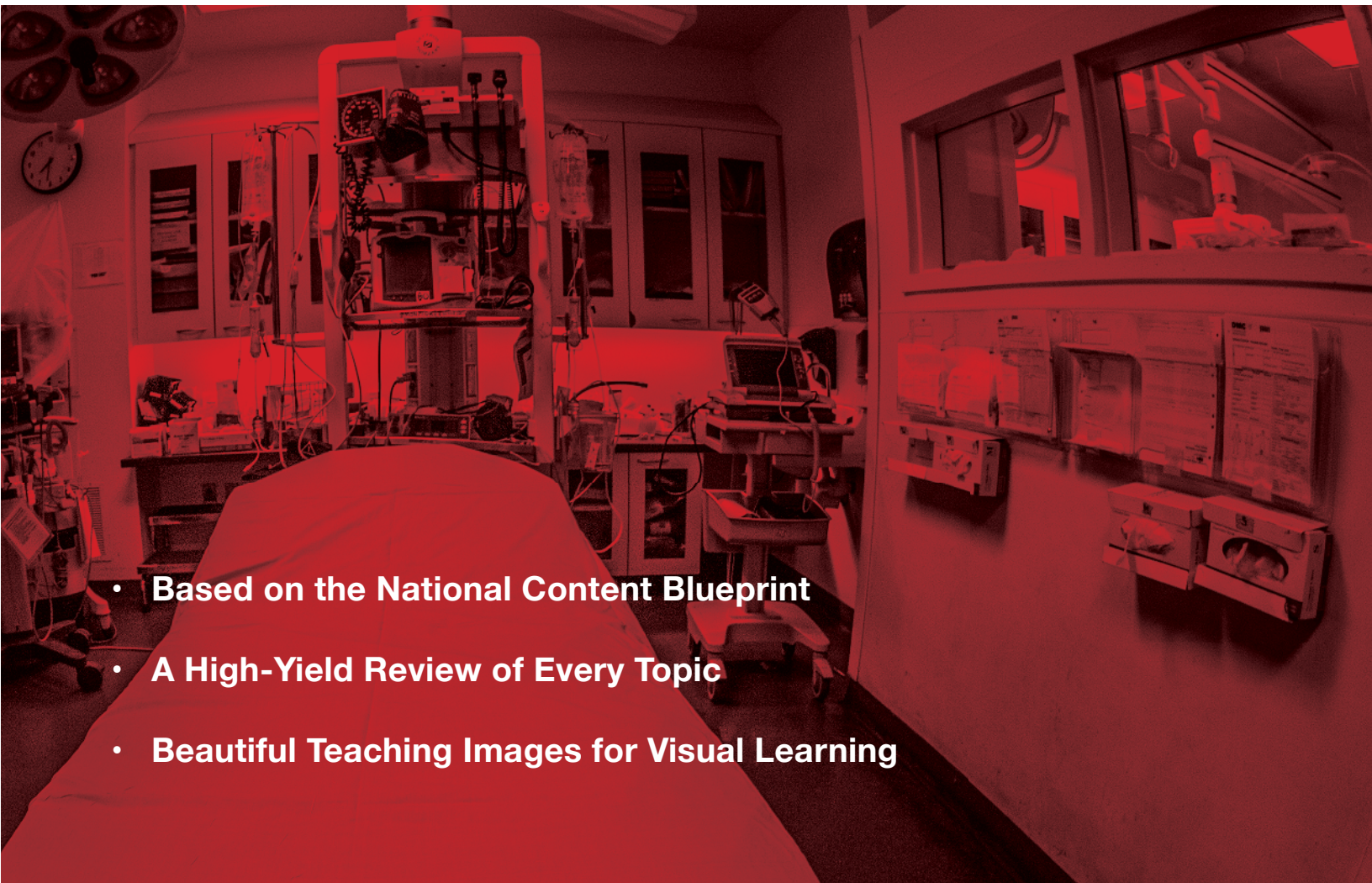
Rosh
RAPID REVIEW

# Emergency Medicine Rotation Exam

1ST EDITION



- Based on the National Content Blueprint
- A High-Yield Review of Every Topic
- Beautiful Teaching Images for Visual Learning

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# RAPID REVIEW

## Emergency Medicine Rotation Exam

**1ST EDITION**

Yehuda Wolf, PA-C
Emily Oslie, PA-C
Adam Rosh, MD

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# Your Content Creation Team

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Yehuda joined the Rosh team in 2015 while still a student in PA school. As Rapid Review author and editor-in-chief, Yehuda continues to ensure the Rapid Reviews are of the highest caliber and standards. Yehuda graduated from Touro College New York with a Masters in Physician Assistant Studies with Honors. Yehuda currently spends his time between a busy pediatric primary care practice and pediatric surgical urology.

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Dr. Adam Rosh is the founder of Rosh Review, which he created in 2011. He received his B.A. in biochemistry and M.S. in microbiology from the University of Wisconsin, Madison. He received his medical degree from Rutgers Medical School and completed his emergency medicine residency training at New York University/Bellevue Hospital Center, where he was chief resident. Dr. Rosh went on to serve as assistant residency director and residency director at Detroit Receiving Hospital. He is the author and editor of Pretest Emergency Medicine and Case Files Emergency Medicine both published by McGraw Hill. He currently is the CEO of Rosh Review and an Attending Emergency Physician.

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Kristian is Senior Content Editor at Rosh Review. Originally hailing from Salzburg, Austria, his training includes three years in med school, a B.S. in communication sciences, and an M.A. in German literature–all of which helped in creating the passionate stickler for format, grammar, and bio-science terminology he is today. Kristian's work is focused on making sure Rosh Review content is well written, correct, and adhering to our house style and good science writing standards.

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Erica Parrish received her B.A. in biology from the College of St. Scholastica and her D.C. from Northwestern Health Sciences University. She gained clinical experience serving patients in both Minnesota and Florida before joining the team at Rosh Review.

## All Of You, Content Impactors

Although most of the content in this book was created by our author team, over the years we've received so much input from our subscribers, we want to shine a light on this contribution. Every comment or feedback email we receive is reviewed, discussed, and if agreed upon, implemented. The power of all of you, contributing your experience and insights, allows us to continually improve the quality of the content we publish. This process is perpetual. As medicine changes, our content must change with it. We value the content partnership we have and hope you'll continue to raise expectations.

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# Shout Outs

## Yehuda Wolf, PA-C

A beloved teacher imparted words I constantly think of. “Life is like an individual positioned on a downward moving escalator. The individual is either climbing upward or being moved downward. No one stays in one place.” While this is ever so true in medicine, which is constantly evolving at a rapid pace, it is even more so with Adam Rosh. Ever since joining Rosh it has never been just one project, just one product. It has always been an attitude of improvement, progression, and advancement. It is with this drive and passion that we are constantly encouraged to improve ourselves at Rosh Review for our consumers, who in essence are our peers, in providing care and compassion in the greatest of all professions. For the compilation of the Rapid Review Rotation Exam Series, I have gratitude to a number of people. Thank you to my most amazing wife, Devorah and our children, Naomi, Ahuva, and Ilana. Their unparalleled support and encouragement allowed me to work on this incredible project after I finally got home from work. Thank you to Emily, Kristian, Erica, and Ora. While I may have spearheaded much of the work, I am nothing but a dwarf on the shoulder of a team of giants.  I am honored to work with an amazing group of dedicated individuals. Finally, thank you to the One Above for His Goodness and Grace, for giving me the energy and strength to continue to climb and grow to further heights.

## Emily Oslie, PA-C

This is for my parents, Myron and Sherri, who have cheered me on through each phase of my education and career; my sister, Maddie; my classmates at Duke who supported me and are the reason I have countless fond memories of my time in the program; the instructors, advisors, and clinical preceptors who challenged me to become a compassionate and competent provider; my co-providers, mentor, supervising physician, and patients at Duke Urgent Care who make me a better PA every day.

## Adam Rosh, MD

A hearty thanks goes out to my family for their love and support, Danielle, Ruby, Rhys, and especially my parents, Karl and Marcia; the incredibly dedicated team at Rosh Review who relentlessly raise expectations; the committed medical professionals of Rutgers Medical School, the emergency medicine departments at New York University/Bellevue Hospital Center, and Wayne State University/Detroit Receiving Hospital; and my patients, who put their trust in me, and teach me something new each day.

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# Purpose & Goals of this book

**Learning and education is a dynamic process, one that is never ending. Once we commit to a life in nursing, we commit to a life of learning. The Rosh Rapid Review series is best suited to serve as an adjunct to your nursing education. It is not meant as a primary source, rather it should help you organize your thoughts and provide ancillary knowledge for a more robust education. I am counting on you to not just regurgitate facts, but rather to "learn how it all works". We are privileged to be in the role of caretaker and thus have a responsibility to our patients to be the most knowledgeable we can be. Use this book on your learning journey. At some point, you will grow out of it. But in the mean time, I hope the hard work by the dedicated Rosh Review team can play just a small role in helping you reach your goals and achieve your dreams.**

Adam Rosh, MD
Founder, Rosh Review

**"To study the phenomena of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all."**

William Osler, Aequanimatas

**"I didn't want to just know the names of things. I remember really wanting to know how it all worked."**

Elizabeth Blackburn,
Nobel Prize for Physiology or Medicine

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# Rosh Core Values

1. We pay attention to detail and always deliver the highest quality content.

2. We believe it is a privilege to interact with and care for individuals.

3. We are always learning and continuously self improving, it is part of our DNA.

Even after multiple reviews, there are sure to be mistakes in this book. As part of Rosh Review's culture of continuously learning, please let us know if you identify an error by sending an email to alwaysimproving@roshreview.com

And if there is a topic not covered in this edition that you'd like included in the next edition, please send an email to alwaysimproving@roshreview.com

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Let’s get started...



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CHAPTER 1

# Cardiovascular System

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CHAPTER 1

# Cardiovascular System


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# I. Endocarditis

**A. Endocarditis**

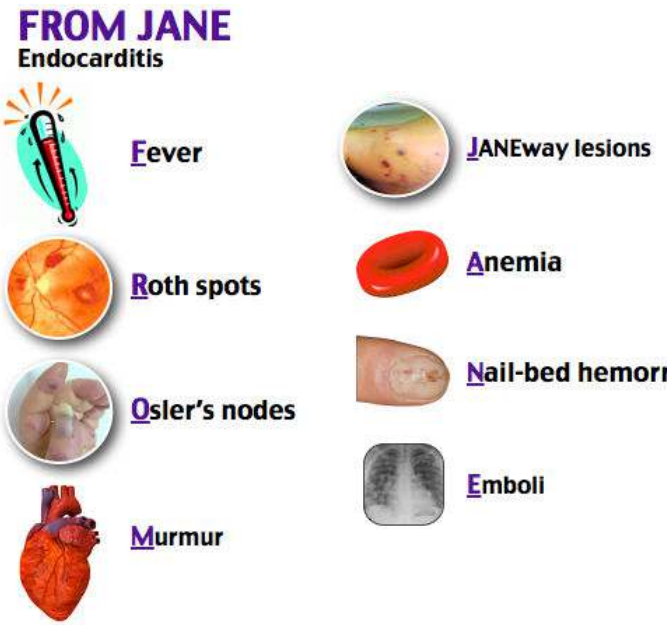
Pathophysiology
- Most commonly caused by:
  - IVDA: ***S. aureus*** (tricuspid affected)
  - Native valve: **streptococci**, ***S. aureus*** (mitral affected)

Presentation
- Fever, cough, rash, myalgias

Physical Exam
- **F**ever, **R**oth spots, **O**sler nodes, **M**urmur, **J**aneway lesions, **A**nemia, **N**ail bed hemorrhages, **E**mboli (**FROM JANE**)

Diagnostic Studies
- **Echocardiography** and **Duke criteria**

Management
- Antibiotics (usually IV following **blood cultures**)




Notes


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### B. Infective Endocarditis

| | Infective Endocarditis | |
|---|---|---|
| | **Right-sided** | **Left-sided** |
| **Vascular involvement** | Pulmonary vascular involvement | Systemic vascular involvement |
| **Population** | Intravenous drug abusers | Acquired valvular disease, congenital heart disease |
| **Onset** | Acute | Acute or subacute |
| **Most commonly affected valve** | Tricuspid valve | Mitral valve |
| **Most common organism** | *Staph. aureus* | *Strep. viridans, Staph. aureus* |

Notes


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### C. Modified Duke Criteria for the Diagnosis of Endocarditis

| Modified Duke Criteria for the Diagnosis of Endocarditis | |
|---|---|
| **Major criteria** | Two separate positive blood cultures with microorganisms typical for infective endocarditis (viridans streptococci, *Streptococcus bovis*, HACEK group, *Staphylococcus aureus*, enterococci) |
| | Echocardiographic evidence of endocardial involvement (typical valvular lesions: vegetations, abscess, or partial dehiscence of a prosthetic valve) |
| **Minor criteria** | Predisposition: predisposing heart condition or IV drug use |
| | Temperature > 38°C |
| | Vascular phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhage, Janeway lesions |
| | Immunological phenomena: glomerulonephritis, Osler nodes, Roth spots, rheumatoid factor |
| | Microbiological evidence: positive blood culture but not meeting major criterion, or serologic evidence of active infection with organism consistent with infective endocarditis |

**For the diagnosis of endocarditis to be made, the following must be present:**

- 2 major criteria
- 1 major criterion and 3 minor criteria
- 5 minor criteria

Notes


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# II. Arrhythmias

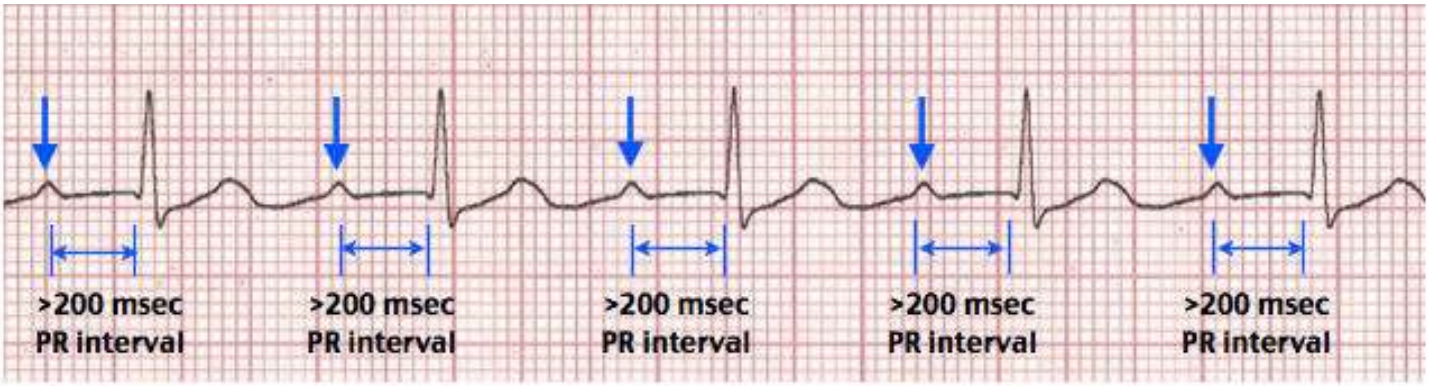
### A. First-Degree Heart Block

Pathophysiology
• Associated with aging, digitalis, ischemia, inflammation, cardiomyopathies, beta-blockers

Physical Exam
• Rhythm will be regular

Diagnostic Studies
• ECG with a PR interval > 0.20 sec (200 msec) and constant

• Normal AV conduction is slightly prolonged
• P waves and QRS complexes are normal
• 1:1 relationship between P and QRS
• Block is most often at the level of the AV node




Notes


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**B. Second-Degree Heart Block (Mobitz I / Wenckebach)**

Physical Exam
• Rhythm will be **irregular**

Diagnostic Studies
• ECG with a PR interval that is **progressively** lengthening until QRS complex fails to conduct after a P wave (**"dropped beat"**)

**• P waves and QRS complexes are normal but...**
- There are **dropped beats** (P waves without QRS complexes)
- P-R interval progressively lengthens
- R-R interval progressively shortens until a beat is dropped
- Leads to **"grouped beating"**
- The block is almost always within the AV node





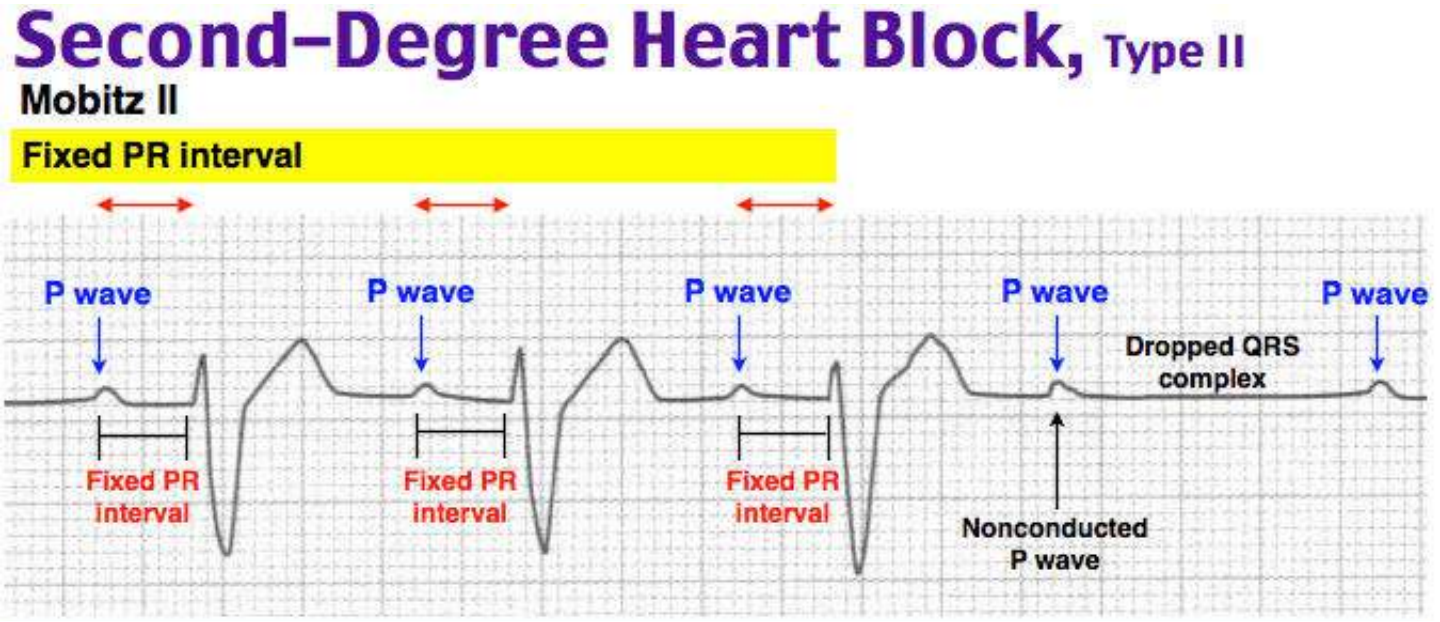
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**C. Second-Degree Heart Block (Mobitz II)**

