodiazepine receptor site.
  - Most appropriate for reversing benzodiazepines administered IV by physicians (conscious sedation).
  - Should not be used in overdose setting, as it may precipitate seizures.

▶ INDUSTRIAL TOXINS

Hydrocarbons

Compounds consisting primarily of carbon and hydrogen.

EXPOSURE

- Household products:
  - Polishes
  - Pine oils
  - Glues
- Petroleum distillates:
  - Kerosene
  - Gasoline

> Sedative–hypnotics are frequently used in suicide attempts, especially in combination with alcohol. Patients should not receive regular prescriptions from the emergency department.

> Flumazenil is of limited use in benzodiazepine overdose due to the risk of seizures.

> - Hydrocarbons with low viscosity and low surface tension are more likely to be aspirated.
> - Halogenated hydrocarbons are associated with myocardial sensitization.

- Abused solvents (inhalants):
  - Nail polish remover
  - Paints, paint stripper
  - Typewriter correction fluid

MECHANISM OF TOXICITY

- The number of carbons determines physical state:
  - 1 to 4 = gas, low viscosity
  - 5 to 19 = liquid, low viscosity
  - 20 to 60 = solids, high viscosity
- Structures:
  - Aliphatics: Saturated straight/branched chain hydrocarbons.
  - Aromatic: Unsaturated, contain at least one benzene ring.
  - Alkene: Contain at least one carbon–carbon double bond.
  - Cycloparaffins: Saturated hydrocarbons in closed rings.
  - Halogenated: Chloride-containing hydrocarbons.

CLINICAL SIGNS OF TOXICITY

- Pulmonary toxicity:
  - Most common organ system affected.
  - Due to aspiration with direct toxic effects and disruption of surfactant.
  - Associated with cough, rales, bronchospasm, tachypnea, pulmonary edema.
  - Forty to 88% will have pneumonitis on chest film.
- CNS toxicity:
  - Seizures and/or coma
- GI toxicity:
  - Ulcers
  - Hematemesis
- Cardiac toxicity:
  - Myocardial sensitization and dysrhythmias.
  - More common with halogenated hydrocarbons.
- Dermatologic toxicity:
  - Dermatitis.
  - Full-thickness burns reported.

MANAGEMENT

- Control ABCs.
- Intubation, mechanical ventilation with positive end-expiratory pressure or jet ventilation for respiratory distress.
- Avoid catecholamines if possible (myocardial sensitization).
- Gastric emptying is controversial:
  - May increase risk of aspiration.
  - Consider if ingestion is > 30 mL.
  - Consider if hydrocarbon is associated with systemic toxicity.

Caustics

Acidic or alkaline substances capable of causing damage on contact with body surfaces.

A 3-year-old boy presents with cough and tachypnea after being found in the kitchen. He smells like pine cleaner. *Think: Aspiration of hydrocarbon with pulmonary toxicity.*

A 15-year-old boy presents after being found by his mother in the garage passed out with a rag soaked in paint stripper. *Think: Inhalation of halogenated hydrocarbon with cardiac toxicity.*

Associated with systemic toxicity: **CHAMPS**
Camphor
Halogenated hydrocarbons
Aromatic hydrocarbons
Hydrocarbons associated with **M**etals
Hydrocarbons associated with **P**esticides
Suicidal ingestions

Most common caustic exposure: Household bleach (sodium hypochlorite)

Common acids:
- Sulfuric acid
- Formic acid
- Nitric acid
- Phosphoric acid
- Acetic acid
- Chromic acid
- Hydrofluoric acid

A**C**ids cause **C**oagulation; a**L**kalies cause **L**iquefaction.

Dermal exposure to hydrofluoric acid, found in rust removers, can result in systemic absorption, hypocalcemia, hypomagnesemia, and death. Treat with supportive care, calcium gluconate paste to dermal burn, and IV calcium.

Acid exposure is associated with a higher mortality than alkali due to systemic absorption of acid.

Acids

EXPOSURE

- Drain cleaners
- Disinfectants
- Rust removers
- Photography solutions

MECHANISM OF TOXICITY

- Acids cause coagulation necrosis.
- Dehydration of superficial tissues produces an eschar that limits tissue damage.
- Systemic absorption of strong acids causes acidosis, hemolysis, and renal failure.
- Acid exposure is associated with a higher mortality than alkali despite less local tissue destruction.

CLINICAL SIGNS OF TOXICITY

- Hematemesis, melena
- Abdominal pain
- Gastric perforation with peritonitis
- Gastric outlet obstruction
- Dermal burns

MANAGEMENT

- Control ABCs.
- Obtain blood gas to detect systemic absorption of acid.
- Obtain upright chest film to look for free air.
- Endoscopy of gastric mucosa.
- Surgical intervention if indicated.
- Supportive care.

Alkalies

EXPOSURE

- Industrial cleaners
- Industrial bleach (sodium hypochlorite)
- Batteries
- Clinitest tablets

MECHANISM OF TOXICITY

- Cause liquefaction necrosis.
- Lipids saponified, proteins denatured, causing deep local tissue injury.
- Alkali exposure is associated with a lower mortality than acid despite more local tissue destruction.

CLINICAL SIGNS OF TOXICITY

- Orofacial burns
- Drooling, odynophagia
- Stridor, dyspnea
- Esophageal perforation, chest pain, mediastinitis
- Dermal burns

- Control ABCs.
- Upright chest film to look for free air, button batteries.
- For patients with alkali ingestions: If patient has orofacial burns, drooling, vomiting, stridor, or inability to drink sips of water, admit for endoscopy within 12 to 24 hours.
- Eye exposure: For both acid and alkali exposures to the cornea, irrigate with normal saline (2 to 10 L) until the pH is 7.5. Alkaline eye exposures may result in continued local tissue destruction and always require ophthalmology consultation.
- Endoscopic removal of ingested batteries is required for batteries lodged within the esophagus. Once they are below the lower esophageal sphincter, they will likely pass without incident.
- Surgical intervention if indicated.
- Supportive care.

Common alkalies:
- Sodium hydroxide
- Lithium hydroxide
- Ammonium hydroxide
- Sodium hypochlorite

► PESTICIDES

Organophosphates

EXPOSURE

- Pesticides
- Animal care
- Household products
- Chemical warfare

MECHANISM OF TOXICITY

- Irreversibly binds to cholinesterase, inactivating it by phosphorylation.
- Acetylcholinesterase in RBC/CNS, pseudocholinesterase in serum.
- Phosphorylation ("aging") takes place between 24 and 48 hours postexposure for most pesticides. Nerve agents age more quickly. Once aging is complete, enzyme must be resynthesized (takes weeks).
- Accumulation of ACh in synapse causes cholinergic crisis.

CLINICAL SIGNS OF TOXICITY

- Muscarinic effects: "SLUDGE" symptoms
- Nicotinic effects:
  - Diaphoresis
  - Hypertension
  - Tachycardia
- Neuromuscular effects:
  - Fasciculations
  - Muscle weakness
- CNS effects:
  - Anxiety
  - Tremor
  - Confusion
  - Seizures
  - Coma
- Long-term effects:
  - Delayed neurotoxicity
  - Delayed polyneuropathy
  - Transient paralysis 24 to 96 hours after exposure

Why endoscope?
- Safe 12 to 24 hours after exposure
- May identify surgical candidates
- Grades injuries and predicts risk of strictures
- Patients who develop strictures are far more likely to develop neoplasm at stricture site than those without exposure.

Organophosphate chemical warfare agents:
GA: Tabun
GB: Sarin
GD: Soman
VX

HIGH-YIELD FACTS

Emergency Toxocology

MANAGEMENT

- Supportive care (including airway).
- Atropine reverses CNS and muscarinic effects (may require multiple doses).
- Pralidoxime (2-PAM) regenerates acetylcholinesterase (must be given before 24 to 36 hours, before "aging" is complete).

Carbamates

Structurally related to organophosphates, carbamates also work by inhibiting cholinesterase.

EXPOSURE

- Insecticides
- Wartime pretreatment (carbamate pyridostigmine given in Gulf War)
- Myasthenic agents

MECHANISM OF TOXICITY

Inhibit cholinesterase via carbamoylation, a transient and reversible process.

CLINICAL SIGNS OF TOXICITY

- Similar to organophosphates: "SLUDGE," nicotinic and neuromuscular effects.
- CNS effects not prominent.
- All effects transient (24 hours).

MANAGEMENT

- Supportive care as with organophosphates.
- Atropine for reversal of muscarinic symptoms.
- 2-PAM usually not necessary (carbamate inhibition is transient).

▶ **ENVIRONMENTAL/OCCUPATIONAL TOXINS**

Methemoglobin

EXPOSURE

- *Environmental:* Nitrites (well water, food, chemicals/dyes).
- *Medications:* Local anesthetics, dapsone, Pyridium, nitroglycerine.
- *Hereditary:* Deficiency of reducing enzymes or abnormal Hgb.

MECHANISM OF TOXICITY

Oxidation of Hgb
- Normal Hgb has Fe^{2+} group (able to bind oxygen). Oxidant stress causes $Fe^{2+} \rightarrow Fe^{3+}$ (methemoglobin).

Inability to Transport Oxygen
- Ferric ion (Fe^{3+}) in methemoglobin is unable to bind oxygen.
- Methemoglobin shifts oxygen dissociation curve to left (impairs release).
- With severe oxidant stress, methemoglobin begins to accumulate.

Organophosphates inactivate acetylcholinesterase, causing cholinergic crisis.

Causes of diaphoretic skin:
SOAP
Sympathomimetics
Organophosphates
Aspirin (salicylates)
PCP

CLINICAL SIGNS OF TOXICITY

General Signs
- Cyanosis with normal pO_2 that doesn't respond to supplemental oxygen.
- "Chocolate-brown" color of blood (compare with normal blood color).
- Confirmation of methemoglobin level by co-oximetry.

Mild
- Methemoglobin level < 20%.
- Cyanosis present.

Moderate
- Methemoglobin level 20 to 50%.
- Cyanosis, dyspnea, headache, fatigue.

Severe
- Methemoglobin level 50 to 70%.
- Seizures/coma, myocardial ischemia, acidosis.

Death
- Methemoglobin level > 70%.

MANAGEMENT

- Supportive care.
- Antidote therapy: Methylene blue:
  - Indicated for patients with moderate to severe symptoms or level > 20%.
  - An electron carrier that allows methemoglobin → Hgb.
  - Utilizes NADPH pathway to reduce itself back to methylene blue.
  - Can't use in patients with glucose-6-phosphate dehydrogenase deficiency (can't generate NADPH).
  - Pulse oximetry will drop transiently due to bluish discoloration of blood.
- Exchange transfusions and hyperbaric oxygen for refractory cases.

Measurement of O_2 in methemoglobinemia:
- Pulse oximetry trends toward 85% (measures color).
- pO_2 from ABG measures dissolved oxygen and may be normal. Calculated O_2 saturation will also be normal.
- Accurate measurement by co-oximetry

Carbon Monoxide (CO)

CO is responsible for the most deaths due to poisoning in the United States.

EXPOSURE

- *Combustion:* Fires, vehicle exhaust, home generators.
- *Metabolism:* Methylene chloride (paint remover) is metabolized to CO in the liver.

MECHANISM OF TOXICITY

- Binds to Hgb with ~250 times greater affinity than oxygen.
- Shifts oxygen–Hgb dissociation curve to left (decreases release of O_2).
- Binds to myoglobin in heart and skeletal muscle.
- Binds to and inactivates cytochrome oxidase.
- Associated with CNS ischemic reperfusion injury.

Fetal Hgb binds to CO with even greater affinity than adult Hgb.

A couple and their son present to the emergency department with flulike symptoms and mild confusion 1 day after using a home generator in the garage. *Think: CO poisoning.*

- Multiple victims
- Initial flulike symptoms
- Exposure to products of combustion

CLINICAL SIGNS OF TOXICITY

Mild
- Headache, nausea, vomiting.

Moderate
- Chest pain, confusion, dyspnea.
- Tachycardia, tachypnea, ataxia.

Severe
- Palpitations, disorientation.
- Seizures, coma, hypotension, myocardial ischemia, dysrhythmias, pulmonary edema, ARDS, rhabdomyolysis, renal failure, multiorgan failure, disseminated intravascular coagulation.

MANAGEMENT

Elimination
- CO dissociates from Hgb at different rates depending on F_{IO_2}:
  - Room air (21% O_2) ~4 hours
  - 100% O_2 (1 ATM) ~90 minutes
  - 100% O_2 (3 ATM) ~23 minutes
- Hyperbaric O_2 (HBO):
  - Enhances pulmonary elimination of CO as above.
  - Displaces CO from myoglobin and cytochromes in peripheral tissues.
  - Decreases reperfusion injury.
  - May decrease delayed neurologic sequelae in some patients.
  - Indications for HBO:
    - Evidence of end-organ ischemia (syncope, coma, seizure, focal neurologic deficits, myocardial infarction, ventricular dysrhythmias).
    - CO–Hgb level > 25% (> 15% in pregnancy, children).
    - Severe metabolic acidosis.
    - Unable to oxygenate (pulmonary edema).
    - No improvement with 100% O_2.

Treat coexistent CO poisoning in fires (further compromises O_2 supply).

Cyanide

Among the most potent and potentially lethal toxins.

EXPOSURE

Inhalation
- Smoke from fires involving chemically treated wool, silk, rubber, polyurethane.

Ingestion or Cutaneous Exposure
- Accidental or intentional ingestion of chemical baths used in photography, jewelry making, and electroplating.
- Food and drug tampering (poisoning).
- Ingestion of plants or fruits containing cyanogenic compounds.

Iatrogenic
- Nitroprusside contains cyanide.

MECHANISM OF TOXICITY

- Inhibits cytochrome oxidase at cytochrome aa_3 of the electron transport chain.
- Causes cellular hypoxia and lactic acidosis.
- Blocks production of adenosine triphosphate.

CLINICAL SIGNS OF TOXICITY

- CNS dysfunction: Headache, seizures, coma.
- Cardiovascular dysfunction: Bradycardia, decreased inotropy, hypotension.
- Pulmonary edema.
- Hemorrhagic gastritis.

MANAGEMENT

- Supportive care (manage airway, fluids)
- Decontamination
- Antidote therapy:
    - Administration of nitrites generates methemoglobin.
    - Methemoglobin draws cyanide groups from cytochrome oxidase.
    - Thiosulfate transfers sulfur group to cyanomethemoglobin.
    - Thiocyanate (relatively harmless) is excreted in urine.
- Cyanide antidote (Lilly antidote kit):
    - Amyl nitrite pearls—crush and inhale.
    - Sodium nitrite—give IV over 20 minutes.
    - Sodium thiosulfate—IV (after nitrites).
    - Monitor excessive methemoglobin production during antidotal therapy.

"Classic" signs of cyanide toxicity such as bitter almond odor and cherry-red skin color are unreliable.

HIGH-YIELD FACTS

Emergency Toxicology

Environmental Emergencies

AIR

DEFINITION

Impaired gas exchange at altitude.

ALTITUDE CLASSIFICATION

- High altitude: 5,000 to 11,500 feet.
- Very high altitude: 11,500 to 18,000 feet.
- Extreme altitude: > 18,000 feet.

SIGNS AND SYMPTOMS

High Altitude
- Decreased exercise performance.
- Increased ventilation at rest.

Very High Altitude
- Maximal SaO < 90%, PaO_2 < 60 mm Hg.
- Stress and sleep hypoxemia.

Extreme Altitude
- Severe hypoxemia and hypocapnia.
- Acclimatization impossible.

▶ HIGH-ALTITUDE ACCLIMATIZATION

DEFINITION

The body's adjustment to lower ambient oxygen concentrations.

PHYSIOLOGY

- Carotid body hypoxemia stimulates increase in ventilation, which leads to decreased $PaCO_2$ and increased PaO_2.
- Without adequate O_2, hyperventilation leads to acute respiratory alkalosis.
- Renal response is to excrete more bicarbonate, returning the pH to normal.
- Increased erythropoietin within 2 hours of ascent gives rise to an increased red cell mass in days to weeks, hence a minimal and subclinical decreased O_2-carrying capacity.

▶ ACUTE MOUNTAIN SICKNESS (AMS)

DEFINITION

Syndrome of several constitutional complaints related to hypobaric hypoxemia and its physiologic consequences.

Late effects: *Chronic mountain polycythemia:*
- Headache
- Sleep difficulty
- Mental slowness
- Impaired circulation

Associated high-altitude symptoms:
- Snow blindness (ultraviolet keratitis)
- Pharyngitis
- Retinopathy

- At 24 hours: Hangover (lassitude, anorexia, headache, nausea, vomiting).
- Then oliguria, peripheral edema, retinal hemorrhages.
- Finally, high-altitude pulmonary edema (HAPE), high-altitude cerebral edema (HACE), death.

TABLE 19-1. **Medications Used for High-Altitude Illness**

DRUG	MECHANISM OF ACTION	INDICATIONS
Acetazolamide	- Decreases the formation of bicarbonate by inhibiting the enzyme carbonic anhydrase - Diuretic action counters the fluid retention of AMS - Decreases bicarbonate absorption in the kidney, resulting in a metabolic acidosis that stimulates hyperventilation (to blow off excess CO_2) - This compensatory hyperventilation is normally turned off as soon as the pH reaches close to 7.4 - By maintaining a constant forced bicarbonate diuresis, acetazolamide causes the central chemoreceptors to continually reset, permitting the hyperventilation to continue, thereby countering the altitude-induced hypoxemia	- Abrupt ascent to over 10,000 feet - Nocturnal dyspnea - AMS - History of altitude illness (used as prophylaxis)
Dexamethasone	- Decreases vasogenic edema - Decreases intracranial pressure - Antiemetic - Mood elevator	- HACE
Oxygen	- Low-flow oxygen improves sleeping problems by ameliorating the normal hypoxemia that occurs during sleep	- AMS - HAPE - HACE
HBO	- Improves hypoxemia for all altitude illness - In nitrogen narcosis, raises ambient pressure and PaO_2 in order to convert nitrogen bubbles back to solution and restore O_2 to deprived areas while the body eliminates the problem gas	- AMS - HAPE - HACE - Nitrogen narcosis
Nifedipine	- Decreases pulmonary artery pressure	- HAPE when descent or oxygen are unavailable
Morphine, furosemide	- Reduce pulmonary blood flow and decrease hydrostatic force, resulting in less fluid available for extravasation	- HAPE

RISK FACTORS

- Childhood
- Rapid ascent
- Higher sleeping altitudes
- Chronic obstructive pulmonary disease
- Decreased vital capacity
- Cold
- Heavy exertion
- Sickle cell disease

TREATMENT (SEE TABLE 19-1)

- Oxygen.
- Descent.
- Acetazolamide.
- HBO chamber.
- Nonsteroidal anti-inflammatory drugs for headache.
- Prochlorperazine for nausea and vomiting.

Other helpful tips:
- Avoidance of alcohol and overexertion.
- High-carbohydrate diet.

Patients with sickle cell disease require supplemental oxygen for high-altitude exposures.

Definitive treatment for all high-altitude syndromes is descent. Descent may be simulated with a Gamow bag, which is a portable hyperbaric oxygen (HBO) chamber.

▶ **HIGH-ALTITUDE PULMONARY EDEMA (HAPE)**

DEFINITION

Noncardiogenic pulmonary edema seen at altitude associated with decreased vasoconstriction, increased pulmonary hypertension, and capillary leak.

SIGNS AND SYMPTOMS

- Dry to productive cough
- Tachypnea
- Tachycardia
- Peripheral cyanosis
- Fatigue
- Orthopnea
- Rales

Best prevention for AMS is graded ascent with enough time at each altitude step for acclimatization.

TREATMENT (SEE TABLE 19-1)

- Oxygen to keep $SaO > 90\%$.
- Immediate descent to lower altitude.
- HBO chamber.
- Continuous positive airway pressure.
- Consider nifedipine if descent or HBO is not available.
- Minimize exertion.
- Keep warm (cold stress elevates pulmonary artery pressures).
- Consider morphine/furosemide.

Early diagnosis of HAPE is key, because it is reversible in the early stages. Patients need not develop any signs of AMS before developing HAPE. Early presentation may be just a dry cough.

► **HIGH-ALTITUDE CEREBRAL EDEMA (HACE)**

DEFINITION

Progressive neurologic deterioration in someone with AMS.

SIGNS AND SYMPTOMS

- Altered mental status
- Ataxia
- Cranial nerve palsy
- Seizure
- Stroke-like symptoms
- Coma (usually not permanent)
- Headache
- Nausea, vomiting

TREATMENT (SEE TABLE 19-1)

- Oxygen to keep SaO > 90%.
- Immediate descent to lower altitude.
- Dexamethasone.
- Loop diuretic.
- HBO chamber.

► **HIGH-PRESSURE DYSBARISM**

Descent Barotrauma

DEFINITION

Barotrauma associated with descent or dive in body spaces that cannot equalize pressure; also known as the "squeeze."