Diagnostic Studies
• ECG with a PR interval that is **fixed** and **consistent** with **"dropped beats"**

Management
• **Consider** permanent pacemaker

• **P waves are normal**
• **QRS complexes** are usually (not always) **wide** due to concomitant bundle branch block
• **PR intervals** are always the **same duration**
• There are **dropped beats**
• The block is **below the AV node**, generally in the **His-Purkinje system**




Notes


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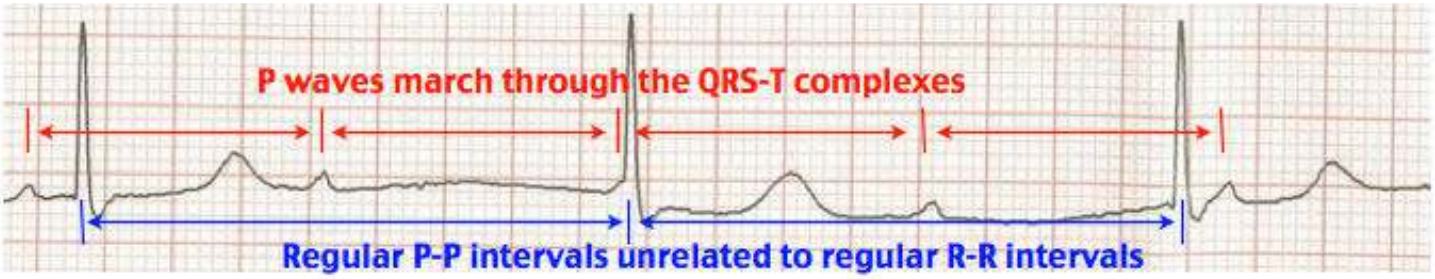
**D. Third-Degree Heart Block**

Pathophysiology
• Associated with aging, anterior or inferior infarction, digitalis

Diagnostic Studies
• ECG with **regular** P-P and R-R intervals that are **unrelated** to one another
• Findings represent the independent beating of the atria and ventricles

Management
• If symptomatic, treat with atropine, dopamine, or temporary pacing
• Definitive treatment is the placement of a **permanent pacemaker**

• No atrial impulses are conducted
• The **atria and ventricles beat independently** of one another
• P waves are normal
• The block can occur at the level of the AV node, the bundle of His or the bundle branches
• **QRS complexes** may be **narrow (above the His bundle)** or **wide (at or below His bundle)** depending on the location of the block
• No relationship between P waves and QRS complexes
• Independent and **regular atrial rate** (constant P-P interval)
• Slower, independent and **constant ventricular rate** (constant R-R interval)
• P waves are not related to the QRS complex
• The **PR interval is variable**




Notes

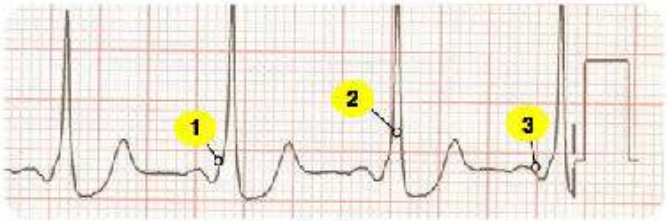

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### E. Wolff-Parkinson-White (WPW) Syndrome

Pathophysiology
• **Most commonly** caused by an accessory pathway (**bundle of Kent**) connecting the atria to the ventricles, bypassing AV node

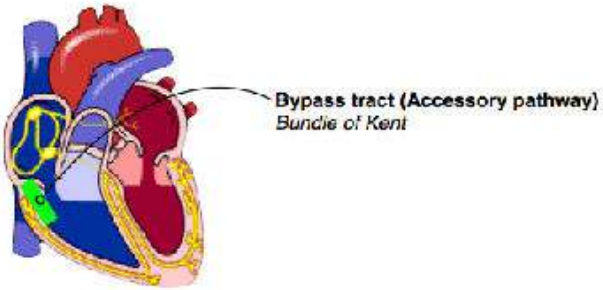
Diagnostic Studies
• ECG with a short P-R interval, wide QRS complex, and slurred upstroke of QRS (**Delta wave**)

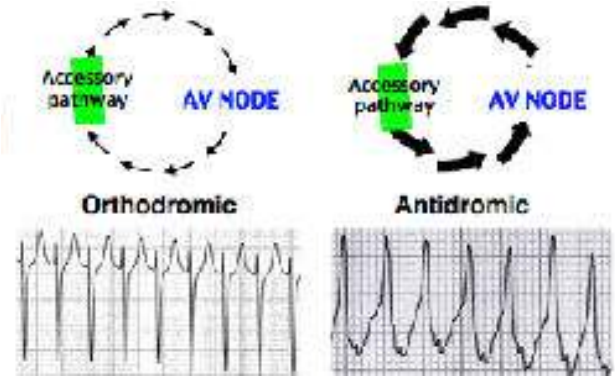
Management
• Procainamide
• Definitive treatment is **radiofrequency ablation**




1. Slurred upstroke of QRS complex (Delta wave)
2. Wide QRS complex (QRS > 120 msec)
3. Short PR interval (PR < 120 msec)

Triad




Notes


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# III. Cardiac Tamponade

### A. Cardiac Tamponade

Presentation
• Dyspnea and chest pain

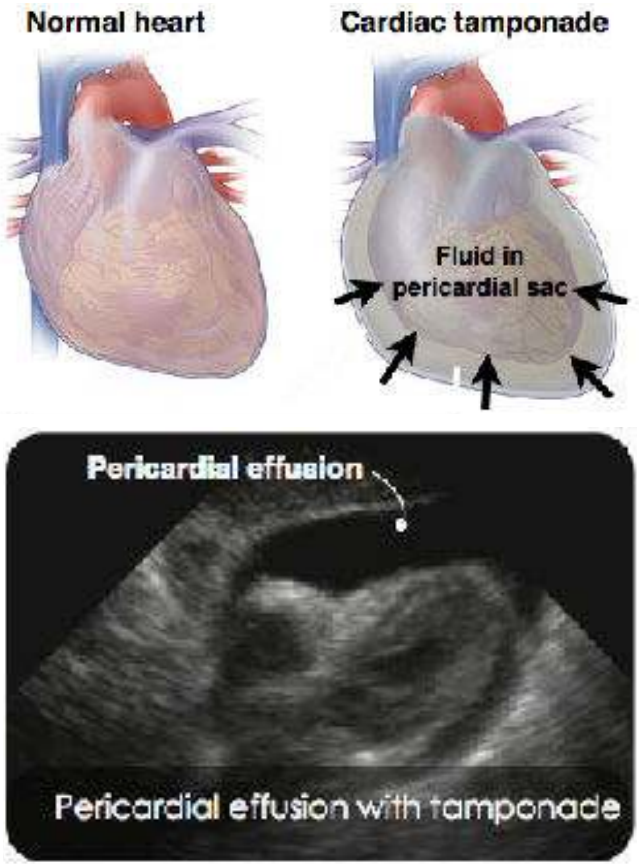
Physical Exam
• **Muffled heart sounds**, **JVD**, **hypotension** (**Beck triad**), pulsus paradoxus

Diagnostic Studies
• ECG with low-voltage QRS and **electrical alternans**
• Echocardiography will show **diastolic collapse** of the right ventricle

Management
• **Pericardiocentesis**

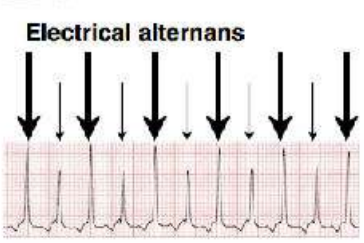
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### B. Pericardial Effusion with Tamponade




**Beck triad**

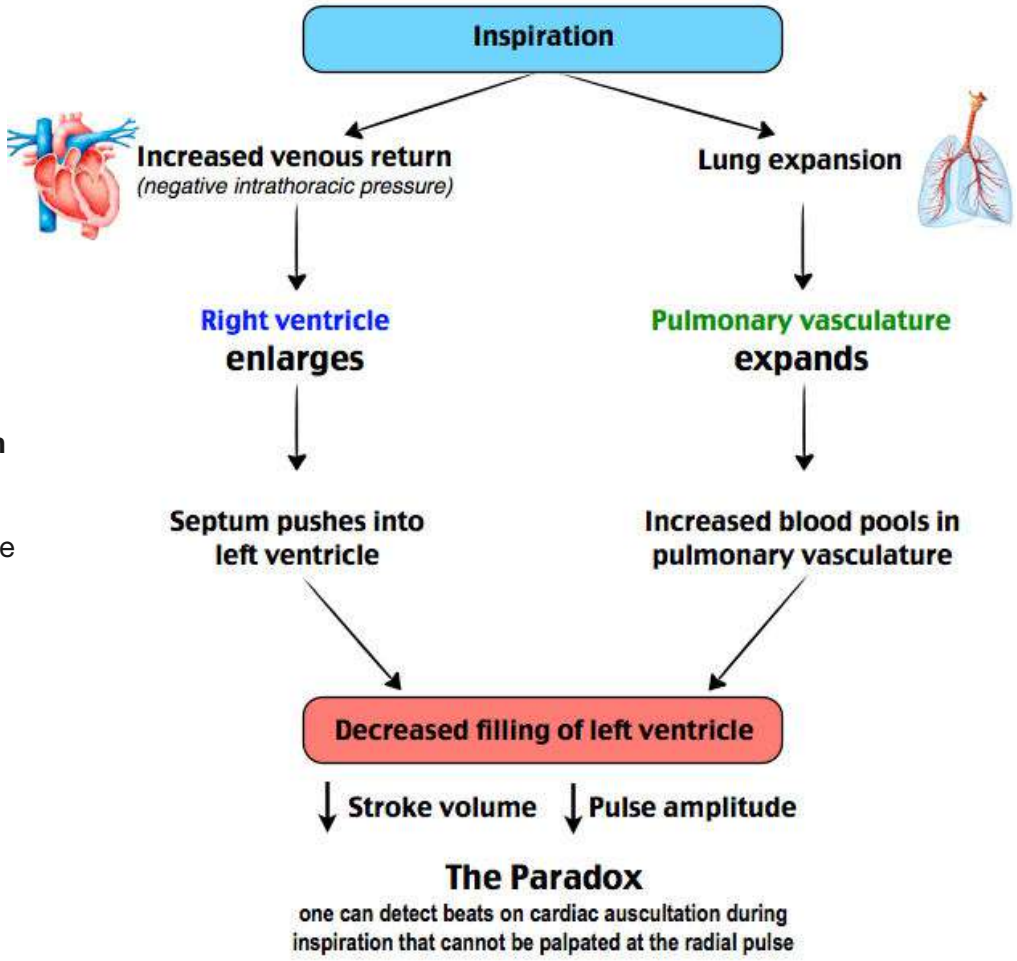
1. **Hypotension**
2. **Jugular venous distension**
3. **Muffled heart sounds**




**Management**
• Normal saline bolus (patient is preload dependent)
• Pericardiocentesis
• Pericardial window


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### C. Pulsus Paradoxus




### Conditions Associated with Pulsus Paradoxus

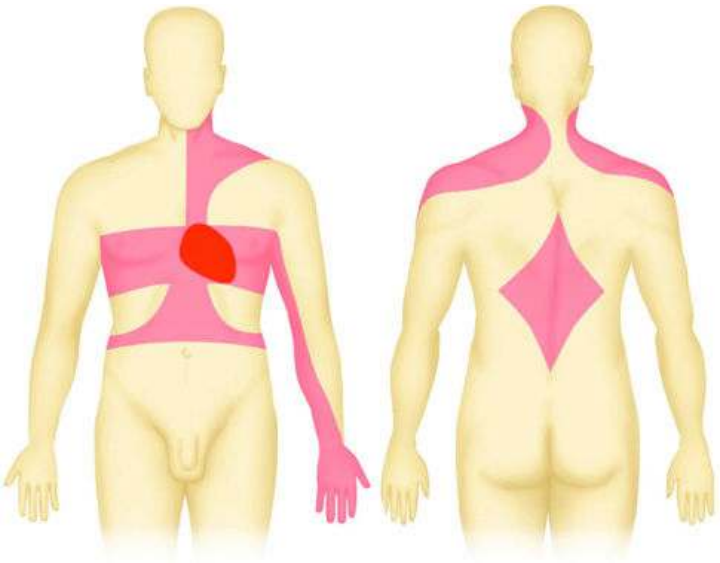
- Obstructive Lung Disease
- Cardiac Tamponade
- Constrictive Pericarditis
- Vena Cava Thrombosis
- Pulmonary Embolism

Notes


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# IV. Chest Pain

### A. Anginal Chest Pain



Notes


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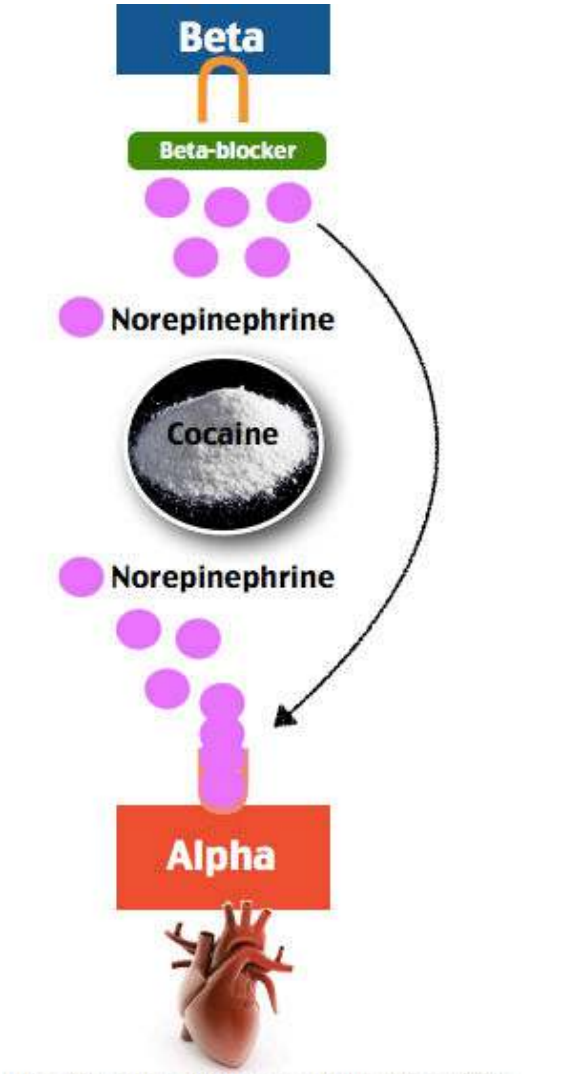
**B. Cocaine-Associated Chest Pain**

Pathophysiology
• Related to **coronary artery vasospasm**

Presentation
• MI, dysrhythmia, aortic dissection, myocarditis, stroke

Diagnostic Studies
• ECG (poor sensitivity)

Management
• Nitroglycerin, aspirin, benzodiazepines
• Avoid beta-blockers




**Unopposed alpha-receptor** stimulation leads to **coronary artery vasoconstriction** and **hypertension**

Notes


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### C. HEART Score

Utilized to risk-stratify patients with symptoms of ACS

| HEART Score for Chest Pain Patients in the Emergency Department | | |
|---|---|---|
| **Use in patients ≥ 21 years old presenting with symptoms suggestive of ACS. Do not use if new ST-segment elevation ≥1 mm or other new ECG changes, hypotension, life expectancy less than 1 year, or noncardiac medical/surgical/psychiatric illness determined by the provider to require admission.** | | |
| **Characteristics** | | **Score** |
| **History** | Highly suspicious | 2 |
| | Moderately suspicious | 1 |
| | Slightly suspicious | 0 |
| **ECG**<br>1 point: No ST depression but LBBB, LVH, repolarization changes (ex: digoxin);<br>2 points: ST depression/elevation not due to LBBB, LVH, or digoxin | Significant ST depression | 2 |
| | Nonspecific repolarization disturbance | 1 |
| | Normal | 0 |
| **Age** | ≥ 65 years | 2 |
| | 45 to 65 years | 1 |
| | < 45 years | 0 |
| **Risk factors**<br>HTN, hypercholesterolemia, DM, obesity (BMI > 30 kg/m2), smoking (current, or smoking cessation ≤ 3 mo), positive family history (parent or sibling with CVD before age 65); atherosclerotic disease: prior MI, PCI/CABG, CVA/TIA, or peripheral arterial disease | > 3 risk factors or history of atherosclerotic disease | 2 |
| | 1 or 2 risk factors | 1 |
| | No risk factors known | 0 |
| **Troponin**<br>Use local assays and corresponding cutoffs | > 2x normal limit | 2 |
| | 1 to 2x normal limit | 1 |
| | ≤ normal limit | 0 |


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## D. Causes of Chest Pain

| Causes of Nontraumatic Chest Pain in Children and Adolescents | | |
|---|---|---|
| **Life-threatening** *(1 to 6%)* | Cardiac conditions | • Hypertrophic cardiomyopathy<br>• Aortic stenosis<br>• Coarctation of the aorta<br>• Coronary artery abnormalities<br>• Variant angina after recreational drug use<br>• Classic angina<br>• Pericarditis<br>• Myocarditis<br>• Dilated cardiomyopathy<br>• Tachyarrhythmias<br>• Aortic aneurysm or dissection<br>• Ruptured sinus of Valsalva aneurysm |
| | • Airway foreign body<br>• Spontaneous pneumothorax<br>• Pulmonary embolism<br>• Pulmonary hypertension<br>• Sickle cell disease with acute chest syndrome<br>• Tumor Nontraumatic esophageal rupture (Boerhaave syndrome)<br>• Spinal cord compression | |
| **Common conditions** *(94 to 99%)* | Musculoskeletal conditions | • Muscle strain<br>• Costochondritis<br>• Slipping rib syndrome<br>• Precordial catch (Texidor's twinge)<br>• Fibromyalgia<br>• Pectus excavatum or carinatum |
| | Psychiatric | • Anxiety<br>• Panic disorder with or without hyperventilation syndrome<br>• Depression<br>• Hypochondriasis<br>• Somatization |
| | Respiratory | • Pneumonia (can be life-threatening)<br>• Asthma (can be life-threatening)<br>• Chronic cough with muscle strain or, if severe, fractured rib<br>• Spontaneous pneumomediastinum |
| | Gastrointestinal | • Gastroesophageal reflux disease<br>• Medication-induced ("pill") esophagitis<br>• Esophageal foreign body<br>• Esophageal spasm and achalasia<br>• Gastritis<br>• Peptic ulcer<br>• Irritable bowel disease<br>• Cholecystitis<br>• Pancreatitis |
| | Breast | • Male adolescents: Gynecomastia<br>• Female adolescents: Pregnancy, thelarche, mastitis, or fibrocystic disease |
| **Other conditions** | • Tietze syndrome<br>• Pleurodynia<br>• Herpes zoster | |