SIGNS AND SYMPTOMS

- Middle ear squeeze (barotitis media):
  - Eustachian tube dysfunction
  - Ear fullness or pain
  - Nausea and vertigo
  - Hemotympanum
- External ear squeeze:
  - Due to occlusion of external ear canal with cerumen
  - Bloody otorrhea
  - Petechiae in canal
- Inner ear squeeze:
  - Rare
  - Associated with rapid descent
  - Tinnitus, vertigo, hearing loss
  - Nausea, vomiting
- Sinus squeeze:
  - Sinus pain or pressure
  - Usually frontal and maxillary
  - Can have epistaxis
  - Associated with preexisting sinus inflammation or blockage

Boyle's Law:
$P \propto K/V$
As pressure increases, volume decreases.
P = pressure
K = temperature in degrees Kelvin
V = volume

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- Lung squeeze:
  - Occurs in divers who hold their breath going down
  - Hemoptysis
  - Shortness of breath
  - Pulmonary edema
- Equipment squeeze:
  - Conjunctival/scleral/periorbital petechiae under face mask
  - Petechiae on skin under suit

TREATMENT

- All types of squeeze:
  - Cease dive.
  - Equilibrate spaces in advance (remove foreign body, use decongestants).
- Give antibiotics for:
  - Frontal or sphenoid sinus squeeze
  - Otitis externa
  - Tympanic membrane rupture
- Inner ear fistula requires surgical repair.

> Most types of descent barotrauma resolve with ascent and rest.

Ascent Barotrauma

DEFINITION

Barotrauma caused by expansion of gas on ascent in body spaces that cannot equilibrate.

SIGNS AND SYMPTOMS

- Reverse ear squeeze:
  - Tympanic membrane rupture
  - Ear pain
  - Bloody otorrhea
  - Occurs with rapid ascent
- Pulmonary barotrauma:
  - Dissection of air into pulmonary tissue with failure to exhale during ascent
  - Associated with:
    - Pneumomediastinum
    - Pneumopericardium
    - Local subcutaneous emphysema
    - Pulmonary interstitial emphysema
    - Pneumothorax

> Risk factor for reverse ear squeeze is upper respiratory tract congestion/infection.

TREATMENT

- All types of ascent barotrauma: Rest.
- Reverse ear squeeze: Ear, nose, and throat consult.
- Pulmonary barotrauma:
  - Oxygen.
  - Observation.
  - Most resolve without intervention.

Dysbaric Air Embolism (DAE)

DEFINITION

Arterial air embolism associated with ruptured alveoli; enters left heart through pulmonary veins and may occlude an area of systemic circulation.

SIGNS AND SYMPTOMS

- Coronary artery emboli:
  - Chest pain
  - Dysrhythmias
- Central nervous system (CNS) emboli:
  - Focal neurologic deficit
  - Aphasia
  - Seizure
  - Dizziness
  - Headache
  - Confusion
  - Visual field loss

TREATMENT

- HBO.
- Avoid air transport (ascent).

Decompression Sickness

DEFINITION

Illness due to nitrogen bubbles in the blood, which form on decompression (ascent).

SIGNS AND SYMPTOMS

Type 1: Skin, Lymphatic, Musculoskeletal "Bends"
- *Skin:* Pruritus, redness, mottling.
- *Lymphatic:* Lymphedema.
- *Musculoskeletal:* Periarticular joint pain.

Type 2: Cardiovascular, Respiratory, CNS "Bends"
- *Cardiovascular:* Tachycardia, acute coronary syndrome.
- *Respiratory:* Dyspnea, cough, pulmonary edema, pneumothorax, hemoptysis.
- *CNS:* Focal neurologic deficit, back pain, urinary retention, incontinence.

TREATMENT

- Transport immediately to HBO chamber.
- Supine position.
- Intravenous (IV) fluids.
- 100% O_2.
- Avoid air evacuation.
- Steroids controversial.

Nitrogen Narcosis

DEFINITION

- The partial pressure of nitrogen in inspired tank air is increased at depth and as it accumulates in the tissues; the inert gas exerts an anesthetic effect on the diver.
- Becomes a problem at 70- to 100-foot dives.

SIGNS AND SYMPTOMS

- Euphoria, disinhibition, overconfidence, poor judgment.
- Slow reflexes.
- Fine sensory discrimination loss.
- At greater depths: Hallucinations, coma, death.

TREATMENT

Ascend at a reasonable rate with assistance.

WATER

▶ **NEAR DROWNING/IMMERSION SYNDROME**

DEFINITION

- *Drowning:* Death from an immersion.
- *Near drowning:* Survival after an immersion.

PATHOPHYSIOLOGY

- Mechanism of injury is suffocation from aspiration and associated laryngospasm.
- Fresh water (lakes, rivers, pools, baths): Hypotonic liquid disrupts surfactant and causes intrapulmonary shunting and fluid retention.
- Sea water (oceans and some lakes): Hypertonic liquid draws intravascular fluid into alveoli and causes intrapulmonary shunting.

SIGNS AND SYMPTOMS

- Vary significantly:
  - Mild cough and shortness of breath.
  - Full cardiac arrest due to pneumonia/pneumonitis.
- Once stable, hospital course can also vary, depending on:
  - Aspiration (usually contaminated water).
  - Physiologic reserve of victim.

TREATMENT

- Rapid and cautious rescue.
- C-spine immobilization.
- Control airway, breathing, and circulation (ABCs).
- Rewarm as needed (see section on hypothermia).
- Treat associated injuries.

High-risk groups for drowning:
- Children < age 4
- Teens (poor judgment)
- Elderly (tubs)
- Alcohol and drug users

"Secondary drowning" is death after initial stabilization.

Risk factors for near drowning:
- Hypoglycemia
- Head trauma
- Seizure

- Obtain chest x-ray, arterial blood gas, finger-stick glucose, electrolytes, toxicology screen, C-spine x-rays.
- No role for empiric steroids or antibiotics.

PROGNOSIS

- Cerebral anoxic injury begins within a few minutes of no oxygen.
- Some authorities believe resuscitation should not be initiated if immersion > 10 minutes.
- Scattered case reports of survival without neurologic deficit in up to 24% of children requiring cardiopulmonary resuscitation.

> ▶ **MARINE LIFE TRAUMA AND ENVENOMATION**

The emergency physician must be familiar with the fauna of ocean and lake environments in order to diagnose and treat injuries and illnesses inflicted by them.

Type of Injury

- Marine life can be grossly divided into those that have stingers to cause injury and those that have nematocysts.
- A nematocyst is a microscopic "spring-loaded" venom gland, discharged by physical contact or osmotic gradient.
- The gland found on tentacles contracts when touched, striking victim repetitively, leaving whiplike scars.
- Gland remains active after the animal dies or tentacle rips off.

Stingers

- Stingrays
- Starfish
- Scorpion fish
- Sea urchins
- Catfish
- Lionfish
- Cone shells

TREATMENT OF STINGER INJURY

- Immerse wound in nonscalding hot water (45°C) for 90 minutes (or until pain is gone) to break down venom.
- X-ray to find and remove stings.
- Aggressive cleaning, antibiotics.

Nematocysts

- Portuguese man-of-war
- Corals
- Fire corals
- Anemones
- Sea wasps
- Jellyfish

They're not dead until they're warm and dead (see hypothermia).

- ABCs.
- Inactivate nematocysts by immersing them in vinegar (5% acetic acid).
- Do not use tap water (causes venom discharge by osmotic gradient).
- Immobilize limb.
- IV access and fluids.
- Antivenin: 1 ampule diluted 1:10 IV (20,000 U/ampule).
- Antihistamines/epinephrine/steroids for anaphylaxis.
- Shave off remaining nematocysts.
- Pain control.
- Tetanus prophylaxis.

The main difference in treatment between a stinger and nematocyst injury is that you use water for a stinger and vinegar for a nematocyst.

Shark Attacks

- Sharks attack humans only when they can't see well enough to tell them apart from seals and sea lions, unless you invade their territory and start bleeding and flailing around haphazardly.
- Great white, mako, hammerhead, blue, bull, reef, and tiger sharks make up the majority of species reported to attack.
- If attacked, a force of ~18 tons per square inch and razor-sharp teeth digging into a limb or torso can quickly be a fatal blow if the victim doesn't immediately seek medical attention.

Sharks:
< 100 attacks per year worldwide with < 10% mortality.

Blue-Ringed Octopus Envenomation

- Found off Australian coast.
- Bites when handled and antagonized.
- Beak injects venom containing tetrodotoxin (TTX), a paralyzing neurotoxin that blocks voltage-gated Na^+ channels.
- Signs and symptoms: Paresthesias, diffuse flaccid paralysis, respiratory failure, local erythema.

TTX also found in puffer fish flesh.

Gila Monster and Mexican Beaded Lizard

- Normally timid, bite those who handle them.
- Venom: Phospholipase-A, hyaluronidase, arginine esterase, and a kallikrein-like hypotensive enzyme secreted by glands in lower jaw.
- Animal sometimes continues to bite/chew; the longer it holds on, the more venom gets in.
- Signs and symptoms:
  - Crush and puncture wounds, may have teeth in wound.
  - Burning pain, radiates up extremity, lasts 8 hours, edema and cyanosis.
  - Rare systemic effects: Weakness, fainting, hypotension, sweating.
- Treatment:
  - Remove animal (if still attached).
  - Remove teeth, clean copiously and aggressively, broad-spectrum antibiotics, tetanus.
  - Observation for systemic effects.

HIGH-YIELD FACTS

Environmental Emergencies

Amphibians

- Colorado River toad, Columbian poison-dart frogs, and several species of newt and salamander secrete toxins in their skin and internal organs:
  - Batrachotoxin: Opens Na^+ channels irreversibly.
  - Tetrodotoxin: Blocks Na^+ channels irreversibly.
  - Bufotalin: Cardiac toxin, acts like digitalis.
  - Samandarine: Opens CNS Na^+ channels irreversibly.
- Treatment is supportive.

> ► **SNAKE ENVENOMATION**

Crotalidae Family (Pit Vipers)

Includes rattlesnakes, massasauga, copperheads, and water moccasins (see Table 19-2).

SIGNS AND SYMPTOMS

Local (see Table 19-3)
- Burning pain (severity related to amount of venom).
- Edema spreading proximally.
- Local petechiae, bullae, and skin necrosis.

Systemic
- Nausea.
- Fever.
- Metallic taste.
- Weakness.
- Sweating.
- Perioral paresthesias.
- Hypotension.
- Fasciculations.
- Compartment syndrome (rare).
- Pulmonary edema.
- Anaphylaxis.
- Shock, intravascular coagulation, hemorrhage, and death.

TABLE 19-2. **Characteristics of Poisonous versus Nonpoisonous Snakes**

POISONOUS	NONPOISONOUS
Triangle-shaped head	Rounded head
Elliptical pupil	Round pupil
Pit between eye and nostril	Absence of pit
Fangs	Absence of fangs

TABLE 19-3. Snake Bite Grading System

GRADE	PIT VIPER	CORAL SNAKE
0	■ Fang marks ■ No pain ■ No systemic symptoms ■ Don't give antivenin	■ No envenomation ■ Minimal fang scratches or punctures ■ Minimal local swelling ■ No systemic symptoms in 24 hours ■ Give 3 vials antivenin
I	■ Fang marks ■ Mild pain/edema ■ No systemic symptoms ■ Give 0–5 vials antivenin (50 mL)	■ Fang scratches or punctures ■ Minimal local swelling ■ Systemic symptoms present, but no respiratory paralysis ■ Give 3 vials antivenin
II	■ Fang marks ■ Severe pain ■ Moderate edema ■ Mild systemic symptoms ■ Give 10 vials antivenin (100 mL)	■ Severe envenomation ■ Respiratory paralysis occurs within 36 hours ■ Give 5–10 vials antivenin
III	■ Fang marks ■ Severe pain/edema ■ Severe symptoms (hypotension, dyspnea) ■ Evidence of systemic coagulopathy ■ Give 15–20 vials (150–200 mL)	

Elapidae Family (Coral Snakes)

- Includes the corals, cobras, kraits, and mambas.
- Venom is a neurotoxin.

SIGNS AND SYMPTOMS

- Painless bite site
- Weak/numb within 90 minutes
- Euphoria
- Drowsiness
- Tremors
- Salivation
- Slurred speech
- Diplopia
- Flaccid paralysis
- Respiratory failure

TREATMENT FOR ALL SNAKE BITES

- ABCs.
- Reassure patient.
- Immobilize extremity.

Color pattern recognition (for U.S. snakes only):
- Red-on-yellow . . . kill a fellow
- Red-on-black . . . venom lack

- Horse antivenin for bite grades I, II, and III (pit vipers).
- Antivenin for all coral snake bites (regardless of symptoms).
- Local wound care.
- Tetanus prophylaxis.
- Prophylactic antibiotics are not recommended.

▶ **SPIDERS**

Brown Recluse Spider

Loxosceles reclusa.

DEFINITION

Identified by violin design on its back (Figure 19-1).

EXPOSURE

- Found in midwestern, mid-Atlantic, and southern states.
- Inhabits warm, dry places—typically woodpiles, cellars, and abandoned buildings.
- Venom: Proteases, alkaline phosphatase, lipase, complement-system substances.

SIGNS AND SYMPTOMS

- Necrosis at bite site due to local hemolysis and thrombosis associated with ischemia:
  - Mild red lesion, may be bluish/ischemic.
  - Varying degrees of pain, blistering, necrosis within 3 to 4 days.
  - May take weeks to heal.
- Systemic symptoms:
  - Fever
  - Chills
  - Nausea
  - Myalgias, arthralgias
  - Hemolysis, petechiae
  - Seizure

> Loxoscelism is a reaction to *Loxosceles* spider venom proportional to amount of venom exposure.
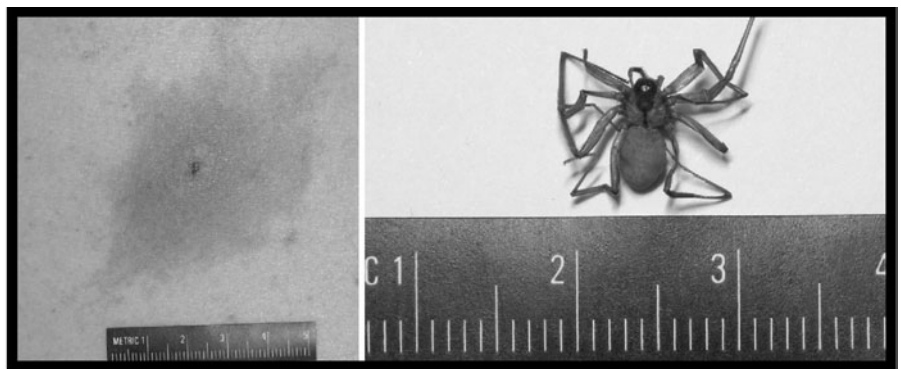
FIGURE 19-1. Brown recluse spider.

(Photo courtesy of Dr. Martin A. Wolfe.)

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- Renal failure
- Death

TREATMENT

- Monitor ABCs.
- Daily wound care.
- Analgesia.
- Tetanus prophylaxis.
- Antibiotics if wound becomes infected.

Black Widow Spider

Latrodectus mactans.

DEFINITION

- Identified by red-orange hourglass on abdomen.
- Female two times size of male, has design, is only one that can envenomate humans.

EXPOSURE

- Found throughout the United States (except Alaska).
- Inhabits warm, dry, protected places, typically woodpiles, cellars, barns, under rocks, etc.
- Venom: Neurotoxic protein causing acetylcholine and norepinephrine release at synapses.

SIGNS AND SYMPTOMS

- Local pinprick sensation, red, swollen.
- Then slow progression of painful muscle spasm of large groups.
- Lasts a few hours, resolves spontaneously.
- Systemic signs:
  - Hypertension
  - Coma
  - Shock
  - Respiratory failure
  - Death (more often in children)

TREATMENT

- Pain control and muscle relaxants (narcotics and benzodiazepines).
- Tetanus, local wound care.
- Antivenin available for severe reactions (one to two vials IV over 30 minutes).

Abdominal muscle spasm of black widow spider bite may mimic peritonitis.

Patients who should get antivenin:
- Extremes of age
- Pregnant
- Underlying medical conditions (check for hypersensitivity/allergy with skin test prior to administration)

► SCORPION

Bark scorpion (*Centruroides exilicauda*).

DEFINITION

Has venom gland and stinger in last segment of tail.

EXPOSURE

- Found in Arizona, California, Nevada, and Texas.
- Bark scorpion inhabits areas around trees.
- Other species usually under rocks, logs, floors, boots.
- Victims usually children and campers/hikers.
- Venom activates Na^+ channels, damaging parasympathetic, somatic, and sympathetic nerves.
- Other proteins may cause hemolysis, hemorrhage, and local tissue destruction.

SIGNS AND SYMPTOMS

- Severe and immediate pain (erythema and swelling are species dependent).
- Then tachycardia, increased secretions, fasciculations, nausea, vomiting, blurred vision, dysphagia, roving eye movements, opisthotonos, respiratory failure, syncope, death (rare).

TREATMENT

- ABCs.
- Sedation with benzodiazepines.
- No opiates, may potentiate venom.
- Tetanus prophylaxis, local wound care.
- Antivenin: Unlicensed, available in Arizona only, derived from goat serum—skin test first, use for extremes of age, severe reactions (1 to 2 vials), observe for 24 hours (especially children).

▶ BEES AND WASPS (APIDS AND VESPIDS)

DEFINITION

- Apids: Honeybees, bumblebees.
- Vespids: Wasps, hornets, yellow jackets.
- "Africanized" honeybees: Much more aggressive but venom contains same substances.
- Yellow jackets cause most allergic reactions.

EXPOSURE

- Venom: Mostly proteins and peptides (phospholipase-A, hyaluronidase, histamine, serotonin, bradykinin, dopamine), also lipids and carbohydrates.
- Systemically, toxicity from venom or anaphylaxis can occur within minutes.

SIGNS AND SYMPTOMS

- Most commonly local: Burning pain, erythema, edema at sting site, lasting ~24 hours.
- Local/systemic delayed reaction up to 1.5 to 2 weeks later.
- Toxicity: Vomiting, diarrhea, fever, drowsiness, syncope, seizure, muscle spasm, and rarely neuritis, nephritis, vasculitis.
- Anaphylaxis possible.

Apids sting only once—stinger detaches in skin, bee then dies.
Vespids can sting again and again—stinger has no retroserrate barbs.

TREATMENT

- ABCs:
  - Airway can be compromised early (ask about prior bee stings).
- For any systemic signs:
  - Epinephrine 1:1,000 0.3 to 0.5 mL SQ in adults, 0.01 mL/kg in children.
  - Antihistamine (e.g., 50 mg diphenhydramine IV).
  - Steroid (e.g., 125 mg methylprednisolone IV).
  - Beta-2 agonist for wheezing (e.g., albuterol nebulizer treatment).
  - Admit and observe.

▶ FIRE ANTS

EXPOSURE

- *Soleneopsis invicta*—"unvanquished ant," Brazilian import to the United States in 1930s.
- The ant bites with its mandibles, then stings with its venom apparatus in its hindquarters.
- Venom:
  - Contains 99% insoluble alkaloid, causing hemolysis, membrane depolarization, local tissue destruction, and activation of complement pathway.
  - Approximately 10 to 16% of population have fire ant hypersensitivity and are susceptible to anaphylaxis.
  - No cross-reactivity with that of bees.

SIGNS AND SYMPTOMS

- Immediate intense burning pain locally.
- Becomes sterile pustule in 6 hours.
- With multiple stings in sensitized individuals, nausea, sweating, dizziness, and anaphylaxis can occur.

TREATMENT

- ABCs.
- Local cleaning.
- Analgesia, ice.
- For systemic reactions: Treat as for bee sting.

▶ TERRESTRIAL ANIMAL TRAUMA

Dogs

- 80 to 90% of reported animal bites.
- Usually lacerations, crush injury, punctures, and avulsions.
- Wounds are infection and tetanus prone, bacteria from animal oral flora (not human skin):
  - Aerobes: *Streptococcus, Staphylococcus aureus, Pasteurella multocida* (20 to 30%), *Staphylococcus intermedius, Eikenella corrodens*.
  - Anaerobes: *Bacteroides* spp., *Actinomyces* spp., *Fusobacterium* spp., *Peptostreptococcus* spp.

Dog bites:
Wound location frequency:
Upper extremity >>
lower extremity >>
head and neck (kids) >>
trunk

SIGNS AND SYMPTOMS

- Ask about ownership of dog and behavior.
- If stray and cannot be observed, initiate rabies immunization.

TREATMENT

- ABCs as appropriate.
- Local wound care: Aggressive irrigation, debridement; loose suturing or leave open for delayed primary closure.
- Tetanus prophylaxis.
- Prophylactic antibiotics for the immunocompromised and frail (amoxicillin/clavulanic acid for outpatient, ampicillin/sulbactam inpatient) (see Table 19-4).

Cat Bites

- 5 to 18% of reported animal bites in United States.
- More likely to contain *P. multocida* in wound.

SIGNS AND SYMPTOMS

- Punctures (57 to 86%)
- Abrasions (9 to 25%)
- Lacerations (5 to 17%)

TREATMENT

See dog bites.

Humans

- Behavior at times animal-like.
- Clenched fist injuries (CFIs) from punches in the face have high incidence of poor wound healing and complications.
- Bites to areas other than the hand have similar rates of infection as nonbite lacerations.