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## E. Pericarditis

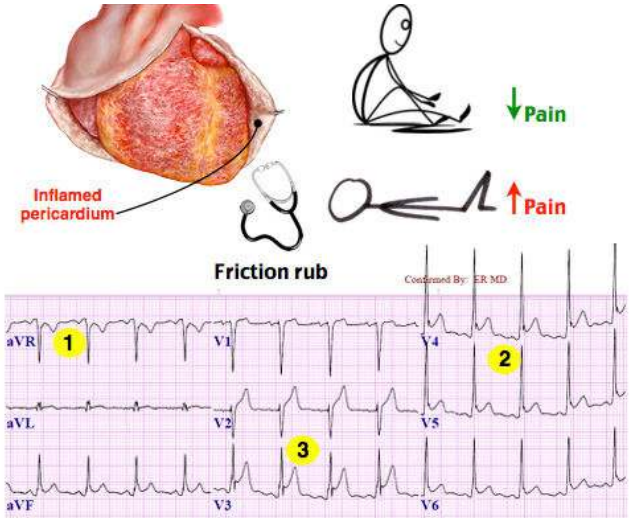
Pathophysiology
• **Most commonly** idiopathic or viral (Coxsackie)

Presentation
• Pleuritic chest pain radiating to the back that is **worse** when **lying back** and **improved** when **leaning forward**

Physical Exam
• Tachycardia and pericardial **friction rub**

Diagnostic Studies
• ECG with PR depression, PR elevation (aVR), **diffuse ST segment elevation** (concave)

Management
• **NSAIDs**, colchicine




1. **PR elevation in a VR (Thumbprint sign)**
2. **PR depression**
3. **Diffuse concave ST elevation**

**Etiology**
• Viral
• Neoplastic
• Uremic
• Connective tissue disorder

**Treatment**
• NSAIDs
• Colchicine
• Steroid (refractory cases)

Notes


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**F. Dressler Syndrome**

Patient
• **History of recent MI**

Presentation
• Pleuritic chest pain radiating to the back, **worse when lying back**, **improved when leaning forward**

Physical Exam
• Tachycardia and **pericardial friction rub**

Diagnostic Studies
• ECG will show PR depression, PR elevation (aVR), **diffuse ST segment elevation** (concave)

Management
• Colchicine, steroids, NSAIDs

Postmyocardial infarction syndrome
Postpericardiotomy pericarditis

- Occurs 2-10 weeks post myocardial infarction
- Persistent low-grade fever
- Chest pain (pleuritic)
- Pericarditis (friction rub on exam)
- Usually resolves spontaneously
- Ibuprofen/ASA, steroids, colchicine

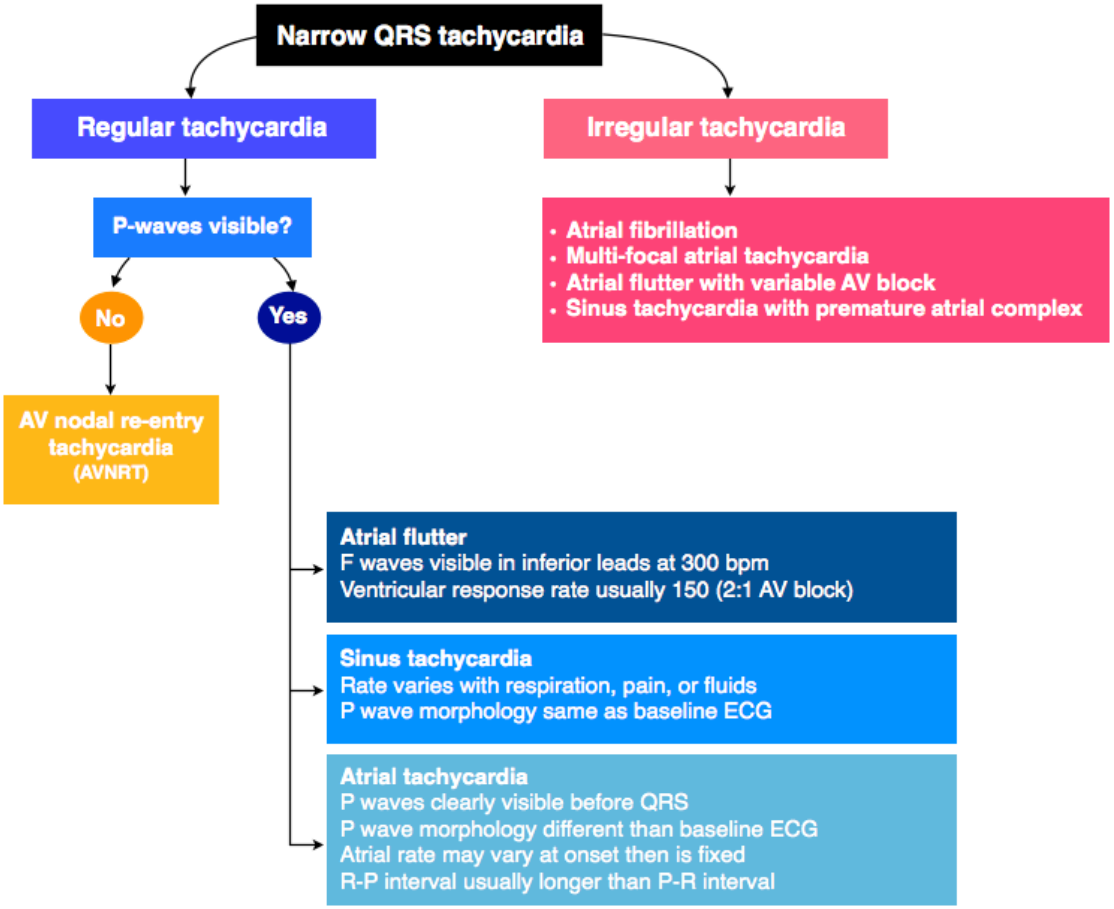
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# V. Conduction Disorders

ATRIAL FIBRILLATION / FLUTTER • SUPRAVENTRICULAR TACHYCARDIA • BUNDLE BRANCH BLOCK
VENTRICULAR TACHYCARDIA / FIBRILLATION • PREMATURE BEATS

## A. Narrow Complex Tachycardias




Notes


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**B. Atrial Fibrillation**

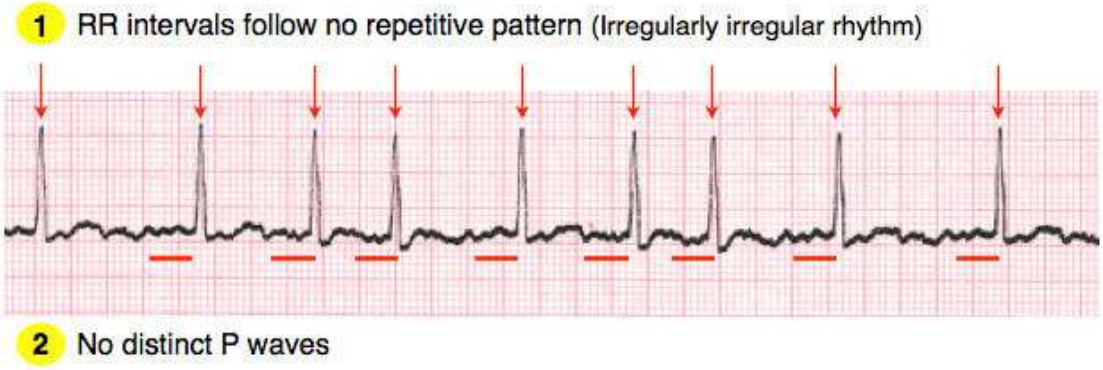
Diagnostic Studies
• ECG will show an **irregular** rate and **irregular** rhythm with **no defined P waves**

Management
• **Rate** control (CCBs, BBs) and **rhythm** control
• Consider medications, cardioversion, anticoagulation, or catheter ablation

Comments
• If condition persists **> 48 hours**, patient should be **anticoagulated** for 21 days prior to cardioversion
• Determine the need for long-term anticoagulation by using **CHA2DS2-VASc**
• **Most common** sustained dysrhythmia in adults

• **No P waves**
• Irregularly **irregular**
• **Uncoordinated** atrial activity
• Most common **sustained dysrhythmia**
• **Variable** ventricular response rate

**Management**
• Supportive care
• Unstable: Synchronized cardioversion
• Stable: Rate control is mainstay (diltiazem, metoprolol)




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### C. Atrial Flutter

Patient
• Associated with COPD, thyrotoxicosis, mitral valve disease, alcohol abuse

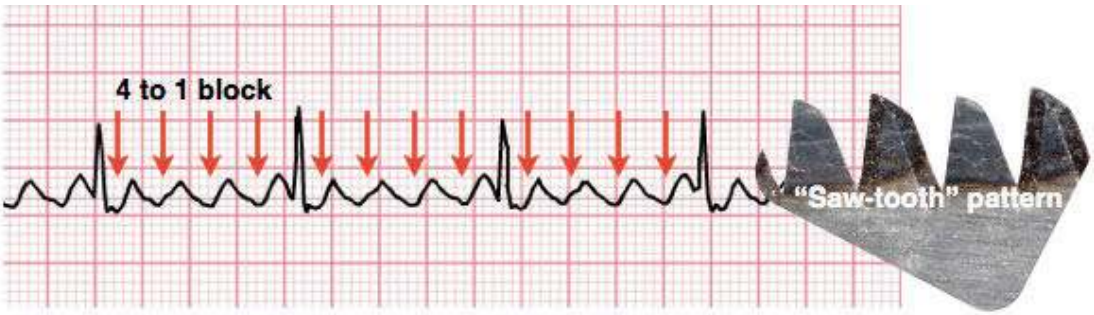
Physical Exam
• Atrial rate will be **rapid** (**250–300 beats/min**)
• Ventricular rate classically 150 bpm with 2:1 conduction

Diagnostic Studies
• ECG will show a **"sawtooth pattern"**

Management
• **Cardioversion if unstable**
• Rate control (CCBs, BBs), ablation of foci

• **Organized** electrical activity (Atrial fibrillation is *disorganized*)
• **Atrial rate** ranges from 250-300 beats per minute
• AV node conducts every **2 or 3 atrial impulses**
• Ventricular rate is classically **150 (2:1 conduction)**
• Characteristic **"saw-tooth" pattern**

**Management**
• Stable: Rate control (calcium channel blockers or beta-blockers)
• Unstable: Synchronized cardioversion




Notes


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### D. Multifocal Atrial Tachycardia

Patient
• Associated with elderly patients and those with **COPD**

Physical Exam
• HR will be **100–200 beats/min**

Diagnostic Studies
• ECG with at least **three different P wave morphologies** and **differing PR interval**

Management
• Treat the underlying cause, CCB

**Etiology**
• Secondary to chronic lung disease (COPD)
• Hypoxia
• Pulmonary hypertension

**Management**
• Oxygen
• Treatment of underlying condition
• Rate control

• At least **3 different P wave morphologies**
• Rate **100 to 180**
• **Irregular**



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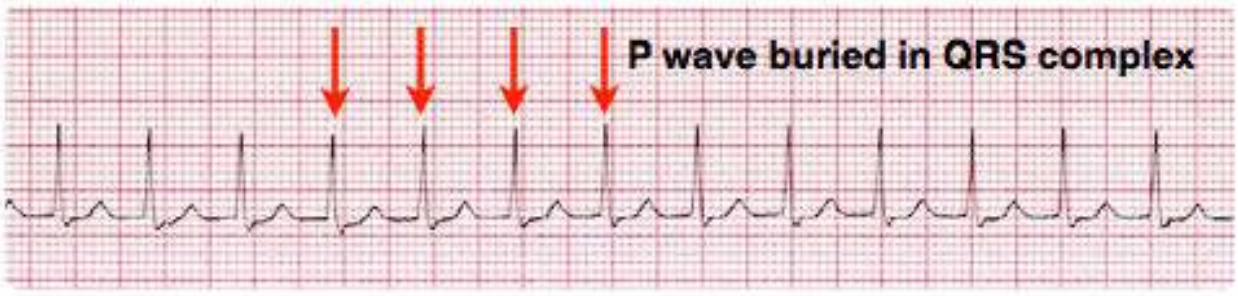
### E. Paroxysmal Supraventricular Tachycardia (PSVT)

Presentation
• Palpitations, anxiety, dyspnea, chest pain

Physical Exam
• HR will be **120–200 beats/min**

Management
• Vagal maneuver, drugs (**adenosine** is drug of choice), cold pack to face
• Cardioversion if unstable

• **P waves may not be visible** and often hidden in the preceding T waves
• Atrial **rate is 120-200**
• **Regular** rhythm
• **QRS complexes are usually narrow** (may be wide due to aberrancy such as LBBB or WPW)
• No extra beats
• If P waves are visible, 1:1 relationship with QRS

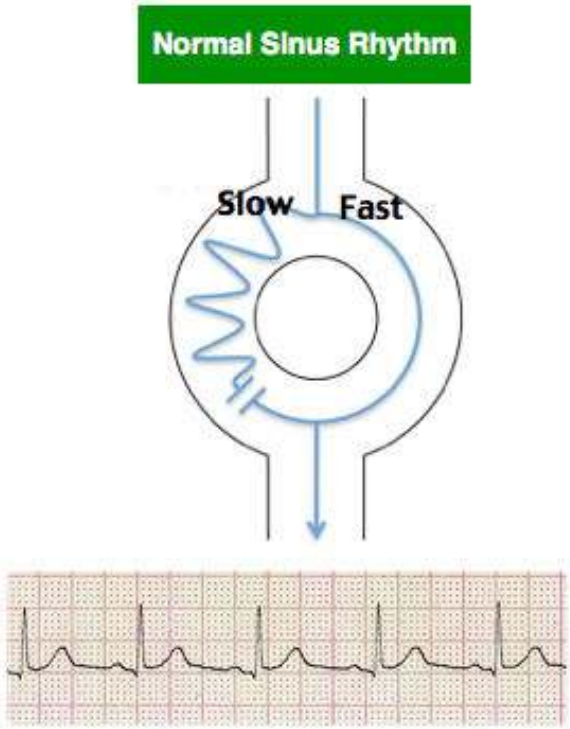


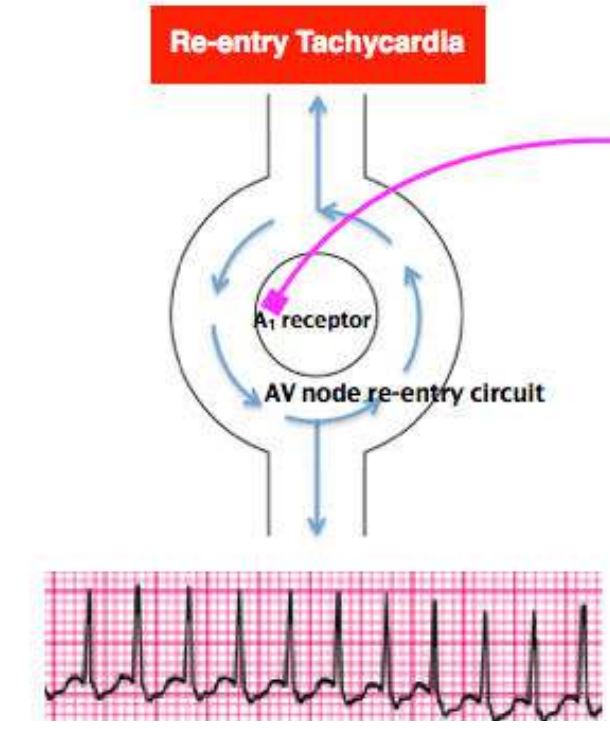

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## F. Supraventricular Tachycardia

Notes




- Purine nucleoside
- Slows conduction through AV node
- Interrupts re-entry pathways

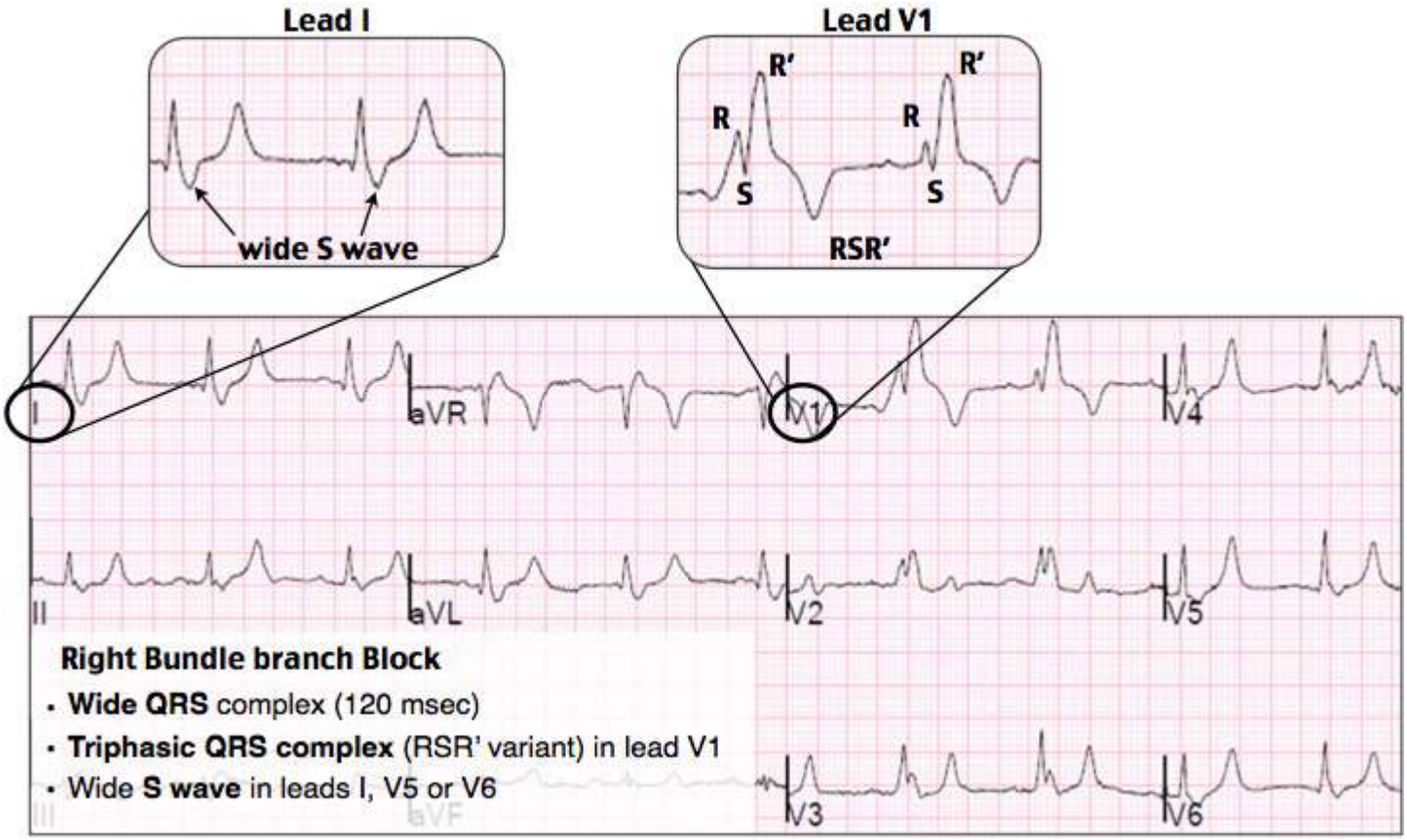
Can attempt **vagal maneuvers** prior to adenosine administration