Cat and dog bite infection rules of thumb:
- Infection in < 24 hours: *P. multocida.* Rx: penicillin, if penicillin allergic, tetracycline, or erythromycin
- Infection in > 24 hours: *Staph* or *Strep* Rx: Dicloxacillin or cephalexin

Cat bites:
Wound location: Upper extremity >> head and neck > lower extremity > trunk

TABLE 19-4. **Closure and Prophylaxis for Bites**

ANIMAL BITE/STING	CLOSE?	MAIN OFFENDING ORGANISM	ANTIBIOTICS?
Dog	Yes, except if crush injury or bite to hand	Capnocytophaga canimorsus	Yes
Cat	No	Pasteurella multocida	Yes
Rodent	No	Multiple	No
Monkey	No	Herpesvirus	Acyclovir for high-risk bites
Human	Yes, except if closed fist injury	Eikenella corrodens	Yes

- Human oral flora is polymicrobial:
  - Aerobes: *Streptococcus viridans*, *S. aureus*, *Haemophilus* spp., *E. corrodens*.
  - Anaerobes: *Bacteroides* spp., *Fusobacterium* spp., *Peptostreptococcus* spp.

TREATMENT

- CFIs: Copious irrigation, debridement, tetanus, penicillin, and second-generation cephalosporin for *Staphylococcus* coverage. Diabetics should get an aminoglycoside.
- Immobilize, daily dressing changes, elevate extremity.

FIRE

▶ THERMAL BURNS

DESCRIPTION OF DEPTH

First Degree
- Superficial burn, epidermis only, mild–moderate erythema, heals without scar.

Second Degree
- Superficial partial thickness, epidermis and part of dermis (follicles and glands spared), blisters and erythema, very painful, heals with or without scar in 2 to 3 weeks.
- Deep partial thickness, epidermis and deeper dermal layers (follicles and glands), blisters, erythema, some charring, painful, heals in 3 to 4 weeks with some scarring.

Third Degree
- Full thickness, epidermis, dermis, subcutaneous fat; pale, charred, painless, leathery; surgical skin grafts necessary for healing; moderate–severe scarring.

Fourth Degree
- Skin, fat, muscle, bone involvement; severe, life-threatening injury.

DESCRIPTION OF SIZE

- Estimate size with rule of nines (Figure 19-2).
- Size of patient's palm roughly 1% of their total body surface area (TBSA).

CATEGORIZATION

Minor Burns
- < 15% TBSA for ages 10 to 50.
- < 10% TBSA for ages < 10 or > 50.
- < 2% TBSA full thickness, any age, no other injury.

Moderate Burns
- 15 to 25% TBSA second degree for ages 10 to 50.
- 10 to 20% TBSA second degree for ages < 10 or > 50.

E. corrodens: A gram-negative rod of the normal human oral flora, distinguished by being susceptible to penicillin but resistant to penicillinase-resistant penicillins, clindamycin, metronidazole, cephalosporins.

Typical cause of burns:
First degree: Sunburn
Second degree: Hot liquids
Third degree: Hot liquids, steam, hot oil, flame
Fourth degree: Flame, hot oil, steam

Poor-risk burn patients:
Those with diabetes, heart and lung disease, age < 10 or > 50.
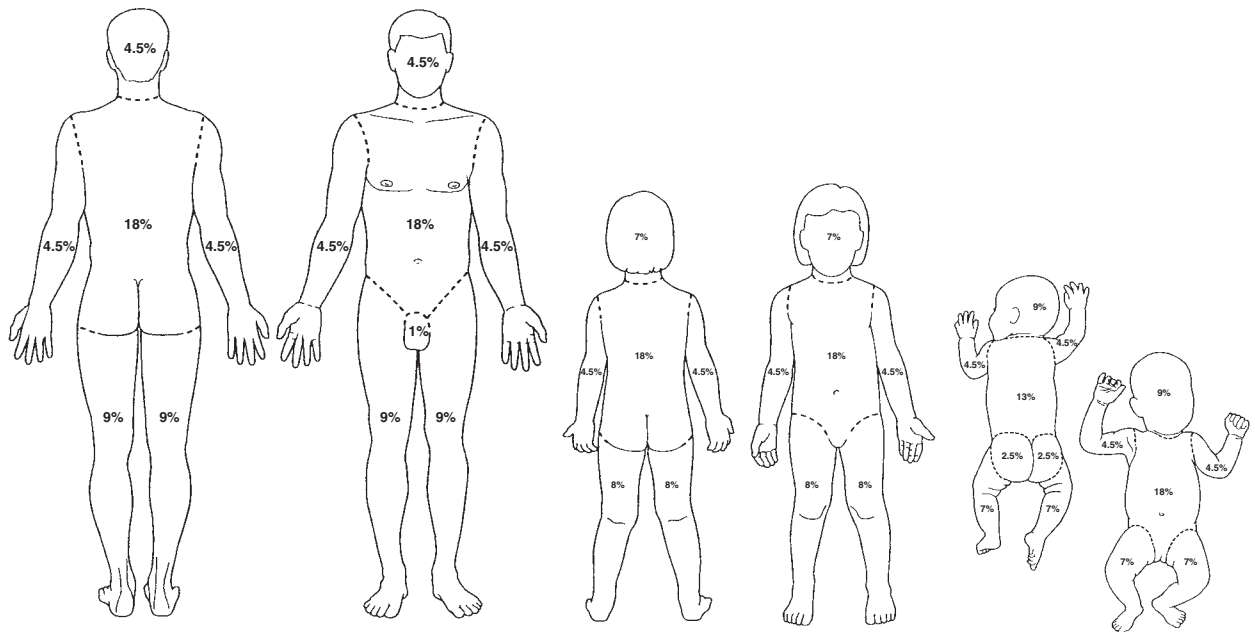
FIGURE 19-2. **Rule of nines for adults, children, and infants.**

(Reproduced, with permission, from Stead L. BRS: *Emergency Medicine.* Philadelphia, PA: Lippincott Williams & Wilkins, 2000: 558.)

- 2 to 10% TBSA full thickness, any age.
- No perineal, facial, foot, hand, or circumferential limb burns.

Major Burns
- > 25% TBSA second degree for ages 10 to 50.
- > 20% TBSA second degree for ages < 10 or > 50.
- > 10% TBSA full thickness, any age.
- Hand, foot, perineal, circumferential limb, major joint, electrical burns.
- Associated inhalation injury or other trauma in elderly, infants, poor-risk patients.

TREATMENT

- Prehospital: Transport to nearest burn-capable hospital, preferably within 30 minutes.
- Emergency department (ED): Ask age, medical history, tetanus status, what burned, was there an explosion/blast injury, were there toxic substances, enclosed space?
- Fiberoptically evaluate airway for edema and injury, or intubate and protect the airway prior to respiratory failure.
- Humidified 100% O_2.
- Fluid resuscitation according to Parkland formula.
- Beware of overaggressive fluid resuscitation leading to excessive pulmonary and peripheral edema.
- Foley catheter to monitor urine output (maintain 1 cc/kg/hr).
- Primary and secondary surveys: Treat all associated injuries appropriately.
- Management of the burn wound:
  - Within 30 minutes: "Put out the fire"—cool water.
  - Always cover with clean, sterile, saline-soaked dressings to small areas.

> **Parkland formula:** 4 mL/kg/% TBSA burned; one half over first 8 hours from time of burn, and remaining one half over next 16 hours. Ringer's lactate is fluid of choice.

> Children have increased TBSA relative to their weight, increased evaporative water loss, and therefore increased fluid requirements.

- Protect against hypothermia: Cover with sterile sheets.
- Escharotomy for full-thickness or circumferential burns.
- Analgesia with morphine.
- Blisters: Best left intact until consulting service can evaluate (skin is protective); incise and drain sterilely all those not on palms and soles if delayed transfer or consultation.
- No role for prophylactic antibiotics.
- Antibiotic skin cream/ointment application only if delay of transfer to burn unit or delay in arrival of consulting service for many hours; silver sulfadiazine or bacitracin.
- Tetanus prophylaxis.

CRITERIA FOR TRANSFER TO BURN UNIT

- > 10% TBSA in ages < 10 or > 50.
- > 20% TBSA for all ages.
- Burns to face, eyes, ears, hands, feet, genitalia, perineum, or major joints.
- > 5% TBSA third-degree burn.
- Electrical and chemical burns.
- Inhalational injury.
- Children.

▶ CHEMICAL BURNS

General

- Determine what chemical is by history and physical examination.
- Remove patient from agent, then remove agent from patient.
- If wet agent, dilute with water.
- If dry agent, wipe off first.
- Remove clothing.
- Assess size and depth of burn.

Hydrofluoric Acid (HF)

Penetrates tissues like alkalies and releases F$^-$, which immobilizes intracellular Ca^{2+} and Mg^{2+}, poisoning enzymes.

TREATMENT

- Dilute with water for 30 minutes.
- Detoxify: Local intramuscular/subcutaneous/transdermal, IV, or intra-arterial 5 to 10% calcium gluconate solution.

Phenol (Carbolic Acid)

Causes local coagulation necrosis, protein denaturation, and systemic life-threatening complications.

TREATMENT

- Dilute with water.
- Isopropyl alcohol decreases local absorption and necrosis.

Any chemical burn to the eye is the number 1 eye emergency and needs immediate irrigation! (Test ocular pH before and after irrigation; goal is 7.3 to 7.7, ideally 7.45.)

HF is found in glass etching, dyes, high-octane gas, and germicides.

Phenol is found in dyes, deodorants, agriculture, and disinfectants.

Lime (Calcium Oxide)

- Desiccates.
- Converted to alkali by water (calcium hydroxide).

TREATMENT

Brush off, then dilute.

Lyes [KOH, NaOH, Ca(OH)$_2$, LiOH]

- Strong alkalies burn more deeply and longer, leading to liquefaction necrosis.
- High tissue absorption.
- If swallowed, aggressively manage airway and have surgical intervention available. Dilute aggressively.

TREATMENT

Dilute with water.

Metals (Industrial, Molten)

Water can cause severe exothermic reaction.

TREATMENT

- Cover hot metal fragments with mineral oil.
- Brush off or excise fragments.

Tar is found in gasoline and hot tars.

Hydrocarbons

- Tar causes deep thermal burns.
- Dissolve tar, don't peel it off (takes skin too).

TREATMENT

- Dilute gasoline with water.
- Aggressively cool tar.
- Use Neosporin (polysorbate) to remove tar.

Do not try to remove tar with acetone. It will make the tar stick to the skin even more and continue burning.

▶ **ELECTRICAL BURNS**

PHYSICS OF ELECTRICITY

- I = current (amps), R = resistance (ohms), E = energy (joules), V = voltage (volts).
- As current flows through a resistor (tissue), energy is deposited as heat; the ↑er R, the ↑er E (burn).
- Current flows through path of least resistance.
- High voltage ≥ 1,000 V (> 600 V clinically).
- U.S. household circuits = 110 V (220 V entering home).
- Tasers = 50,000 V, 10 to 15 pulses per second.

Ohm's law: $V = I \times R$
Joule's law: $E = I^2 \times R \times$ time

- On scene: Look for source, entrance/exit wounds, extent of cutaneous injury (may reflect internal).
- Underlying internal damage far exceeds skin burns (rule of nines need not apply).
- Blood vessels, nerves, and muscle damaged most (leads to compromised vasculature, vasospasm, rhabdomyolysis, paralysis, neuropathies).
- Children biting electric cords: Risk of delayed hemorrhage from labial artery at mouth edges.
- Delayed cataracts with ocular involvement.

TREATMENT

- Scene safety.
- ABCs, support respirations.
- Advanced cardiac life support (ACLS) protocol as appropriate.
- IV access and fluids—20 cc/kg bolus.
- Treat thermal burns.
- Tetanus prophylaxis.
- Admit if poor risk, pregnant, high voltage, or systemic injury.

Lightning Injury

PHYSICS OF LIGHTNING

- 10 million to 2 billion V.
- Unidirectional impulse of current.
- Temperature 14,432 to 90,032°F.
- Hot, humid days.
- Strikes metal or tall objects.
- Electricity flows over the body (flashover) as well as through, causing pathognomonic fern-shaped mark on the skin caused by electron showering.

DEFINITIONS

- Direct strike: Lightning versus person.
- Contact strike: Lightning versus object person is touching.
- Side flash: Lightning versus object near person, then flash from object.
- Ground current: Lightning versus ground near person, then up person's foot.
- Stride potential/step voltage: Lightning versus ground near person, then up one foot and down the other; temporarily cold, numb, paretic, pulseless legs.

SIGNS AND SYMPTOMS

- Fernlike pattern to skin (cutaneous streaking) (Figure 19-3).
- Ruptured tympanic membranes.
- Often unconscious.
- Cardiac arrhythmias.

TREATMENT

- ABCs, secure airway.
- ACLS protocol as appropriate.

- Myonecrosis leads to rhabdomyolysis, which can lead to renal pigment disease.
- Alkalinize urine (IV 44 mEq $NaHCO_3$ in 1 L of IV fluid).

HIGH-YIELD FACTS

Environmental Emergencies

A 29-year-old man is found lying down in a big open field outdoors, not breathing and pulseless. His clothes are tattered, he has no shoes, but has blood in his ear canals. He is awake but confused. You notice fernlike burns on his skin. *Think: Lightning injury.*
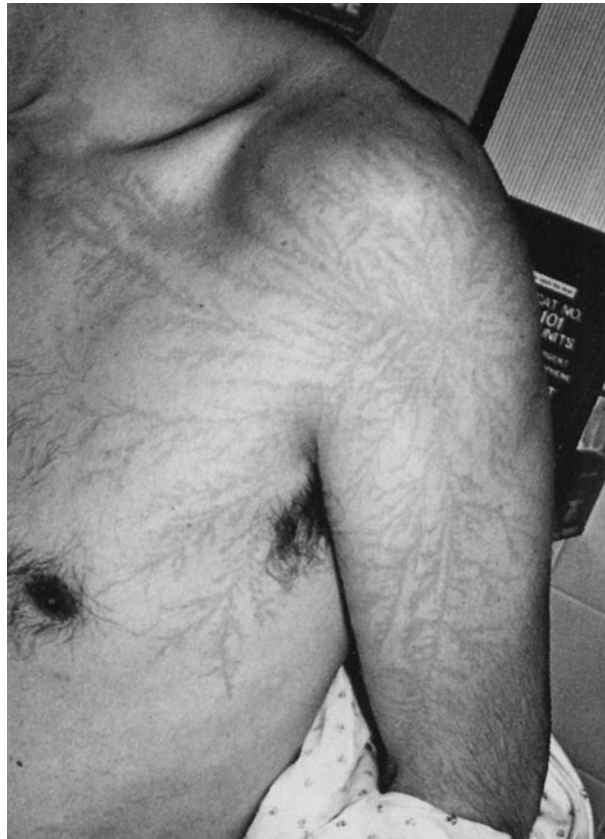
FIGURE 19-3. Characteristic cutaneous streaking pattern of lightning injury.

(Reproduced, with permission, from Tintinalli JE, Kelen GD, Stapczynski JS. *Emergency Medicine: A Comprehensive Study Guide*, 5th ed. New York: McGraw-Hill, 2000: 1300.)

- Reverse triage if multiple injured: Care for "dead" first—they recover if supported.
- Immobilize C-spine.
- Tetanus prophylaxis.
- Electrocardiogram (ECG) and cardiac monitor.
- Urinalysis, complete blood count, creatine kinase (CK) and CK-MB, lytes, blood urea nitrogen (BUN)/creatinine.
- Treat any bony fractures; admit for observation.

► **HEAT ILLNESS**

Heat Transfer

- Radiation:
  - 65% of normal body cooling.
  - Heat transfer from electromagnetic waves.
- Convection:
  - 10 to 15% of normal body cooling.
  - Heat transfer from cooler water vapor in air current.

- Conduction:
    - 2% of normal body cooling.
    - Heat transfer from direct physical contact.
- Evaporation:
    - 15 to 25% of normal body cooling.
    - Heat transfer from evaporated sweat/breath.

Heat Exhaustion

DEFINITION

Syndrome of vague constitutional symptoms associated with salt and water depletion, and heat exposure or heavy exertion.

SIGNS AND SYMPTOMS

- Dizziness or fatigue with normal mental status.
- Nausea/vomiting.
- Headache.
- Positional hypotension/syncope.
- Mildly elevated temperature.
- Diaphoresis.
- Heat-related illnesses: Prickly heat, heat cramps, or heat tetany.

TREATMENT

- Remove from heat.
- Rest.
- IV hydration with normal saline, oral hydration with sports drinks.
- Check and correct electrolytes.
- Observe for resolution of symptoms.

Associated heat-related symptoms:
- Heat syncope (postural hypotension)
- Prickly heat (maculopapular rash under clothed areas)
- Heat tetany (carpopedal spasm, hyperventilation)
- Heat cramps (muscle cramping due to electrolyte loss)

Heatstroke

DEFINITION

Rapid rise in core temperature (> 104.9°F) associated with:
- Altered mental status
- Symptoms of heat exhaustion
- Anhydrosis
- Loss of temperature regulation

SIGNS AND SYMPTOMS

- CNS abnormalities:
    - Ataxia
    - Combativeness
    - Hallucination
    - Seizure
    - Posturing
    - Hemiplegia
    - Coma
- Renal failure:
    - Decreased Ca^{2+}
    - Hypo- or hypernatremia
    - Decreased PO_4^-
    - Hypo- or hyperkalemia

- Coagulation:
  - Increased bleeding times.
  - Consumptive coagulopathy and disseminated intravascular coagulation.
- Liver failure:
  - Abnormal liver function tests.
  - Increased creatine phosphokinase (CPK) and rhabdomyolysis.
- Hypotension
- Death

TREATMENT

There is no role for acetaminophen in heatstroke.

- Rule out other causes of fever and altered mental status (sepsis, thyrotoxicosis, meningitis, etc.).
- Remove from heat sources.
- Control ABCs, O_2, monitor, rectal temperature.
- Use cooling techniques; avoid rebound hypothermia.
- Correct electrolyte abnormalities.
- Check complete blood count, urinalysis, CPK, prothrombin time, partial thromboplastin time, BUN/creatinine, and ECG.
- Benzodiazepines for shivering.

► COOLING TECHNIQUES

Evaporation

- Water mist and blowing fans
- Heat dissipated by evaporation
- Rapid
- Easiest in the ED

Immersion

- Tub of water and ice
- Heat dissipated by conduction
- Impractical
- Can't monitor patient

Ice Packing

- Ice bags to groin and axillae
- Heat dissipated by conduction
- Easy
- Slow
- Poorly tolerated

Cool Lavage

Gastric
- Via nasogastric tube
- Heat dissipated by conduction
- Invasive
- Slow
- Poorly tolerated

Peritoneal

- Via peritoneal catheter
- Heat dissipated by conduction
- Invasive
- Very rapid
- Questionable sterility

DEFINITION

Autosomal dominant pseudocholinesterase deficiency.

SIGNS AND SYMPTOMS

- Hyperthermia.
- Rhabdomyolysis.
- Muscle rigidity.
- Not related to exogenous heat sources.
- Pathophysiology is distinct from neuroleptic malignant syndrome.

TREATMENT

- Dantrolene 2 to 3 mg/kg IV q6h.
- No more succinylcholine.

Malignant hyperthermia is associated with haloperidol and succinylcholine administration.

Chilblains

DEFINITION

- Local injury from dry cold at nonfreezing temperatures.
- Most commonly affects extremities and ears.

SIGNS AND SYMPTOMS

- Local edema
- Nodules or blisters
- Erythema or cyanosis
- Rarely ulcers or bullae

TREATMENT

- Reversible with gentle rewarming
- Moisturizer
- Avoidance of the cold

Trench Foot

DEFINITION

- Nonfreezing injury from wet cold.
- Due to prolonged immersion in standing water.
- Causes direct soft-tissue injury and nerve damage.

SIGNS AND SYMPTOMS

- Numbness/tingling, permanent numbness possible.
- Pallor, mottling.
- Lack of pulses.

TREATMENT

- Rest, elevation, local skin care.
- Avoidance of the cold.

Frostbite

DEFINITION

- Freezing injury when skin temperatures fall below 32°F from body trying to maintain normal core temperature (prefreeze state).
- Ice crystals form in extracellular space (freeze state).
- Tissue loss.
- Most commonly occurs on extremities and face.

SIGNS AND SYMPTOMS

- Throbbing, shooting pain in joints.
- Numbness, tingling.
- Edema.
- Blisters (clear or hemorrhagic).
- Eschars (develop over a few days).

TREATMENT

- Active rewarming in warm water (104 to 108°F).
- Tetanus, analgesia.
- Aspirate clear blisters.
- Limb elevation.
- Topical aloe vera.
- Treat for associated hypothermia (see below).

Hypothermia

DEFINITION

- Core temperature < 95°F.
- Usually due to prolonged overwhelming cold exposure; can occur in any season.

SIGNS AND SYMPTOMS

Mild Hypothermia (90 to 95°F)
- Shivering
- Excitation
- Tachypnea
- Tachycardia
- Apathy
- Poor judgment
- Dysarthria
- Ataxia

Frostnip: Mild reversible, superficial frostbite.

At risk for hypothermia:
- Extremes of age
- Alcohol users
- Homeless
- Altered mental status (including psychiatric disorders)
- Trauma victims
- Underlying chronic illness
- Hypoglycemia
- Sepsis

Moderate Hypothermia (82 to 90°F)
- Shivering ceases
- Stupor
- Bradycardia
- Dysrhythmias (often atrial fibrillation)
- Dilated pupils

Severe Hypothermia (< 82°F)
- Coma
- Hypotension
- Decreased cardiac output
- Areflexia
- Decreased respiratory rate
- Dysrhythmias (often ventricular fibrillation, agonal, or asystole)
- May appear dead

TREATMENT

All Patients
- Remove wet clothing.
- Get rectal temperature.
- Get ECG (Figure 19-4).
- Look for and treat concomitant illness (alcohol, hypoglycemia, trauma).