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### G. Right Bundle Branch Block (RBBB)

Diagnostic Studies

- Notable ECG features:
  - **Wide QRS** (> 0.12 sec)
  - Predominantly positive QRS in V1
  - **rSR'**, **rsR'**, or **Rsr'** in lead V1
  - Wide **S wave** in lead I, V5, or V6

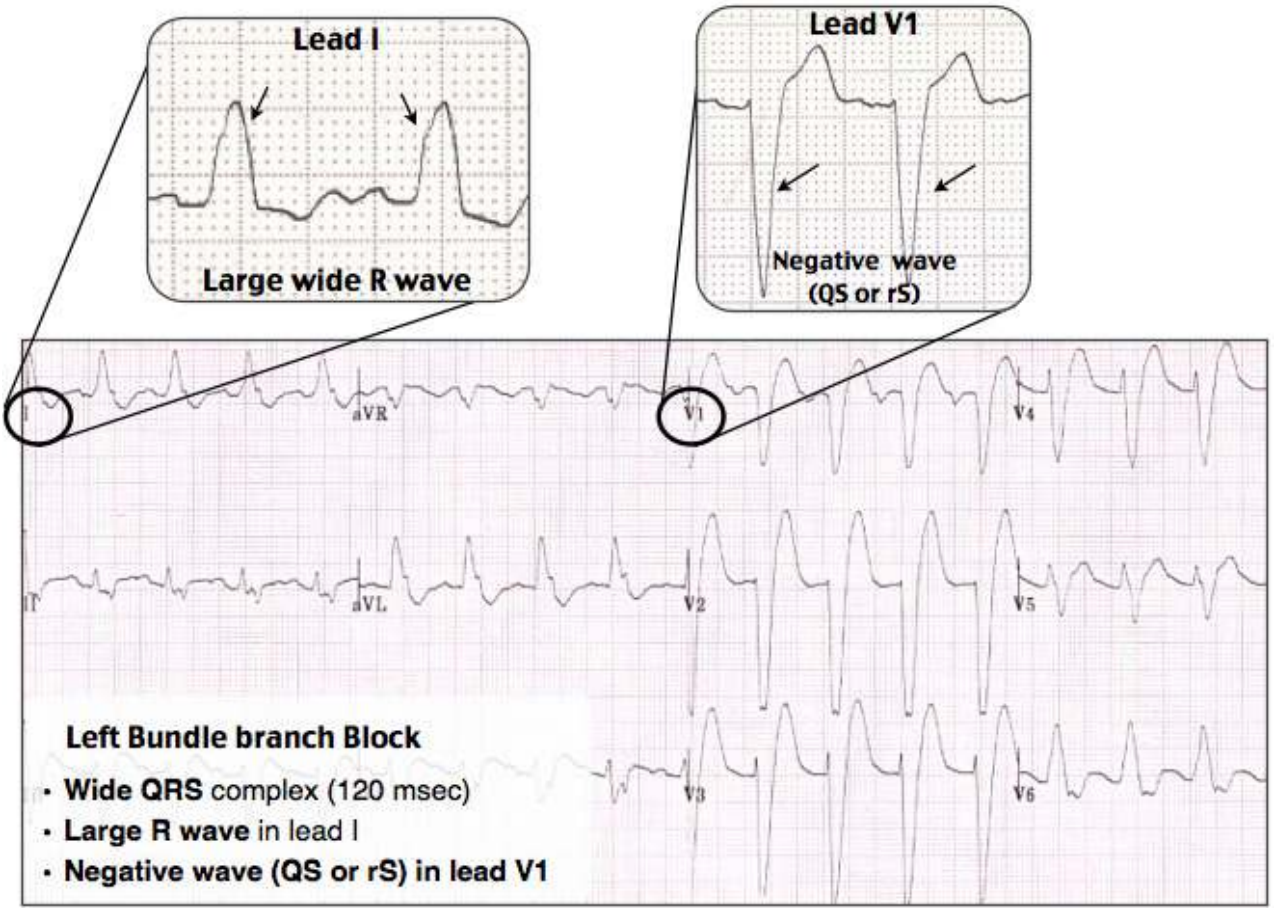



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## H. Left Bundle Branch Block (LBBB)

- Notable ECG features:
  - **Wide QRS** (> 0.12 sec)
  - Broad, slurred R wave in V4 and V6
  - Deep S wave in V1 and V2
  - ST elevations in V1–V3




Notes


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## I. Ventricular Fibrillation

Patient
• Associated with ischemic heart disease and ventricular dysfunction

Diagnostic Studies
• **Uncoordinated**, **disorganized** electrical activity on ECG

Management
• **Defibrillation** at maximum joules (**200 J** biphasic)

Comments
• Condition is life-threatening

• Completely **disorganized**
• Immediate cessation of cardiac output, **no associated pulse**
• No discernible P waves, QRS complexes, or T waves
• **Incompatible** with life



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## J. Ventricular Tachycardia

Pathophysiology
• Associated with electrolyte imbalance, acid-base changes, hypoxemia, MI

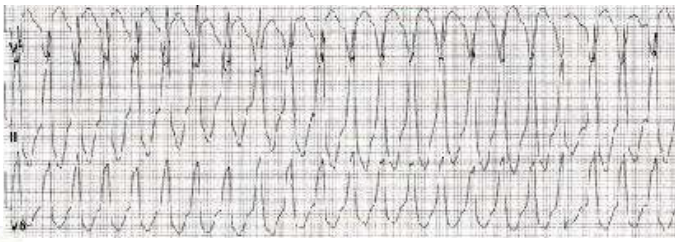
Physical Exam
• HR will be **> 100 beats/min**

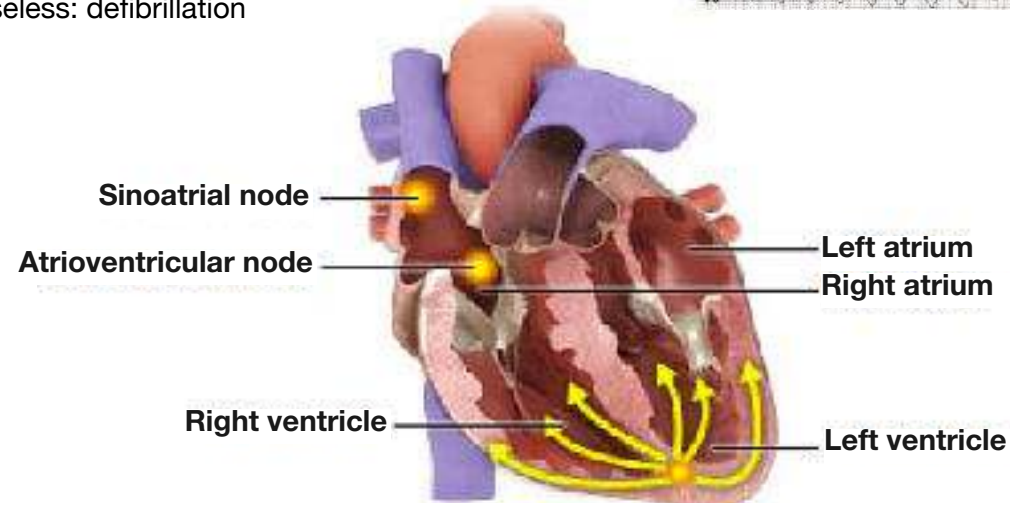
Diagnostic Studies
• Notable ECG features:
  • QRS interval **> 120 msec**
  • QRS complex **wide and bizarre** appearing, though somewhat uniform, AV dissociation, presence of fusion beats

Management
• Stable: amiodarone, lidocaine, procainamide
• Unstable: cardioversion
• Pulseless: defibrillation

**Management**
• Supportive care
• Synchronized cardioversion (unstable)
• Anti-dysrhythmic

• AV dissociation
• QRS > 120 msec
• Rate > 100 (typically 150-200)
• Hypoglycemia
• Obstructive sleep apnea






Notes


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## K. Torsades de Pointes (TdP)

Pathophysiology
• **Most common** cause is acquired or congenital long QT **interval syndrome**
• Use of antipsychotic medications, methadone

Physical Exam
• HR will be **> 100 beats/min**

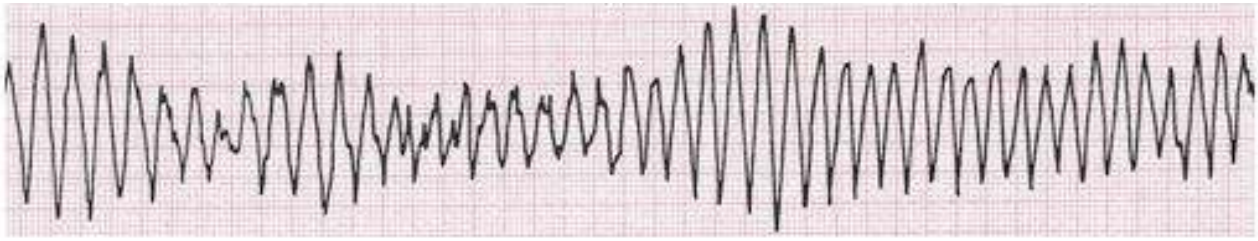
Diagnostic Studies
• ECG with frequent variation in the QRS axis and morphology

Management
• Unstable: defibrillation
• Stable: intravenous **magnesium sulfate** and cessation of the offending drug

**Torsades de pointes**
**Polymorphic ventricular tachycardia** due to **QT prolongation**

**Some Causes**
• Precipitated by drugs that prolong the QT interval
• Class IA antidysrhythmics (procainamide, quinidine)
• Class IC antidysrhythmics (propafenone, flecanide)
• Cyclic antidepressants
• Droperidol
• Phenothiazines
• Erythromycin
• Fluoroquinolones
• Methadone
• Electrolyte disorders (hypomagnesemia, hypokalemia)

**Management**
Pulseless: Defibrillation
Unstable: Cardioversion
Stable: Intravenous magnesium sulfate



**QRS axis swings from positive to negative direction single lead**

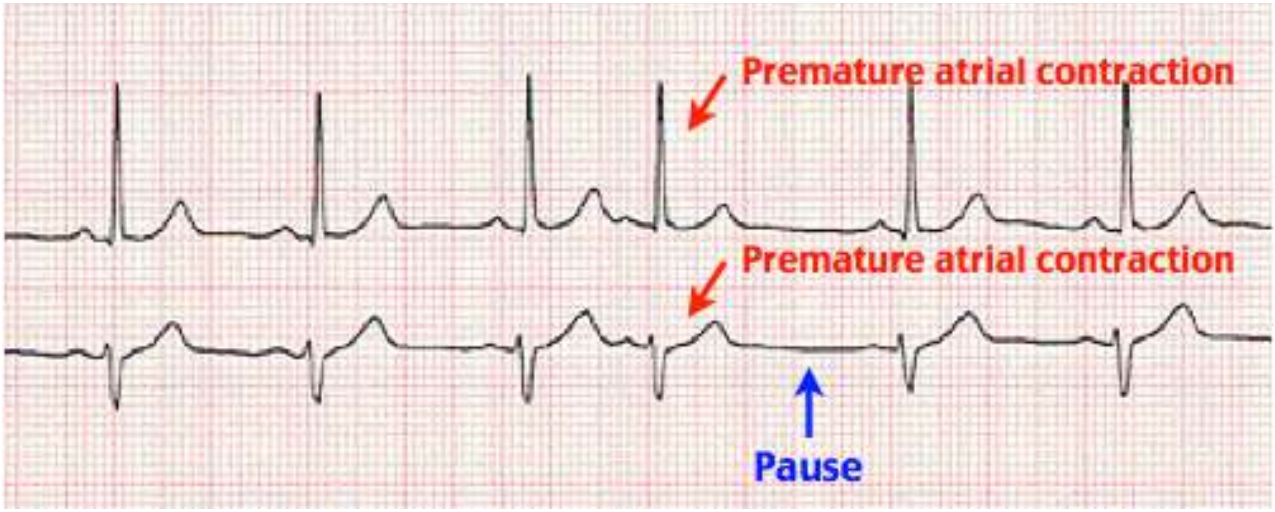
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### L. Premature Atrial Contractions (PACs)

- Abnormally shaped **P wave** followed by a QRS complex

- **Extra beats** that originate outside the sinus node
- From **ectopic atrial pacemaker**
- Appear **interspersed** throughout an underlying rhythm




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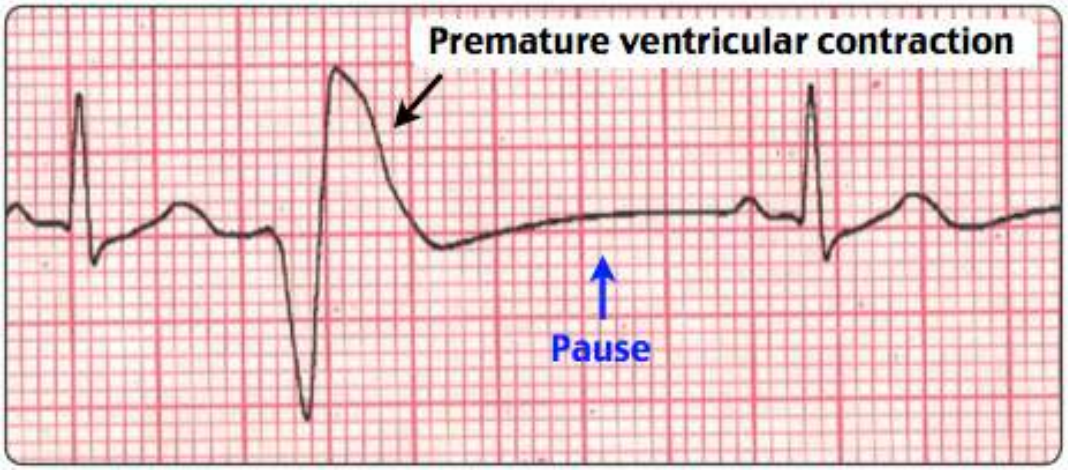

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### M. Premature Ventricular Contractions (PVCs)

• Early, wide, "bizarre" QRS, no P wave seen

• **Occur earlier** than the next expected normal QRS
• **Wider** than a normal QRS
• QRS morphology is generally **bizarre**
• Preceding **P wave is absent**

• Deflection of the ST segment and T wave is **opposite** that of the QRS
• Followed by a **compensatory pause**




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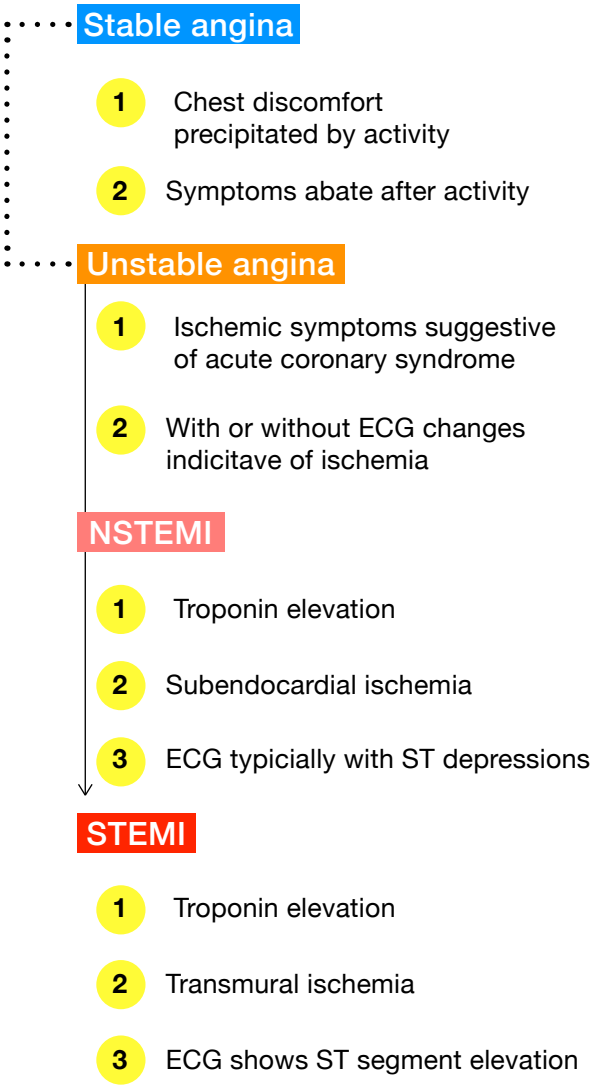

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# VI. Coronary Heart Disease

NON-ST ELEVATION MYOCARDIAL INFARCTION • ST ELEVATION MYOCARDIAL INFARCTION
ANGINA PECTORIS • UNSTABLE ANGINA • PRINZMETAL/VARIANT ANGINA

## A. Coronary artery disease

• **Stable angina (angina pectoris)**: chest discomfort precipitated by activity but **symptoms abate after activity**
• **Unstable angina**: ischemic symptoms suggestive of an acute coronary syndrome with or without ECG changes indicative of ischemia
• **NSTEMI**: troponin elevation, ECG typically with ST segment **depressions** but may be normal
• **STEMI**: troponin elevation, ECG shows ST segment **elevation**