Mild Hypothermia
- Passive rewarming with blankets.

Moderate and Severe Hypothermia
- Active rewarming.
- Handle with care: Sudden manipulation can precipitate cardiac dysrhythmias.
- Cardiac monitoring.

Cardiac Dysrhythmias
- Treat cardiac dysrhythmias as per ACLS protocol.
- Remember, the best treatment for cardiac dysrhythmias in hypothermia is rewarming.

Tympanic membrane temperature measurements are not reliable below 94°F. *Get a rectal temperature!*

Methods of active rewarming (in order of invasiveness):
- Warmed blankets
- Mechanical warming blanket
- Warmed IV fluids
- Warmed bladder irrigation
- Warmed gastric lavage
- Warmed peritoneal irrigation
- Warmed cardiopulmonary bypass
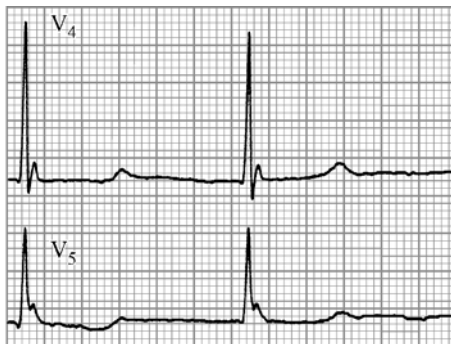
F I G U R E   1 9 - 4 .   Osborn (J) wave of hypothermia.

HIGH-YIELD FACTS

Environmental Emergencies

- Severely hypothermic patients who appear dead can have normal or near-normal neurologic outcomes: **Continue resuscitation until warm!**

Special note for trauma patients: Patients who arrive with hypothermia and obvious head trauma may have improved neurologic outcome if hypothermia is maintained. Consult with neurosurgeons.

Ethics, Medicolegal Issues, and Evidence-Based Medicine

Beneficence	To act in the best interest of one's patient.
Nonmaleficence	To do one's patient no harm: This includes protecting one's patient from personnel not qualified to deliver appropriate care due to lack of training, experience, or impairment.
Privacy and confidentiality	Physicians have a duty to protect the confidentiality of patient information. Disclosure of sensitive information is appropriate only when such disclosure is necessary to carry out a stronger conflicting duty, such as a duty to protect an identifiable third party from serious harm or to comply with a just law.
Autonomy	The ability to function independently and to make decisions about one's care free from the undue influence or bias of others.
	▪ All patients are considered autonomous if they have the ability to understand the situation as evaluated with competency examination by psychiatrist (capacity) and are not a danger to self or others (suicidal, homicidal, demented, delirious).
	▪ Physicians have a duty to respect the health care preferences of their patients.
Justice	The principle of equal and fair allocation of benefit.
	▪ Provision of emergency medical treatment should not be based on gender, age, race, socioeconomic status, or cultural background.
	▪ No patient should ever be abused, demeaned, or given substandard care.

The "duty to warn" was established by the Tarasoff case. In this case, a physician caring for a patient who made homicidal statements about another person was found liable for her death because he failed to protect or warn her, or to report the situation to police.

Advance Directives

- Oral or written instructions from a patient to family members and health care professionals about health care decisions.
- May include living wills; designation of a health care proxy; specific instructions about which therapies to accept or decline, including intubation, surgery or medical treatments, and Do Not Attempt Resuscitation orders.
- A Do Not Resuscitate order applies only to advanced cardiac life support resuscitation and does not include intubation and ventilation unless specifically addressed.

Medical Decision Surrogates

Also known as a health care proxy; appointed by the patient.

Physicians have a duty to report an impaired or incompetent colleague to the chief of staff or appropriate regulatory agency. Many states have mechanisms in place whereby anonymous reporting can be done. Physicians who conscientiously fulfill this responsibility should be protected from adverse political, legal, or financial consequences. Action toward the impaired physician may include internal discipline and/or remedial training. It does not necessarily mean the physician will lose his or her license to practice medicine.

► **MEDICAL RECORD DOCUMENTATION GUIDELINES**

General Principles

- Medical record should be legible and complete.
- Rationale for ordering laboratory and other ancillary tests should be documented.
- Patient's emergency department course, including response to any treatment, should be documented.
- Documentation in record should support Current Procedural Terminology and ICD-9-CM codes submitted on insurance form and billing statement.

Key Elements of Chart

- Reason for encounter (chief complaint).
- History, physical exam (including addressing of any abnormal vital signs).
- Assessment, clinical impression, or diagnosis.
- Care plan.
- Date and legible identity of observer.

► **LEGAL CONCEPTS**

Battery	Unwanted touching
Malpractice	Four elements: - Duty - Breach of duty - Causation - Harm
Emergency Medical Treatment and Active Labor Act	Federal law that says all persons presenting to an emergency department have the right to: - A medical screening exam to determine if they have a medical emergency. - Receive necessary stabilizing treatment for any medical emergency including active labor in a pregnant woman. - Transfer to appropriate facility once the patient is stable.

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Cost Containment

The practice of conscientiously limiting medical expenses without sacrificing the medical care of the patient. The factor most directly in the control of the physician is the judicious use of laboratory and radiographic tests.

Child Abuse

All human services professionals, including physicians, are required by all 50 states to report known or *suspected* child abuse. There is no penalty for the reporting of cases that turn out not to be cases of abuse.

Communicable Diseases

The following infectious diseases were designated as notifiable at the national level in the United States, as of 1997:

- Acquired immune deficiency syndrome
- Anthrax
- Botulism
- Brucellosis
- Chancroid
- *Chlamydia trachomatis*, genital infections
- Cholera
- Coccidioidomycosis
- Cryptosporidiosis
- Diphtheria
- Encephalitides
- *Escherichia coli* O157
- Gonorrhea
- *Haemophilus influenzae*, invasive
- Hansen disease (leprosy)
- Hantavirus pulmonary syndrome
- Hepatitis
- Legionellosis
- Lyme disease
- Malaria
- Measles
- Meningococcal disease
- Mumps
- Pertussis
- Plague
- Poliomyelitis
- Psittacosis
- Rabies, animal and human
- Rocky Mountain spotted fever
- Rubella
- Salmonellosis

- Shigellosis
- *Streptococcus pneumoniae*, invasive drug-resistant disease
- Syphilis
- Tetanus
- Toxic shock syndrome
- Trichinosis
- Tuberculosis
- Typhoid fever
- Yellow fever

► PRINCIPLES OF EVIDENCE-BASED MEDICINE

Evidence-based medicine is the practice of incorporating the best available evidence from the medical literature for a diagnostic test or treatment into daily patient care. It is an active process that requires five steps:

1. Identify a clinical problem.
2. Formulate a question.
3. Search for the best evidence.
4. Appraise the evidence.
5. Apply the information to the clinical problem.

A thorough search of the medical literature requires a computer or Internet search of MEDLINE, through OVID, Grateful Med, or Pub Med. All relevant articles should be considered. The best evidence is most often provided by meta-analysis or randomized clinical trials.

Sensitivity

(People with disease who tested positive)/(All people with disease)
true positive (TP)/(TP + false negative [FN])
Low rate of false negatives gives high value.

> The more sensitive a test, the less likely the test is to fail to detect a positive result.

Specificity

(People without disease who tested negative)/(All people without disease)
true negative (TN)/(TN + false positive [FP])
Low rate of false positives gives high value.

> The more specific a test, the less likely the test is to fail to detect negative result.

Positive Predictive Value

(People with disease who tested positive)/(All people who tested positive)
TP/(TP + FP)
All positive variables

Negative Predictive Value

(People without disease who tested negative)/(All people who tested negative)
TN/(TN + FN)
All negative variables

A high positive LR increases the pretest likelihood that a patient has a disease.

Likelihood Ratio (LR)

Measures the fixed relationship between the chance of given test result in a patient with the disorder and the chance of the same test result in a patient without the disorder.

LR for a positive test result = Sensitivity/(1 − specificity)
LR for a negative test result = (1 - sensitivity)/Specificity

Confidence Interval (CI)

Measures the range of values in which the true value studied (treatment difference, proportion, or probability) resides. Even if a study shows a benefit or harm of a given intervention, if the CI crosses zero, no treatment difference may exist. A study result with a CI that excludes zero is statistically significant.

Number Needed to Treat

Measures the number of patients with a given disease that a clinician would need to treat with the tested therapy in order to see one beneficial event or prevent one adverse event.

▶ SOURCES OF MEDICAL EVIDENCE

Meta-analysis

Evaluates the data of many trials that address the same question and attempts to combine the information: These studies are best used when the clinical problem is infrequent and large randomized trials cannot be done.

Randomized Controlled Clinical Trial (RCT)

The selected population is randomized to receive either the treatment in question or a placebo, and the outcome is measured. The ideal RCT is triple-blinded, meaning that the treating physician, the patient, and the investigators do not know which treatment has been given until the analysis is complete. These studies can establish cause and effect.

Cohort Study

The selected population is identified as being exposed or not exposed and is monitored for subsequent effects. These studies are used when the exposure cannot be assigned for logistical or ethical reasons.

Case Control Study

Populations with and without a given outcome are selected, and historical (retrospective) data are collected on exposure to a given agent or treatment.

Awards and Opportunities for Students Interested in Emergency Medicine

Who Can Join EMRA?

Any medical student who is interested in emergency medicine as a career choice. EMRA's bylaws require that all members also be members of the American College of Emergency Physicians (ACEP).

Why Join EMRA?

EMRA membership provides a number of member benefits that will help you understand how emergency medicine fits into the provision of health care to the nation's citizens. Here's a short list of what you receive when you join.

- *EM Resident*—A bimonthly newsletter featuring articles, opinions, and information that affects emergency medicine residents and students. Each issue also features job placement information.
- *Top 30 Problems in Emergency Medicine*—This book is designed as a quiz and reference book you can keep in your pocket and test yourself on during down time or refer to during a case. References to other books, medical journals, and publications are provided as sources that can be accessed for more information on a specific topic. Topics include: rapid sequence intubation, useful formulas and mechanical ventilation, ATLS, infectious disease and emergency dosages.
- *Guide to Antibiotic Use in the Emergency Department*—This handy pocket reference is a great guide you can keep for easily accessible help in determining type and dosages of the most common antibiotics used in the emergency department.

- *Emergency Medicine in Focus*—This handy guide gives vital information of preparing for, applying to, and being successful in an emergency medicine residency and career.
- **Representation to Other Key Medical Organizations**—EMRA sends representatives to such medical specialty organizations as the ACEP, the American Academy of Emergency Medicine, the Society for Academic Emergency Medicine, and the American Medical Association so your views and concerns can be presented directly to members of these important organizations.
- **Medical Student Forum Admission**—Membership gains you free addmission to EMRA's annual Medical Student Forum held in conjunction with ACEP's Scientific Assembly.
- **The Residency Fair**—This is a key opportunity to meet residency directors and get information about specific residency programs. Free to you, this meeting is held during ACEP's Scientific Assembly.