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### B. Initial Therapy for Unstable Angina and Non-ST Elevation Myocardial Infarction

| | |
|---|---|
| **Supplemental oxygen** | • Oxygen saturation < 90%<br>• Respiratory distress<br>• High risk features for hypoxemia |
| **Nitroglycerin** | • Start with sublingual<br>• Intravenous for persistent pain<br>• Avoid in right ventricular infarct, severe aortic stenosis<br>• Contraindicated with phosphodiesterase inhibitors (w/in 24 hrs for ED) |
| **Morphine** | • Avoid if possible, associated with adverse outcomes<br>• Reserved for patients with severe pain |
| **Beta blockers** | • Benefit in acute myocardial infarction |
| **Statin therapy** | • All patients with acute coronary syndrome<br>• High-intensity statin therapy regardless of baseline levels |
| **Antiplatelet therapy** | • Aspirin for all patients, unless absolute contraindication |
| **Anticoagulation** | • Unfractionated heparin<br>• Low molecular weight heparin |

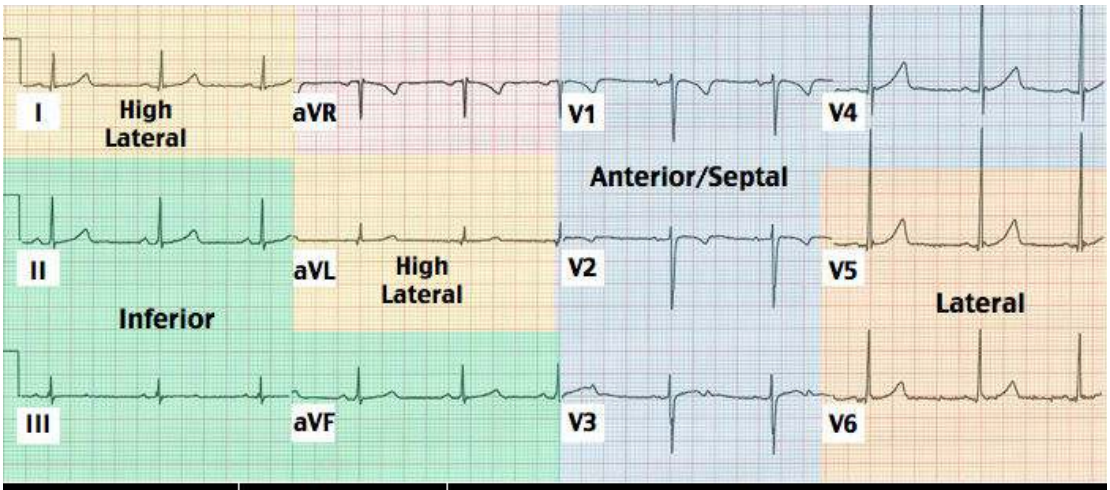
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### C. Acute Coronary Syndrome

• **Anterior and Septal**: V1–V4; left anterior descending (**LAD**)
• **Inferior**: II, III, aVF; right coronary artery (**RCA**) **more common** than left circumflex (LCx)

• **Lateral**: I, aVL, V5–V6; **LCx**, diagonal of left anterior descending




| Location | Leads | Artery |
|---|---|---|
| **Anterior and Septal** | V1–V4 | Left anterior descending (LAD) |
| **Inferior** | II, III, aVF | Right coronary artery (RCA) (70%+) or Left circumflex (LCx) |
| **Lateral** | I, aVL, V5–V6 | Left circumflex (LCx), Diagonal of Left anterior descending |

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## D. Initial Interventions for Management of Acute Coronary Syndrome

**ABCs**
- Assess and stabilize airway, breathing, and circulation.

**IV, $O_2$, Monitor**
- Attach cardiac and oxygen saturation monitors; provide supplemental oxygen as needed to maintain $O_2$ saturation > 90%. Establish IV access.

**Dysrhythmia**
- Treat sustained ventricular dysrhythmia rapidly according to ACLS protocols.

**Aspirin**
- Give aspirin 325 mg (nonenteric coated) to be chewed and swallowed (unless aortic dissection is being considered). If oral administration is not feasible, give as rectal suppository.

**H and P**
- Perform focused history and examination: Look for signs of hemodynamic compromise and left heart failure; determine baseline neurologic function, particularly if fibrinolytic therapy is to be given.

**Labs**
- Obtain blood for cardiac biomarkers (troponin preferred), electrolytes, hematocrit/hemoglobin. Perform coagulation studies for patients taking anticoagulants or as otherwise indicated

**Pain Control, NTG**
- Give three sublingual nitroglycerin tablets (0.4 mg) one at a time, spaced five minutes apart, or one aerosol spray under tongue every five minutes for three doses **IF** patient has persistent chest discomfort, hypertension, or signs of heart failure **AND** there is no sign of hemodynamic compromise (e.g., right ventricular infarction) and no use of phosphodiesterase inhibitors (e.g., for erectile dysfunction); add IV nitroglycerin for persistent symptoms.

**Antiplatelet therapy**
- In the absence of an absolute contraindication, antiplatelet therapy with aspirin and a platelet P2Y12 receptor blocker (e.g. clopidogrel, ticlopidine) is indicated in all patients with a non-ST elevation ACS.

**Anticoagulation**
- For all patients with non-ST elevation ACS, anticoagulant therapy is recommended as soon as possible after the diagnosis.

**Heart Failure**
- Treat left heart failure if present: Give afterload-reducing agent (e.g., nitroglycerin sublingual tablet and/or IV drip at 40 mcg/minute provided no hypotension and no phosphodiesterase inhibitors [e.g., for erectile dysfunction]; titrate drip up quickly based on response); give loop diuretic (e.g., intravenous furosemide); administer noninvasive positive pressure ventilation (eg, BLPAP) to appropriate patients.

**Beta-Blocker**
- Give beta-blocker (e.g., metoprolol tartrate 25 mg orally) **IF** no signs of heart failure and not at high risk for heart failure and no signs of hemodynamic compromise, bradycardia, or severe reactive airway disease. If hypertensive, may initiate beta blocker IV instead (e.g., metoprolol tartrate 5 mg intravenous every five minutes for three doses as tolerated).

**Persistent Pain**
- Give morphine sulfate (2 to 4 mg slow IV push every 5 to 15 minutes) for unacceptable, persistent discomfort or anxiety related to myocardial ischemia. [Controversial]

**Statin**
- Start 80 mg of atorvastatin as early as possible and preferably before PCI in patients not on statin. If patient is taking a low to moderate intensity statin, switch to atorvastatin 80 mg.


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### E. Prinzmetal Angina (Variant Angina)

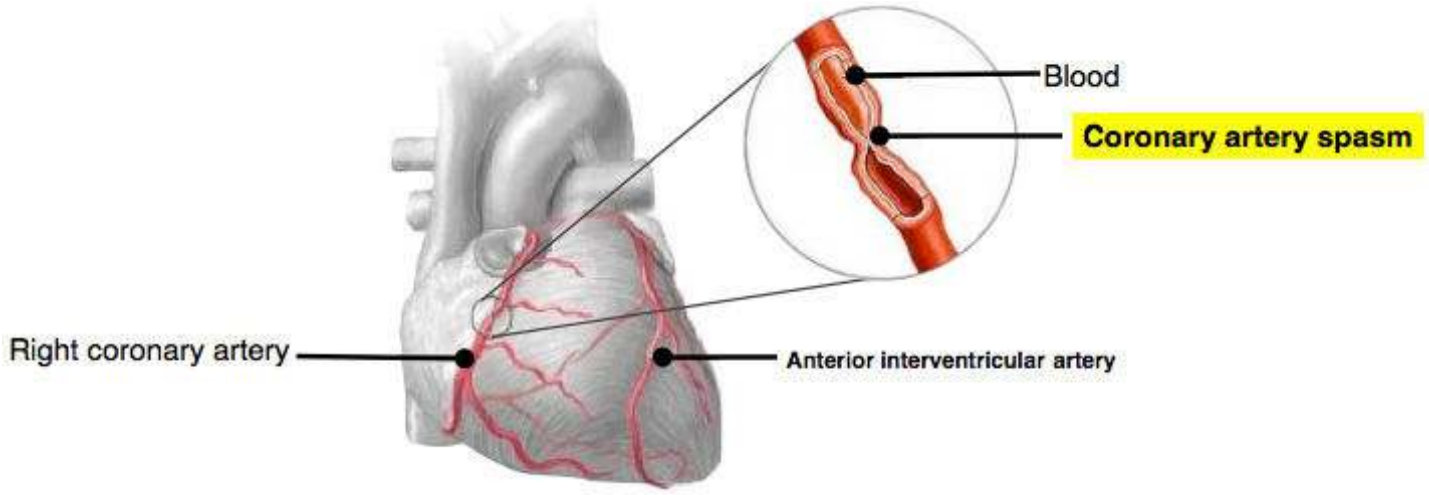
Pathophysiology
- **Most commonly** caused by **coronary artery spasm**

Patient
- History of HTN, **smoking**, DM, obesity, or **cocaine use**

Presentation
- Squeezing, pressure-like chest discomfort **at rest**

Diagnostic Studies
- ECG with **transient ST segment elevation**
- **Cardiac enzymes** will be **normal**
- Diagnosis is made by a **cardiac stress test**

Management
- **CCBs** and **nitrates**




**COVADIS Diagnostic Criteria for Vasospastic Angina**

**1. Nitrate-responsive angina (during spontaneous episode, with at least one of the following):**
- Rest angina, especially between night and early morning
- Marked diurnal variation in exercise tolerance, reduced in morning
- Hyperventilation can precipitate an episode
- Calcium channel blockers (but not beta blockers) suppress episodes

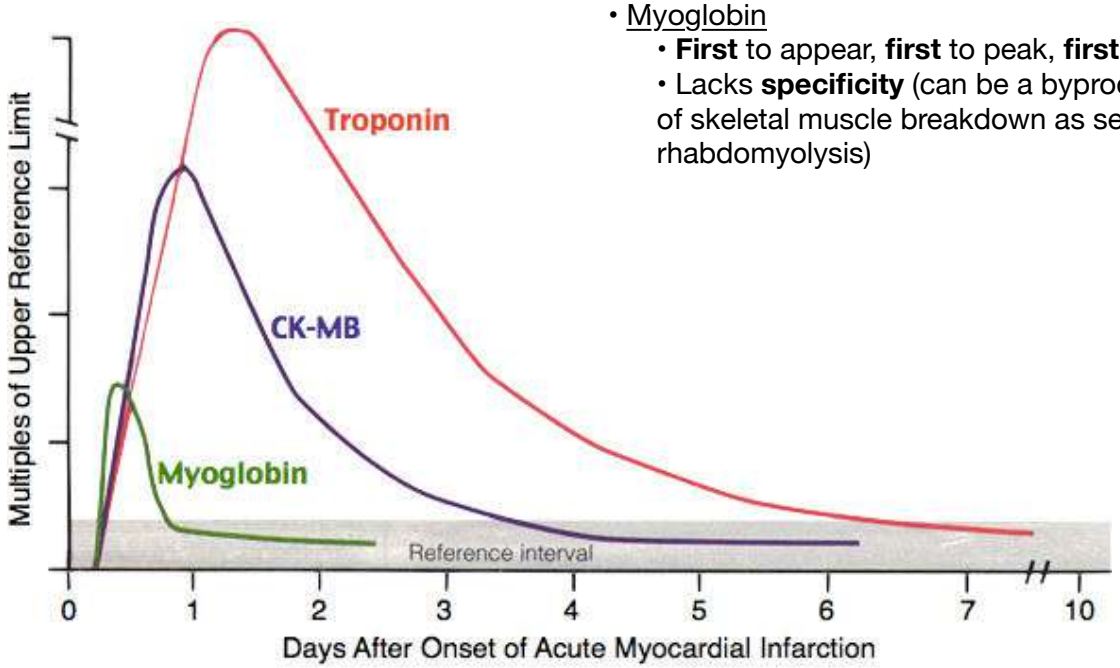
**2. Transient ischemic ECG changes (during spontaneous episode, including any of the following in at least two contiguous leads):**
- ST segment elevation ≥ 0.1 mV
- ST segment depression ≥ 0.1 mV
- New negative U waves

**3. Coronary artery spasm:** Defined as transient total or subtotal coronary artery occlusion (> 90% constriction) with angina and ischemic ECG changes either spontaneously or in response to a provocative stimulus (typically acetylcholine, ergot, or hyperventilation)


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### F. Cardiac Biomarkers

- Troponin
  - **Highest** sensitivity and specificity
  - Time detectable from onset: 3–12 hours
  - Peak: 24–48 hours
  - Return to baseline: 5–14 days
- CK-MB
  - Time detectable from onset: 3–12 hours
  - Peak: 24 hours
  - Return to baseline: 48–72 hrs
  - Useful for **diagnosing reinfarction**
- Myoglobin
  - **First** to appear, **first** to peak, **first** to decline
  - Lacks **specificity** (can be a byproduct of skeletal muscle breakdown as seen in rhabdomyolysis)




| | Time Detectable from Onset of Ischemia | Peak | Return to Baseline |
|---|---|---|---|
| Troponin T & I | 3-12 hrs | 24-48 hrs | 5-14 days |
| Creatine kinase (CK) | 3-12 hrs | 24 hrs | 48-72 hrs |
| Myoglobin | 1-2 hrs | 8-10 hrs | 1-2 days |

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# VII. Dyspnea on Exertion

### A. Dyspnea on exertion

• Defined as difficulty breathing when engaged in a simple activity like walking up stairs or going a short distance

### B. Physical Examination of Dyspnea

| Sign | Clinical significance | Possible diagnosis |
|---|---|---|
| **Absent or diminished breath sounds** | Decreased air movement | • COPD<br>• Severe asthma<br>• Pneumothorax<br>• Tension pneumothorax<br>• Hemothorax |
| **Accessory muscle use** | Muscle weakness, fatigue | • Respiratory failure<br>• Severe COPD<br>• Severe asthma |
| **Expiratory or mixed stridor** | Air flow obstruction below vocal cords | • Croup<br>• Foreign body<br>• Bacterial tracheitis |

Notes


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| Sign | Clinical significance | Possible diagnosis |
|---|---|---|
| **Inspiratory stridor** | Air flow obstruction above vocal cords | • Foreign body<br>• Epiglottitis<br>• Angioedema |
| **Hyperventilation** | - | • Acidosis<br>• Sepsis<br>• Salicylate poisoning<br>• Anxiety |
| **JVD with clear lungs** | Right heart failure | • Cardiac tamponade<br>• Pulmonary embolism |
| **JVD with crackles** | Right and left heart failure | • Acute decompensated heart failure<br>• ARDS |
| **Heart murmur** | Valvular disease | • Valvular dysfunction |
| **Hepatojugular reflex** | Right heart failure | • Acute decompensated heart failure |
| **Pulsus paradoxus** | Poor right heart filling | • Right heart failure<br>• Pulmonary embolism<br>• Cardiogenic shock<br>• Pericardial tamponade<br>• Asthma exacerbation |
| **Crackles (rales)** | Interalveolar fluid | • Acute decompensated heart failure<br>• ARDS<br>• Pneumonia |
| **Wheezes** | Obstruction below trachea | • Asthma exacerbation<br>• Foreign body<br>• ADHF<br>• COPD |

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# VIII. Edema

### A. Edema

• Swelling caused by excess fluid trapped in the body's tissues, may be a sign of right-sided heart failure

Notes


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# IX. Heart Failure

### A. Acute Decompensated Heart Failure

Presentation
• Exertional dyspnea, **orthopnea**, paroxysmal nocturnal dyspnea

Physical Exam
• Pitting edema, **$S_3$** heart sound

Diagnostic Studies
• **Elevated BNP**
• Chest X-ray will show cardiomegaly, cephalization, **Kerley B lines**, effusions
• **Echocardiogram** is the most useful diagnostic tool

Management
• **BPAP**: **increase** oxygenation, **decrease** work of breathing, **decrease** preload/afterload
• **Nitroglycerin**: **decrease** preload/afterload
• **Furosemide**: diuresis
• Hypotension without signs of shock: **dobutamine** (may worsen hypotension)
• Severe hypotension with signs of shock: **norepinephrine** (**increase** systemic vascular resistance, **increase** HR, **increase** BP, **increase** myocardial oxygen demand)

| High-output heart failure | Low-output heart failure |
|---|---|
| Due to a **high metabolic state** or shunting of blood that increases myocardial oxygen demand | Most commonly caused by a **depressed ejection fraction** |
| • Hyperthyroidism<br>• Beriberi<br>• AV fistula<br>• Paget's disease<br>• Severe anemia<br>• Pregnancy | • Dilated cardiomyopathy<br>• Chronic hypertension<br>• Valvular heart disease |

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### B. Heart Failure Staging/Classification

- American Heart Association and American College of Cardiology staging:
  - Stage A: high risk without symptoms or disease
  - Stage B: structural disease without symptoms
  - Stage C: structural disease and symptoms
  - Stage D: refractory heart failure
- New York Heart Association classification:
  - I: asymptomatic
  - II: symptoms with ordinary activity
  - III: symptoms with minimal activity
  - IV: symptoms at rest

| New York Heart Association (NYHA) Classification | | |
|---|---|---|
| Class | Functional state | Symptoms |
| I | No limitation | Asymptomatic during usual daily activities |
| II | Slight limitation | Mild symptoms (dyspnea, fatigue, or chest pain) with ordinary activities |
| III | Moderate limitation | Symptoms noted with minimal activity |
| IV | Severe limitation | Symptoms at rest. Any activity results in limitations. |

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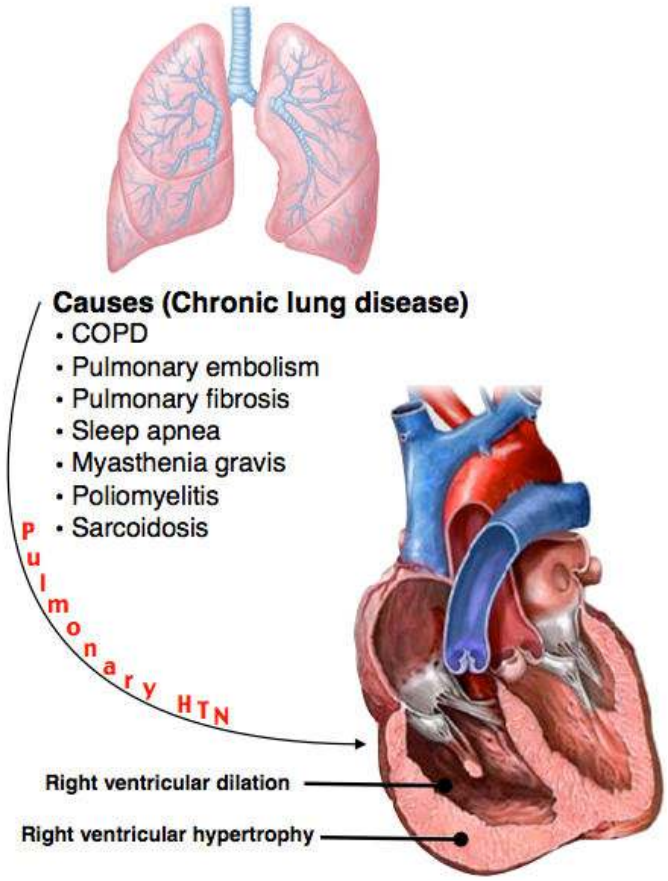
### C. Cor Pulmonale

Pathophysiology
• **Most common** chronic cause: **COPD**
• **Most common** acute cause: **pulmonary embolism**

Presentation
• Peripheral edema, dyspnea, fatigue, and signs of **right-sided heart failure**

Physical Exam
• Signs of pulmonary HTN and **right ventricular hypertrophy**

Management
• Right heart catheterization




Notes


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# X. Hypertensive Urgency and Emergency

### A. Hypertensive Urgency and Emergency

| Condition | Proposed definition | Notes |
|---|---|---|
| **Hypertensive urgency** | Severe hypertension (≥180/≥120 mm Hg) in asymptomatic patients who are not experiencing end-organ damage | • No proven benefit from rapid reduction in BP<br>• Can be addressed in the office setting |
| **Hypertensive emergency** | Severely elevated blood pressure or an acute rise in blood pressure with acute end-organ damage | • Gradually reduce blood pressure by 10 to 20 percent in the first hour and by a further 5 to 15 percent over the next 23 hrs<br>• Often results in target BP <180/<120 mm Hg for the first hour and < 160/<110 for the next 23 hrs |

Notes


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### B. Treatment of Hypertensive Emergencies

| Drug | Onset of action | Adverse effects | Special indications |
|---|---|---|---|
| **Vasodilators** | | | |
| **Sodium nitroprusside** | Immediate | Nausea, vomiting, muscle twitching, thiocyanate and cyanide poisoning | • May cause reflex tachycardia<br>• Combine with beta-blocker<br>• Caution with azotemia<br>• Caution with elevated intracranial pressure |
| **Nicardipine** | 5-10 min | Tachycardia, headache, flushing, local phlebitis | • Caution with coronary ischemia and heart failure |
| **Fenoldopam** | < 5 min | Tachycardia, headache, nausea, flushing | • Caution with glaucoma |
| **Nitroglycerin** | 2-5 min | Headache, vomiting | • Preferred in coronary ischemia |
| **Enalaprilat** | 15-30 min | Precipitous fall in BP in high renin states | • Preferred in acute left ventricular failure<br>• Avoid in acute myocardial infarction |
| **Hydralazine** | 10-20 min | Tachycardia, flushing, headache, aggravation of angina | • Eclampsia |
| **Adrenergic inhibitors** | | | |
| **Labetalol** | 5-10 min | Vomiting, scalp tingling, bronchoconstriction, heart block, orthostatic hypotension | • Caution in acute heart failure |
| **Esmolol** | 1-2 min | Hypotension, nausea, asthma, first degree heart block, heart failure | • Preferred in aortic dissection |
| **Phentolamine** | 1-2 min | Tachycardia, flushing, headache | • Preferred in catecholamine excess states |

Notes


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# XI. Hypotension

CARDIOGENIC SHOCK • ORTHOSTATIC HYPOTENSION

### A. Cardiogenic Shock

Pathophysiology
• **Most commonly** caused by complications of **acute MI**

Presentation
• Lethargy, confusion, somnolence

Physical Exam
• Weak peripheral pulses, rapid HR, hypotension (< 90 mm Hg systolic)

Management
• Stabilize with **fluids**, pressers, oxygen

**Hemodynamics of Shock**

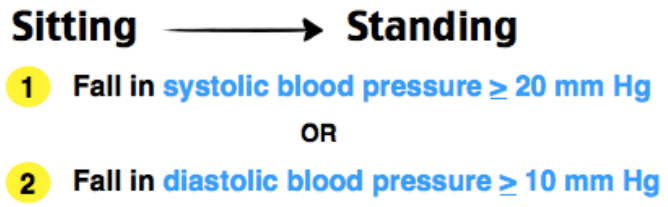
| Type of Shock | Pulmonary capillary wedge pressure (PCWP) | Cardiac output (CO) | Systemic vascular resistance (SVR) |
|---|---|---|---|
| Hypovolemic | ↓ | ↑ | ↑ |
| Cardiogenic | ↑ | ↓ | ↑ |
| Distributive (e.g. septic, neurogenic) | ↑ | ↑ | ↓ |

Notes


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**B. Orthostatic hypotension**

• Drop of ≥ 20 mm Hg systolic, 10 mm Hg diastolic, or both, two to five minutes after change from supine to standing




Notes


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# XII. Orthopnea

### A. Orthopnea

• Dyspnea while lying down flat

Notes


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# XIII. Palpitations

### A. Palpitations

• A noticeably rapid, strong, or irregular heartbeat

### B. Causes of Palpitations

| | |
|---|---|
| **Cardiac** | • Any dysrhythmia<br>• Cardiac and Extracardiac shunts<br>• Valvular heart disease<br>• Pacemaker<br>• Atrial myxoma<br>• Cardiomyopathy |
| **Psychiatric disease** | • Panic attack and disorder<br>• Generalized anxiety disorder<br>• Somatization<br>• Depression |
| **Medications** | • Sympathomimetic agents<br>• Vasodilators<br>• Anticholinergic drugs<br>• Beta blocker withdrawal |
| **Habits** | • Cocaine<br>• Amphetamines<br>• Caffeine<br>• Nicotine |
| **Metabolic disorders** | • Hypoglycemia<br>• Thyrotoxicosis<br>• Pheochromocytoma<br>• Mastocytosis<br>• Scombroid food poisoning |
| **High output states** | • Anemia<br>• Pregnancy<br>• Paget's disease<br>• Fever |
| **Catecholamine excess** | • Stress<br>• Exercise |

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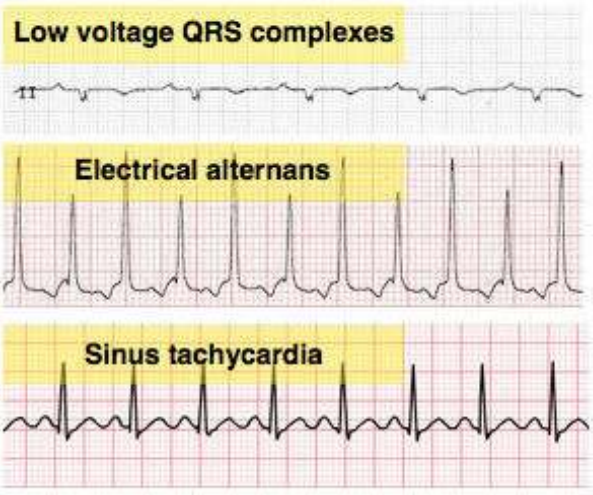
# XIV. Pericardial Effusion

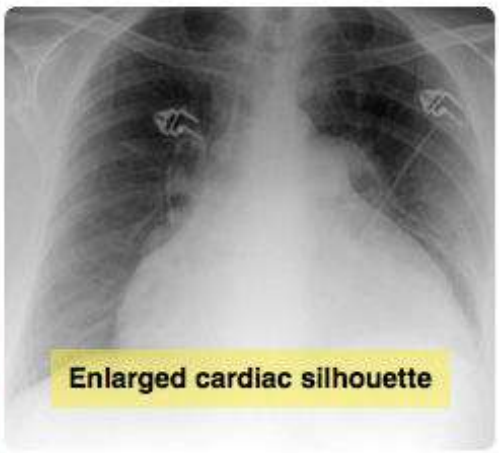
### A. Pericardial Effusion

• See topic "Cardiac Tamponade"








Notes


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# XV. Peripheral Vascular Disease

### A. Peripheral Artery Disease

Pathophysiology
• **Most commonly** caused by atherosclerotic disease

Presentation
• Pain in affected extremity related to the activity (**intermittent claudication**)

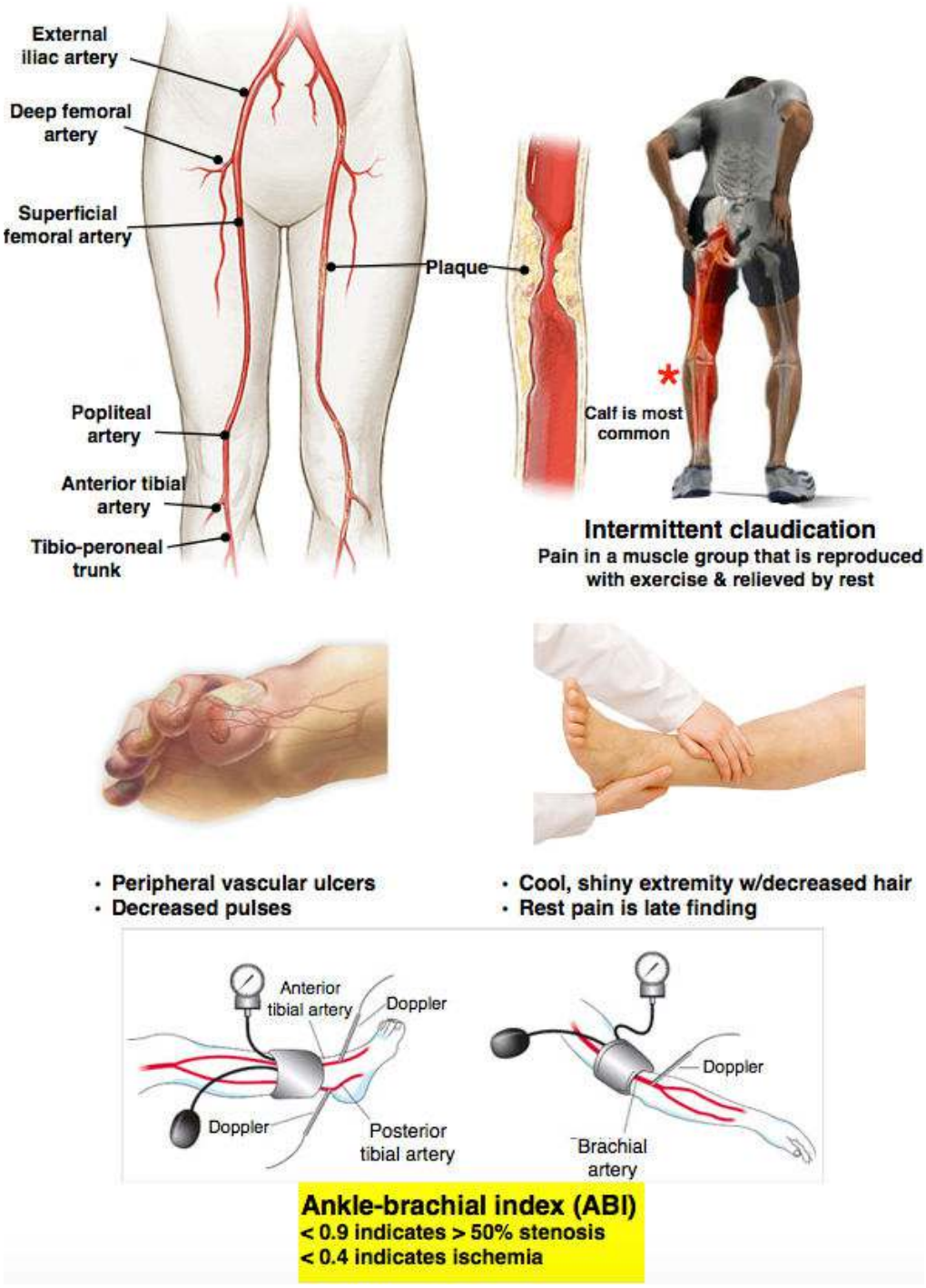
Physical Exam
• Cool extremities with absent or diminished **pulses**

Diagnostic Studies
• **Ankle-brachial index** establishes the diagnosis

Notes


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**Peripheral Artery Disease cont.**





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# XVI. Syncope

### A. Causes of Syncope

| Syncope | | |
|---|---|---|
| **Reflex (neurally mediated)** | **Vasovagal**<br>• Mediated by orthostatic or emotional stress | **Situational**<br>• Cough, sneeze, micturition, post-exercise, post-prandial |
| | **Carotid sinus** | **Unknown** |
| **Orthostatic** | **Primary autonomic**<br>• Pure autonomic, Parkinson's, Lewy body dementia | **Secondary autonomic**<br>• Diabetes, amyloidosis, uremia, spinal cord injuries |
| | **Drug induced**<br>• Alcohol, vasodilators, diuretics, phenothiazines, antidepressants | **Volume depletion**<br>• Hemorrhage diarrhea, vomiting |
| **Cardiac** | **Bradydysrhythmia**<br>• AV block, tachy-brady syndrome | **Tachdysrhythmia**<br>• Supraventricular tachycardia, ventricular tachycardia |
| | **Structural heart disease**<br>• Drug-induced, valvular disease, ischemia, cardiomyopathy, tamponade | **Other**<br>• Pulmonary embolism, aortic dissection, pulmonary hypertension |

Notes


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### B. High-Risk Factors of Syncope

| Syncope | |
|---|---|
| **Term** | **Risk Factors** |
| **Short-Term Risk Factors (≤ 30 days)** | ***History: Outpatient clinic or ED evaluation***<br>• Male sex<br>• Older age (> 60 years)<br>• No prodrome<br>• Palpitations preceding loss of consciousness<br>• Exertional syncope<br>• Structural heart disease<br>• Heart failure<br>• Cerebrovascular disease<br>• Family history of sudden cardiac death<br>• Trauma |
| | ***Physical exam or laboratory investigation***<br>• Evidence of bleeding<br>• Persistent abnormal vital signs<br>• Abnormal ECG<br>• Positive troponin |
| **Long-Term Risk Factors ( > 30 days)** | ***History: Outpatient clinic or ED evaluation***<br>• Male sex<br>• Older age (> 60 years)<br>• Absence of nausea/vomiting preceding event<br>• Ventricular dysrhythmia<br>• Cancer<br>• Structural heart disease<br>• Heart failure<br>• Cerebrovascular disease<br>• Diabetes mellitus<br>• High CHADS-2 score |
| | ***Physical exam or laboratory investigation***<br>• Abnormal ECG<br>• Lower GFR |

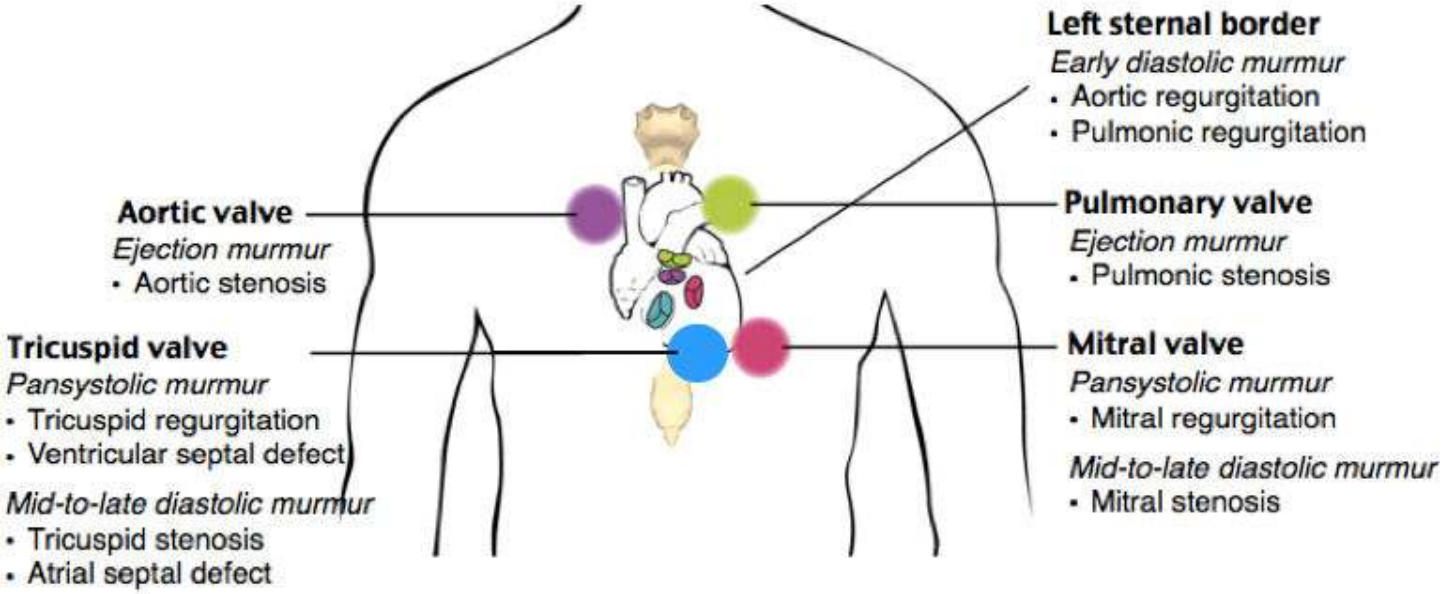
Notes


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# XVII. Valvular Disease

AORTIC STENOSIS • AORTIC REGURGITATION • MITRAL STENOSIS • MITRAL REGURGITATION

## A. Cardiac Murmurs




| Valve | Side Best Auscultated | Location |
|---|---|---|
| **Aortic valve** | Right | 2nd intercostal space, just lateral to the sternum |
| **Pulmonic valve** | Left | 2nd intercostal space, just lateral to the sternum |
| **Tricuspid valve** | Left | 4th-5th intercostal space, over left sternal border |
| **Mitral valve** | Left | 5th intercostal space, midclavicular line (Apex) |

Notes


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**B. Aortic Valve Stenosis**

Pathophysiology
• **Most commonly** caused by degenerative calcification

Patient
• Elderly with a history of diabetes, HTN

Presentation
• **Dyspnea**, **chest pain**, **syncope**

Physical Exam
• **Crescendo-decrescendo** systolic murmur r**adiating to the carotids** which **decreases** with Valsalva
• Paradoxically **split S2**
• **S4 gallop**