What Does the EMRA Medical Student Committee (MSC) Do?

The MSC has developed goals and objectives that are in addition to EMRA's specific goals and objectives. The MSC focuses strictly on the medical students who indicate they are working toward a specialty in emergency medicine. The MSC:

- Educates medical students about the specialty of emergency medicine and its importance to the practice of medicine in this country.

- Provides information to those students interested in emergency medicine regarding career options and emergency medicine training programs.
- Develops a network of physician advisors for third- and fourth-year medical students who may not have access to role models in emergency medicine.
- Establishes a peer network for medical students aspiring to careers in emergency medicine and allows them the opportunity to assume leadership roles in organized emergency medicine.
- Provides ACEP and Society for Academic Emergency Medicine (SAEM) with a resource for dissemination of materials and information to medical students and training institutions.

What Are EMRA's Key Spheres of Influence?

EMRA is active within the house of medicine and, most particularly, in established liaison relationships with other emergency medicine–related organizations.

Listed below are some of the organizations that EMRA interacts with routinely in order to ensure that your voice as an emergency medicine resident is heard in the larger community of organized medicine and organized emergency medicine.

- ABEM–EMRA has an ongoing opportunity to work with the American Board of Emergency Medicine on a variety of issues.
- ACEP–EMRA holds four voting positions on ACEP's Council, as well as voting representation on the college's

Academic Affairs Committee and a number of other ACEP committees. EMRA also has a reporting representative at all ACEP Board meetings.

- AMA/AOA–EMRA maintains liaison relationships with students and house staff organizations such as the American Medical Association Resident Physician Section (AMA/RPS) and the American Osteopathic Association (AOA).
- SAEM–EMRA has an ongoing relationship with the SAEM and has a reporting representative at SAEM Board meetings.
- Other voting relationships —EMRA elects a voting member to the Residency Review Committee for Emergency Medicine (RRC/EM) and appoints a representative to the Emergency Medicine Foundation (EMF) Board of Directors.

ACEP/EMRA Mentorship Program

The American College of Emergency Physicians (ACEP) has joined with EMRA (Emergency Medicine Residents' Association) to create a mentorship program in which seasoned emergency physicians and emergency medicine residents with similar interests can meet and learn from each other.

If you are interested in joining the mentorship program, please contact Sonja Montgomery of ACEP at *smontgomery@acep.org,* or 800-798-1822, ext. 3202.

EMRA LOCAL ACTION GRANTS

In order to promote the involvement of emergency medicine residents in community service and other activities that support the specialty of emergency medicine, EMRA has instituted a Local Action Grant Program. Grants will be available to any EMRA member (**medical students,** residents, fellows) or any emergency medicine interest group whose principal applicant is an EMRA member. Recipients will be selected to receive an award of up to $500. Grants will be awarded at the spring membership meeting at SAEM's Annual Meeting. The deadline for nominations is March 1.

Regions Hospital Student EM CD Now Downloadable

The Regions EM student program is now available for download from the Internet. It contains an overview of the student program, student responsibilities, an example of our feedback card system, presentation and documentation guidelines for students, a study guide for readings, case studies, and procedural workshops on suturing, splinting, use of the slit lamp and clearing the cervical spine. Each student that rotates at Regions (MN) receives this CD. You can download this from *http://www.healthpartners.com/regions/em/homepage/index.html.*

EMRA Medical Student Award

The EMRA Medical Student Award recognizes a student who displays a significant interest in emergency medicine. More importantly, this student "goes out of his or her way" for patients and colleagues. This student demonstrates a service-oriented attitude even when faced with difficult situations and stresses. The recipient receives a plaque and $250. This award is presented annually at the Spring Awards Reception held during SAEM's Annual Meeting and EMRA will notify the recipient's Dean of the honor. The deadline for nominations is March 1. To apply for this award, send an informal, anecdotal essay of 500 words or less that demonstrates the student's commitment to service. The application must also include the applicant's name, address, phone number and e-mail address, the name and address of the student's medical school and name of the school's dean.

Catalog of EM medical student electives may be found at: *http://www.saem.org/rotation/contents.htm*

Emergency Medicine Medical Student Interest Group Educational Grants

SAEM recognizes the valuable role of emergency medicine interest groups (EMIG's) for medical students interested in emergency medicine as a career. The Society will offer grants of up to $500 each to established or developing EMIG's located at medical schools with or without emergency medicine residencies. Grant monies can be used for supplies, consultation and seed money to support activities such as skills laboratories (suturing, casting, airway etc), lectures, or workshops. Grant proposals should focus on educational activities or projects related to undergraduate education in emergency medicine.

Individuals or institutions interested in applying for a grant should submit an application to the SAEM office. The deadline for submission is August 15 of the grant year with a funding date of October 1. Grants will be reviewed by a subcommittee of the Undergraduate Education Committee.

Medical Student Excellence in Emergency Medicine Award

This award is offered annually to each medical school in the United States and Canada. It is awarded to the senior medical student at each school who best exemplifies the qualities of an excellent emergency physician, as manifested by excellent clinical, interpersonal, and manual skills, and a dedication to continued professional development leading to outstanding performance on emergency medicine rotations. The award, presented at graduation, conveys a one-year membership in SAEM, which includes subscriptions the SAEM monthly journal, *Academic Emergency Medicine*, the SAEM Newsletter, and an award certificate.

Announcements describing the program and applications are sent to the Dean's Office at each medical school in February. Coordinators of emergency medicine student rotations then select an appropriate student based on the student's intramural and extramural performance in emergency medicine. Each school must submit the name of their recipient no later than June 1. The list of winners is published in a summer issue of the SAEM Newsletter.

Over 110 medical schools currently participate in this award. Contact SAEM at saem@saem.org if your school would like to participate.

SAEM Medical Student Emergency Medicine Symposium

Each year during the SAEM annual meeting, an Emergency Medicine Medical Student Forum is held. This session is designed to help the medical student understand the residency and career options that exist in emergency medicine, develop an optimal senior year schedule, evaluate residency programs, and navigate the residency application process. The medical student also learns to recognize and begin management of common and potentially life-threatening problems that present to the emergency department. A Medical Student/Resident Visual Diagnosis Photography Contest is also held at the SAEM annual meeting. Small prizes are awarded and winners are acknowledged in the SAEM Newsletter.

MEDICAL STUDENT MEMBERSHIP IN SAEM

Medical students interested in emergency medicine are invited to become members of SAEM. Membership benefits include a reduced registration fee to attend the SAEM annual meeting; a subscription to *Academic Emergency Medicine*, the monthly SAEM journal; and a subscription to the SAEM Newsletter (6 issues per year). Annual dues are $75 (includes journal subscription) or $50 (not including journal subscription).

MEDICAL STUDENT EM RESEARCH GRANTS

SAEM and the Emergency Medicine Foundation sponsor annual grants of up to a maximum of $2,400 over 3 months for medical students. Applications can be obtained from EMF. The deadline is in late January every year.

AAEM is one of the smaller EM organizations whose mission is to support fair and equitable practice environments for EM physicians. They offer medical students membership, an e-mail contact list, and a "medical stu-dent forum" feature in their monthly newsletter.

PUBLISH IN JEM!
The Journal of Emergency Medicine is looking for medical student authors to submit contributions on topics of interest to medical stu-dents such as residency applica-tion, away clerkships, resources for students, book/Web site re-views, or interviews. If interested, please contact the Student Rep-resentative on the AAEM/RES Board of Directors.

WEB SITE RESOURCES

virtualer.com

Site offers links to many Web pages related to emergency medicine. Connect to tutorials, image libraries, professional or-ganizations, job searches, free medline searches, and more!

trauma.orhs.org

A Web site dedicated to trauma and critical from Orlando Re-gional health care system. It fea-tures a trauma and emergency surgery e-mail discussion group and weekly trauma case studies.

embbs.com

Features photographic case re-ports with clinical pearls from *Academic Emergency Medicine,* the official journal of the Soci-ety of Academic Emergency medicine.

mdchoice.com

Site features interesting radiol-ogy (CT and plain film) cases.

erl.pathology.iupui.edu

Site featuring dermatopathology cases from the University of In-diana. Excellent photos of all the derm conditions you need to know in EM.

CDC.org

Presents a wealth of informa-tion including clinical guide-lines, up-to-date info on most major diseases, and an excellent search engine with links to other governmental agencies.

NCEMI.ORG

Web site of the National Center for Emergency Medicine Infor-matics. Presented as a daily newspaper page with question of the day, and ECG, x-ray photo and cartoon of the week. Summarizes important abstracts in EM. Has several excellent to other EM sites. Has many clever medical calculators and facts and formulae.

emedicine.com

This Web site has online text-books in emergency medicine and most other primary spe-cialties for use free of charge. These textbooks have four lev-els of peer review and are con-tinually updated. Opportuni-ties for authorship in one of the many textbooks are avail-able.

medschool.com

Founded by the original cre-ator of the First Aid series, Dr. Vikas Bhushan, this site has multiple weekly features such as pearl, question, and image of the week; medical humor; book and Web site reviews, online chat forums, a premed resource center, a USMLE study center, and a way to ask faculty questions directly. A must visit site!

RESIDENCY APPLICATION GUIDE ONLINE

This guide by the University California at San Francisco may be found at *http://www.som.ucsf.edu/ education/student/orgs/emig/appguide /index.htm.*

Catalog of emergency medicine residencies may be found at *http://www.saem.org/rescat/ contents.htm.*

EM Residency Formats

U.S. allopathic programs are structured in three different formats:

1–4 programs: There are 14 of these 4-year programs.

2–4 programs: There are 22 of these 3-year programs (3 years after a separate 1-year internship).

1–3 programs: There are 86 of these 3-year programs (3 years including the internship year).

There are also Canadian and osteopathic programs.

Combined programs with internal medicine, pediatrics, and family medicine also exist, and these are generally 5 years in length.

Articles of Interest That Can Be Found at saem.org

- Pro vs Con: Four vs Three—Pro: Four Years are Optimal versus Con: Three Years are Enough
- Medical Students and Research
- Taming the Residency Application Process
- Bibliographic Citation Guidelines for EM Residency Applicants
- ERAS Made Easy . . . or how your life has been made better by the AAMC!
- EM Resident Career Satisfaction
- Advice to Students Seeking an Academic Career in Emergency Medicine
- Advice to Students Beginning a Medical Student Rotation in Emergency Medicine
- The 2000 NRMP Match in Emergency Medicine
- Emergency Medicine as a Career Choice
- Top Ten Questions Regarding the Organization of Your Senior Year
- Emergency Medicine Clubs
- 10 Things To Do Before Applying To An Emergency Medicine Residency.
- Selection Criteria for Emergency Medicine Residency Applications
- Does Interview Date affect Match List Position in the Emergency Medicine National Residency Matching Program Match?

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