Management
• Aortic valve replacement

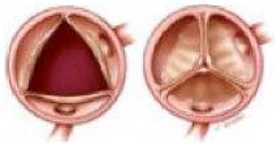


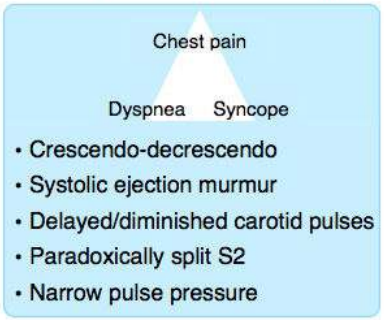
Aortic Valve Stenosis



Healthy Aortic Valve




Notes


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### C. Aortic Insufficiency (Aortic Regurgitation)

Pathophysiology
• **Most commonly** caused by abnormal leaflets or a proximal aortic root

Patient
• History of infectious endocarditis, aortic dissection, **Marfan syndrome**

Presentation
• CHF symptoms: orthopnea, dyspnea, paroxysmal nocturnal dyspnea, fatigue

Physical Exam
• Wide pulse pressure
• Bounding **"water hammer"** peripheral pulses
• Head bobbing with systole (**de Musset sign**)
• Prominent nail pulsations (**Quincke pulse**)
• Hyperdynamic apical pulse displaced to the left
• **Diastolic blowing murmur**, best heard over left sternal border
• Systolic or diastolic thrill or murmur heard over the femoral arteries (**Duroziez sign**)
• In severe AR, a mid-diastolic murmur (**Austin-Flint murmur**)

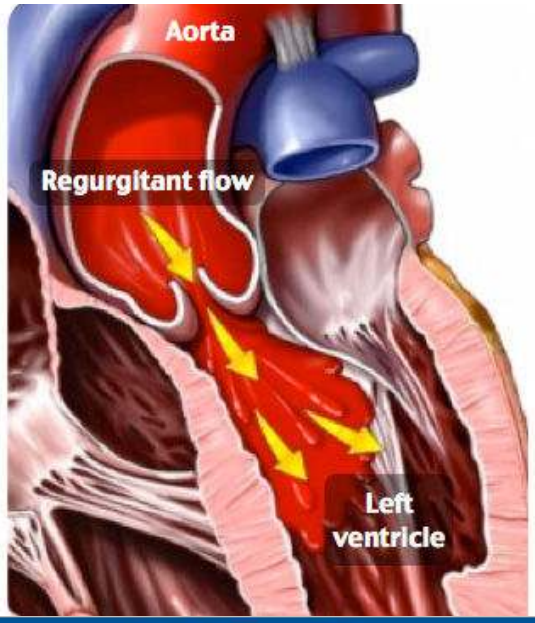
Diagnostic Studies
• Echocardiogram determines the severity of regurgitation

Management
• Diuretics, digoxin, ACEI, salt restriction (if CHF present)
• Surgery indicated if symptomatic, EF < 55%, or end-diastolic dimension > 55 mm

Notes


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**Aortic Insufficiency (Aortic Regurgitation) cont.**




| Aortic Regurgitation | | |
|---|---|---|
| | **Acute** | **Chronic** |
| **Causes** | • Endocarditis<br>• Aortic dissection<br>• Trauma | • Rheumatic heart disease<br>• Congenital (bicuspid) valve<br>• Prior endocarditis |
| **Clinical** | • Severe dyspnea<br>• Pulmonary edema<br>• Acute heart failure | • Exertional dyspnea<br>• Fatigue<br>• Heart failure |
| **Pulse pressure** | • Normal | • Wide |
| **Murmur/Heart sound** | • Short, diastolic mumur, S3 | • High-pitched decrescendo diastolic blowing murmur best heard along left sternal border |

Notes


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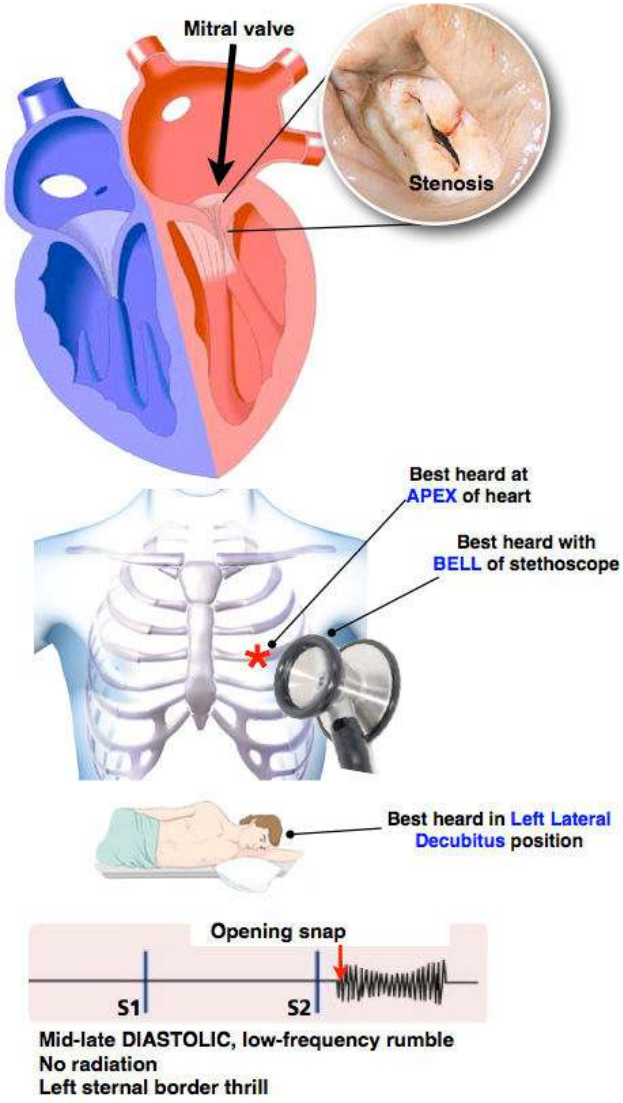
### D. Mitral Stenosis

Pathophysiology
• **Most commonly** caused by **rheumatic heart disease**

Presentation
• Exertional dyspnea, hemoptysis

Physical Exam
• **Loud S1**, **opening snap**, **low-pitched and rumbling diastolic apical murmur**

Comments
• Antibiotic prophylaxis if undergoing procedures prone to bacteremia




Notes


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### E. Mitral Regurgitation

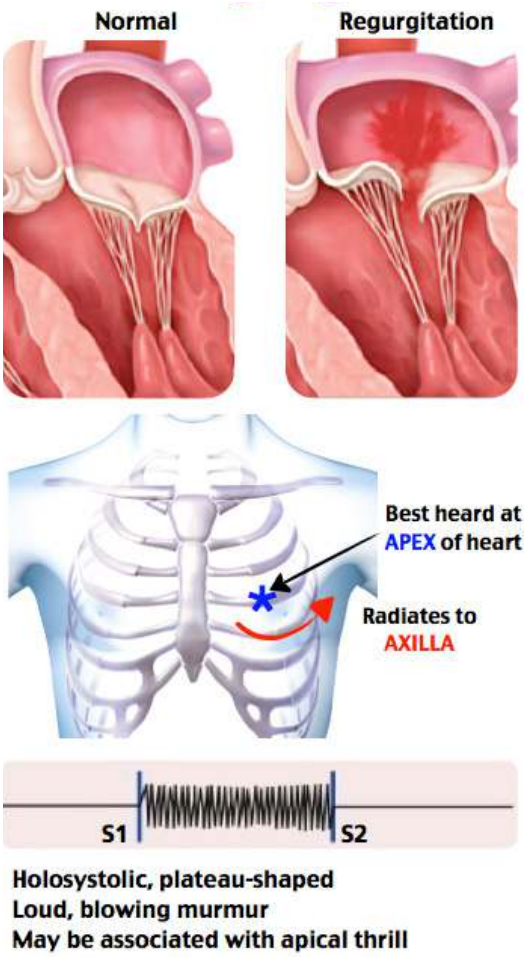
Patient
• History of ischemic heart disease, endocarditis, MI, trauma

Presentation
• Dyspnea

Physical Exam
• **Blowing holosystolic murmur** best heard at the **apex** with **radiation to the axilla**
• Signs of cardiogenic shock if acute

Diagnostic Studies
• Echocardiogram is diagnostic

Management
• Nitroprusside, dobutamine, intra-aortic balloon pump, emergency surgery




Notes


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# XVIII. Vascular Disease

AORTIC ANEURYSM/DISSECTION • ARTERIAL OCCLUSION/THROMBOSIS • PHLEBITIS

### A. Abdominal Aortic Aneurysm (AAA)

Patient
• Older man with a history of **smoking** and HTN

Presentation
• Abdominal/flank pain, or asymptomatic

Physical Exam
• Hypotension and **pulsatile abdominal mass**

Diagnostic Studies
• **Ultrasound** as a screening tool
• **CT** to monitor progression

Management
• > 4 cm: screen every six months
• > 5.5 cm: surgical intervention

Comments
• The **USPSTF** recommends one-time screening via ultrasonography in men aged **65–75** who have **ever** smoked

Notes


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**Abdominal Aortic Aneurysm (AAA) cont.**







| AAA Risk Factors |
| --- |
| Advanced age (> 60 years) |
| Male gender |
| Caucasian race |
| Family history |
| Smoking |
| Presence of other large vessel aneurysms |
| Atherosclerotic risk factors |

**AAA Triad**

1. Abdominal pain
2. Hypotension
3. Pulsatile abdominal mass

Notes


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## B. Aortic Dissection

Patient
• Older, with a history of HTN, smoking, Marfan syndrome

Presentation
• Sudden **"ripping"** or **"tearing"** chest pain radiating to back

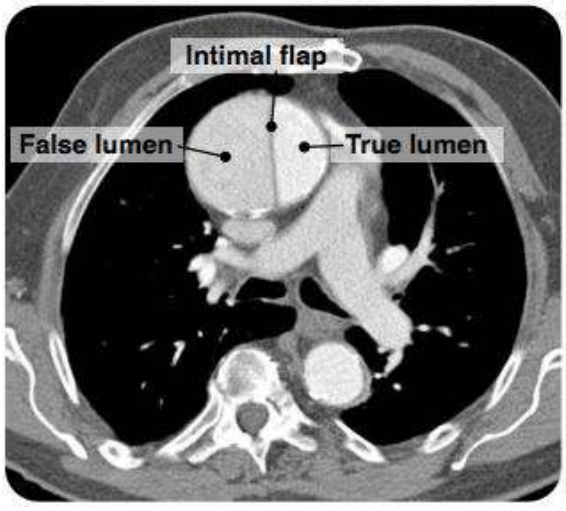
Physical Exam
• Asymmetric pulses/BP

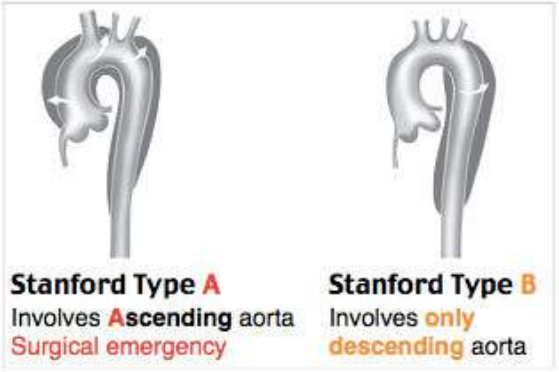
Diagnostic Studies
• Chest X-ray will show a **widened mediastinum**
• CT or transesophageal echocardiogram establishes diagnosis

Management
• Focused on **reducing BP and HR (beta-blockers)**
• Consider **surgical correction** depending on location of dissection

**Management**
*Anti-impulse therapy*
• Reduce BP to lowest tolerable level (SBPs 100 200 mm Hg
• Reduce HR < 60 bpm
• Intravenous beta-blockers (esmolol, labetalol, propranolol)
• Nitroprusside (only after HR is controlled)
• Pain control

Notes





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### C. Thromboembolism

Pathophysiology
• **Most common** source of thrombus formation is the left heart

Patient
• History of recent MI or atrial fibrillation

Presentation
• Sudden onset of **paresthesias**, **pallor**, **pulselessness**, **poikilothermia**, **paralysis**, **and pain out of proportion to exam (6 Ps)**

Comments
• **Most common** site is **femoral artery bifurcation**

### D. The 6 Ps of Acute Arterial Occlusion

1. Paresthesia
2. Pallor
3. Pulselessness
4. Poikilothermia
5. Paralysis
6. Pain out of proportion to exam

Notes


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## E. Superficial Thrombophlebitis

Patient
• History of varicose veins, pregnancy, or catheter placement

Physical Exam
• Induration, redness, and **tenderness along the course of a palpable vein**

Diagnostic Studies
• Diagnosis is made **clinically**
• Doppler ultrasound used to **rule out DVT**

Management
• Warm, moist compresses
• **NSAIDs** for pain

**Diagnosis**
• Clinical
• Ultrasonography

### Superficial Thrombophlebitis

**Pain and inflammation of a vein (in the absence of thrombus)**




**Management**
• Extremity elevation
• Warm or cool compresses
• NSAIDs
• Compression therapy
• Remain ambulatory

| Condition | Description |
|---|---|
| **Superficial phlebitis** | The presence of pain and inflammation involving a vein in the absence of thrombus |
| **Superficial thrombophlebitis** | The more frequent use of duplex ultrasound has allowed the distinction of thrombophlebitis from phlebitis by confirming the presence or absence of thrombus within a vein |
| **Superficial vein thrombosis** | The recognition that thrombosis of the axial veins (e.g., great saphenous vein, small saphenous vein) can lead to thromboembolism, particularly when the more proximal vein is affected, has led to the use of the term "superficial vein thrombosis" |

Notes


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CHAPTER 2

# Orthopedics/ Rheumatology

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CHAPTER 2

# Orthopedics/Rheumatology


68 **BACK STRAIN/SPRAIN/LOW BACK PAIN**

72 **BURSITIS/TENDONITIS**

75 **CAUDA EQUINA**

77 **COSTOCHONDRITIS**

78 **FRACTURES/DISLOCATIONS**
Shoulder
Forearm/wrist/hand
Hip
Knee
Ankle/foot

117 **GOUT**

121 **HERNIATED DISK**

122 **OSTEOMYELITIS**

123 **SEPTIC ARTHRITIS**



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# I. Back Strain/Sprain/Low Back Pain

### A. Back Strain/Sprain

Pathophysiology
• Caused by an injury to the **paravertebral spinal muscles**

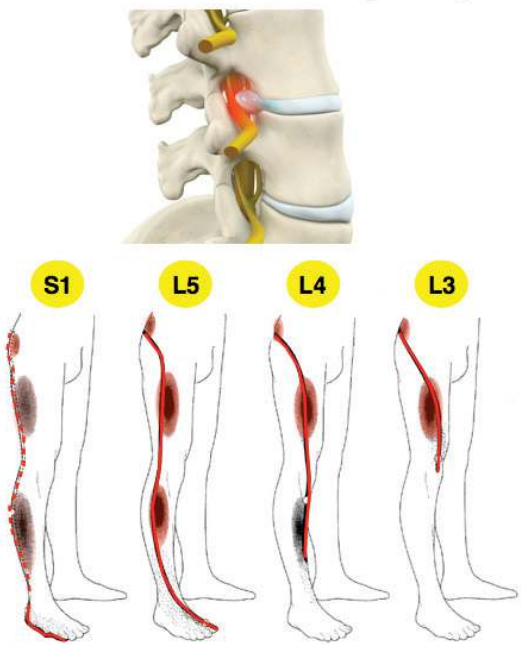
Patient
• History of repeated **lifting** and **twisting**

Physical Exam
• Diffuse tenderness in the lower back or SI region with decreased ROM, especially flexion

Diagnostic Studies
• Imaging is indicated only if **"red flags"** are present: pain at rest, pain at night, weight loss, acute bony tenderness, or significant trauma

Management
• **Conservative management** with continued activity as tolerated, NSAIDs, physical therapy (avoid opioids)

### B. Lumbar Radiculopathy




Notes


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### C. Pharmacologic Therapy for Acute Low Back Pain

| | |
|---|---|
| **Initial Monotherapy** *(2 to 4 weeks)* | • NSAIDS<br>• Acetaminophen (if NSAID contraindication) |
| **Second-line Combination Therapy** | Combination with muscle relaxants<br>• Cyclobenzaprine<br>• Diazepam<br>• Methocarbamol<br>• Carisoprodol |
| **Refractory or Severe Pain** | • Opioids<br>• Tramadol |
| **Other** | • Antidepressants<br>• Glucocorticoids<br>• Antiepileptics |
| **Adjunctive** | • Exercise<br>• Physical therapy<br>• Spinal manipulation<br>• Acupuncture |

### D. Nonpharmacologic Therapies for Treatment of Acute Low Back Pain

• Heat (may reduce pain and disability)

• Massage (no evidence for clinical benefit, but may increase patient satisfaction)

• Acupuncture (inconsistent results for clinical benefit)

• Spinal manipulation (may provide modest improvement in pain and function)

• Exercise and physical therapy (no clear clinical benefit)

Notes


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### E. Red Flags of Back Pain

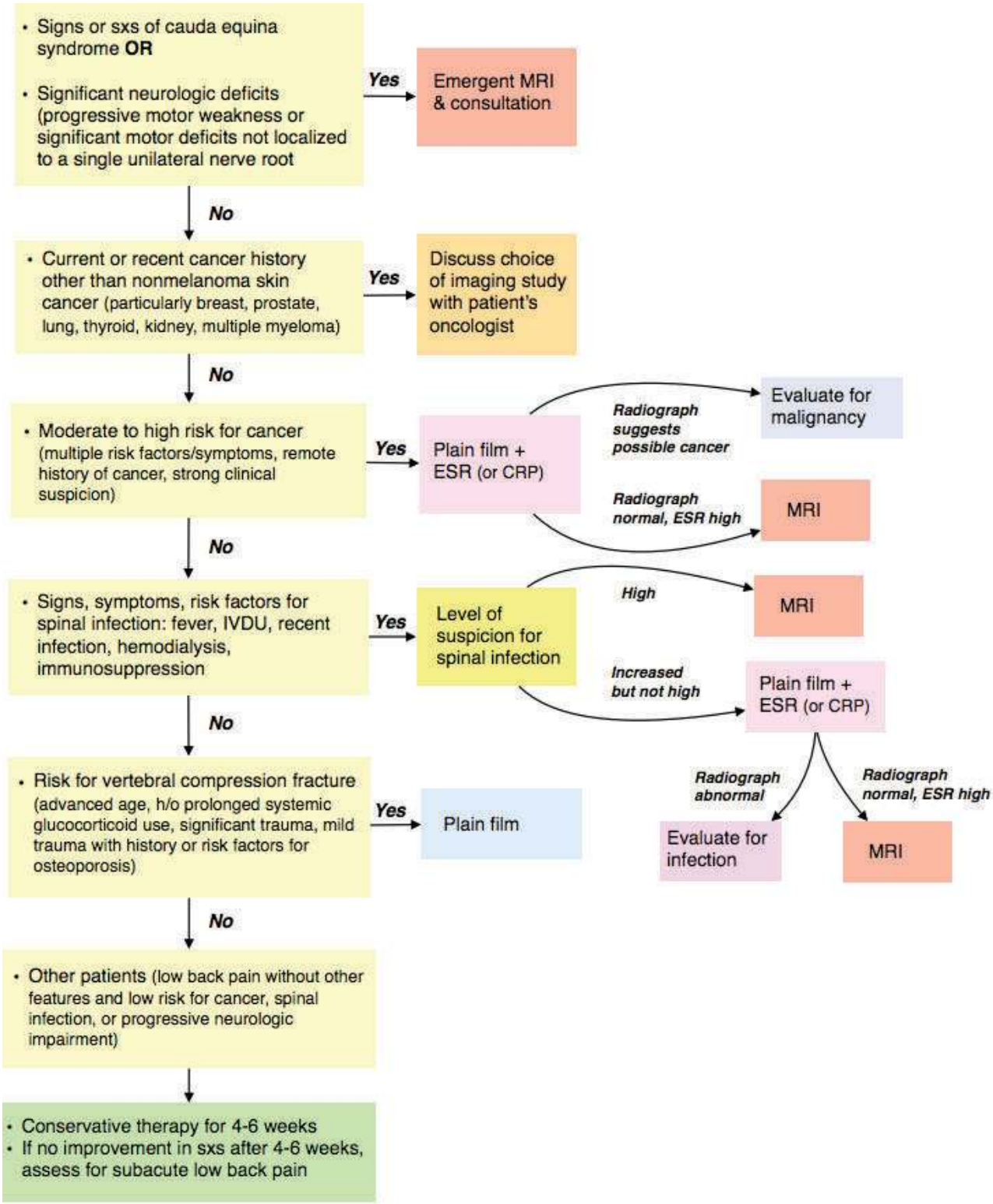


1. Night pain and weight loss **(Tumor)**
2. Fevers, chills, and sweats **(Bone or disk infection)**
3. Acute bony tenderness **(Fracture)**
4. Morning stiffness > 30 min in young adult **(Seronegative spondyloarthropathy)**
5. Urinary/bowel retention, saddle anesthesia **(Cauda equina)**
6. Recent spinal instrumentation **(Spinal epidural abscess or hematoma)**
7. Immunodeficiency **(Bone or disk infection)**
8. Extremes of age
9. Coagulopathy **(Spinal epidural hematoma)**

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## F. Imaging Evaluation for Acute Low Back Pain





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# II. Bursitis/Tendonitis

### A. Prepatellar Bursitis

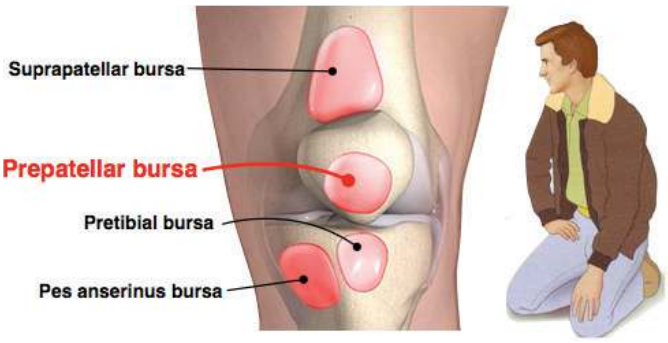
Pathophysiology
• **Most commonly** caused by **chronic kneeling** (housemaid's knee)

Physical Exam
• **Dome-shaped swelling** over the anterior aspect of the knee

Diagnostic Studies
• Aspiration of the bursa identifies inflammatory, hemorrhagic, or septic etiology

Management
• NSAIDs, ice, activity modifications, steroid injection

Comments
• **Most common** bursitis of the knee




**Due to recurrent trauma**
Seen in individuals who frequently kneel

**Bursitis**: Prefer to lie with knee fully **extended**
**Joint effusion**: Prefer knee **flexed**



**Aspiration**
1. Rule out infection
2. Diagnose microcrystalline disorder

**Management**
• Joint protection
• NSAIDs

Notes


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## B. Olecranon Bursitis

Pathophysiology
• Trauma, arthritis, hemorrhage, sepsis

Presentation
• Elbow pain and swelling

Physical Exam
• Discrete area of **soft swelling** directly overlying the olecranon process
• **Decreased active** ROM, **normal passive** ROM

Management
• NSAIDs, ice, activity modifications, steroid injection




Located **posteriorly** over the **olecranon process** of the **ulna**



**Normal passive range of motion**
(Joint effusion will cause pain with full extension)

Notes

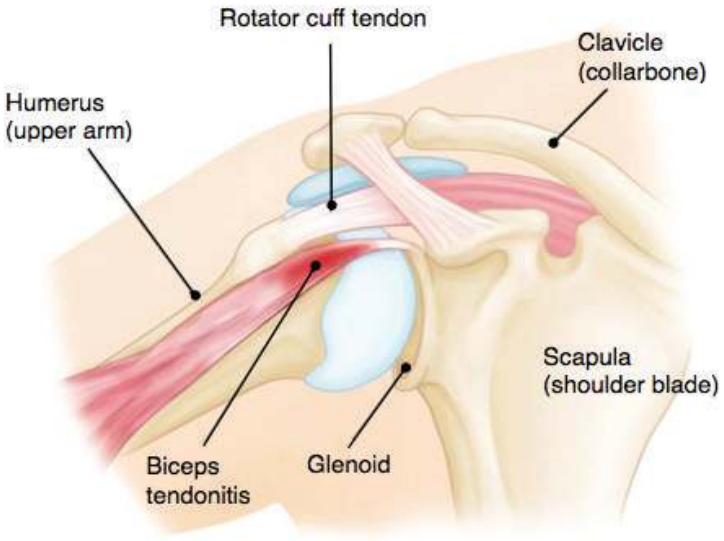



**Causes**
• Trauma
• Hemorrhage
• Sepsis
• Inflammatory arthritis

**Management**
• Joint protection
• NSAIDs


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### C. Biceps Tendinopathy




Patient
• Athlete, 18 – 35 years of age

Presentation
• Anterior shoulder pain with distal radiation over the biceps muscle that worsens with lifting, pulling, or performing repetitive overhead activities

Physical Exam
• **Point tenderness** of the **biceps tendon** has been regarded as pathognomonic
• **Speed test**
  • Arm is fully supinated and extended with the shoulder flexed, the patient is asked to elevate the arm against resistance by the examiner.
  • Elicited anterior shoulder pain is considered a positive Speed's test.
• **Yergason test**
  • Arm is adducted to the patient's side, is pronated, and flexed 90° at the elbow the patient attempts to supinate and externally rotate the arm against resistance by the examiner.
  • Elicited pain at the long biceps tendon is considered a positive test.

Diagnostic Studies
• Aspiration of the bursa identifies inflammatory, hemorrhagic, or septic etiology

Management
• **Rest** and **activity modification** indication considered initial treatment of biceps tendinopathy and usually with a trial of **NSAIDs**
• Physical therapy and ultrasound-guided glucocorticoid injections can be used in cases of long-standing biceps tendinopathy or when symptoms are refractory to initial conservative treatment

Comments
• Biceps tendon rupture is a potential complication; patients can present with a **"popeye"** sign

**Clinical**
• Pain in the anterior shoulder with radiation to biceps
• Pain worse with lifting, pulling, repetitive overhead activities

**Management**
• Rest, NSAIDs
• Glucocorticoid injection

**Complications**
•Tendon rupture

Notes


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# III. Cauda Equina Syndrome

**A. Cauda Equina Syndrome**

Pathophysiology
• Most commonly caused by a **herniated disc** or **IVDU**

Patient
• History of **trauma**, **malignancy**, **epidural abscess** or **hematoma**

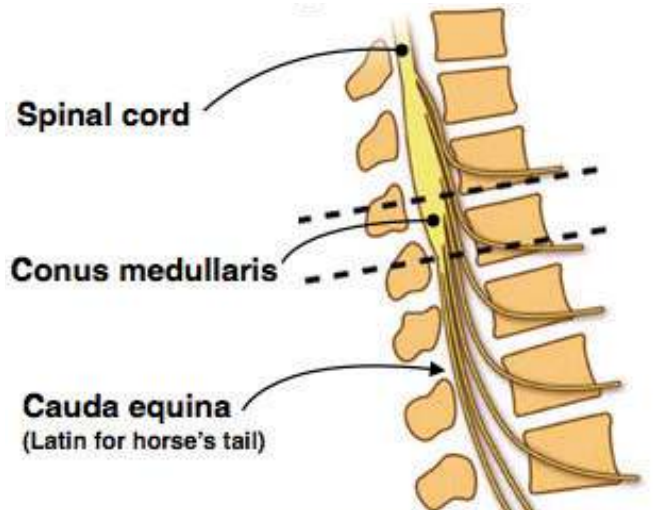
Presentation
• **Acute** onset of **low back pain** with **weakness** and **numbness**

Physical Exam
• **Urinary retention**, **saddle anesthesia**, **decreased rectal tone**

Diagnostic Studies
• **MRI** or **CT myelogram**

Management
• **Emergent** operative decompression

**Etiology**
• Compression of cauda equina by:
• Herniated disk
• Tumor
• Abscess




**Diagnosis**
• Clinical suspicion
• MRI or CT myelogram

**Clinical**
• **Urinary retention** (most sensitive) followed by incontinence
• Post-void residual urine vol > 100 mL
• Bowel dysfunction
• Saddle anesthesia
• Flaccidity of lower extremities (lower motor neuron symptoms)
• Loss of deep tendon reflexes (DTRs)
• Loss of rectal tone

Notes


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**B. Conus Medullaris vs. Cauda Equina Syndromes**

| Characteristic | Conus Medullaris Syndrome | Cauda Equina Syndrome |
|---|---|---|
| **Vertebral level** | • L1 - L2 | • L2 - sacrum |
| **Spinal level** | • Sacral cord segments and roots | • Lumbosacral nerve roots |
| **Presentation** | • Sudden and bilateral | • Gradual and unilateral |
| **Radicular pain** | • Less severe | • More severe |
| **Low back pain** | • More | • Less |
| **Motor strength** | • Symmetrical<br>• Less marked hyperreflexic distal paresis of lower limbs<br>• Fasciculation | • More marked asymmetric areflexic paraplegia<br>• Atrophy more common |
| **Reflexes** | • Ankle jerks affected | • Both knee and ankle jerks affected |
| **Sensory** | • Localized numbness to perianal area<br>• Symmetrical and bilateral | • Localized numbness at saddle area<br>• Asymmetrical<br>• Unilateral |
| **Sphincter dysfunction** | • Early urinary and fecal incontinence | • Tends to present late |
| **Impotence** | • Frequent | • Less frequent |

Notes


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# IV. Costochondritis

### A. Costochondritis

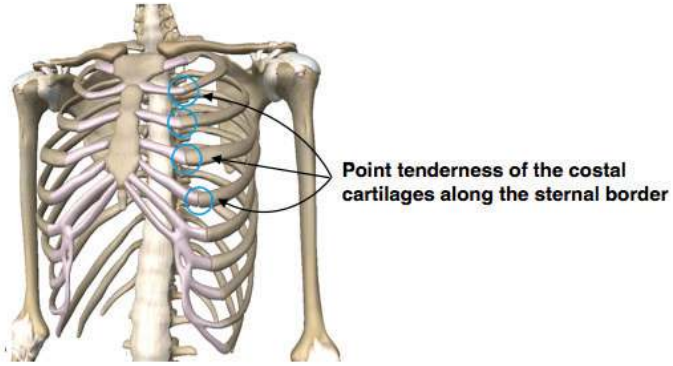
Pathophysiology
• **Atypical chest pain** (noncardiac) originating from the chest wall itself
• Originating from trauma or rheumatologic conditions

Physical Exam
• **Reproducible** costochondral tenderness

Diagnostic Studies
• Rule out life-threatening thoracic pathology

Management
• NSAIDs, ice, PT

Comments
• **Tietze syndrome**: pain with associated swelling




**Clinical**
• Pain is reproducible
• Most common cause of pediatric chest pain
• Carrying school bag on one shoulder may precipitate process

**Management**
• Physical therapy
• NSAIDs
• Corticosteroids (recalcitrant cases)

Notes

### B. Ecchymosis

• Non-raised skin discoloration caused by the escape of blood into the tissues from ruptured blood vessels


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# V. Fractures/Dislocations

SHOULDER • FOREARM/WRIST/HAND • HIP • KNEE • ANKLE/FOOT

## 1. SHOULDER

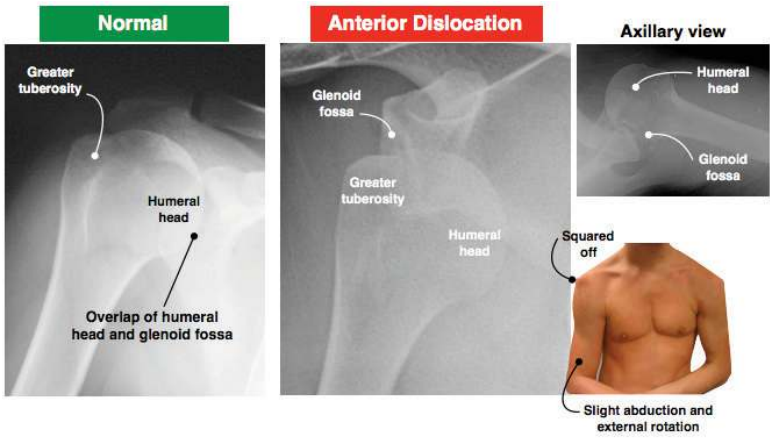
### A. Anterior Shoulder Dislocation

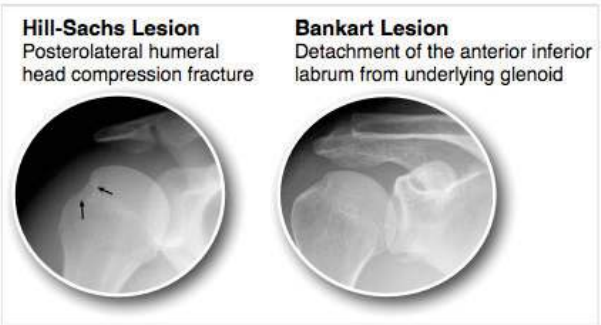
Pathophysiology
• **Most commonly** caused by trauma from a **fall** or **forceful throwing motion**

Physical Exam
• Arm is **abducted**, **externally** rotated
• Positive apprehension test

Diagnostic Studies
• Anteroposterior (AP) and axillary shoulder X-rays will show an **inferiorly** and **medially displaced humeral head**

Comments
• **Most common** type of shoulder dislocation
• Complications include **axillary nerve** damage, **Bankart lesion**, **Hill-Sachs deformity**




Notes

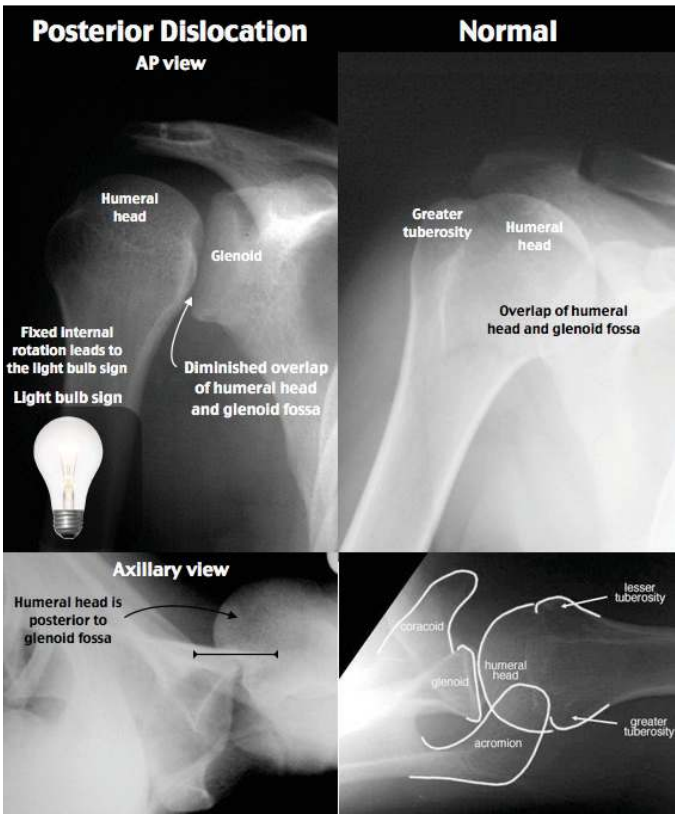

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### B. Posterior Shoulder Dislocation

Pathophysiology
• **Most commonly** caused by **seizure** or **electrical shock** (struck by lightning)

Physical Exam
• Arm will be **adducted** and **internally rotated**

Diagnostic Studies
• AP X-ray will show **"light bulb"** sign




Notes

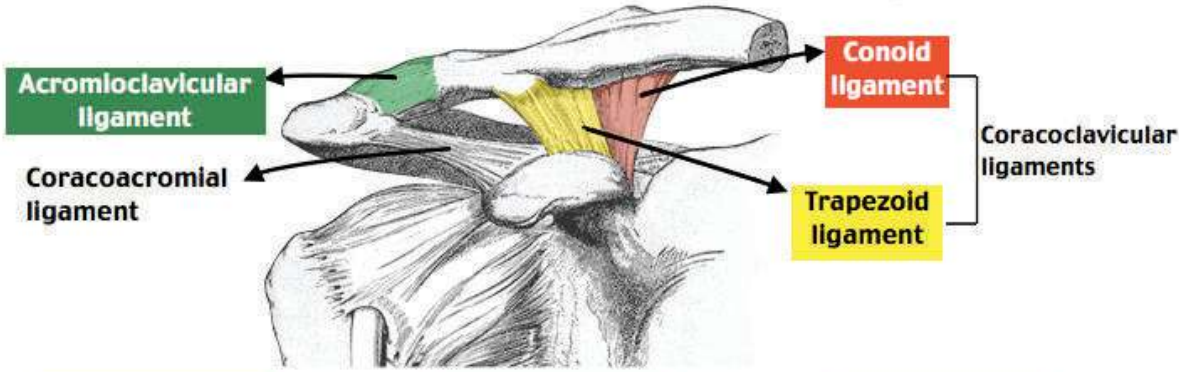

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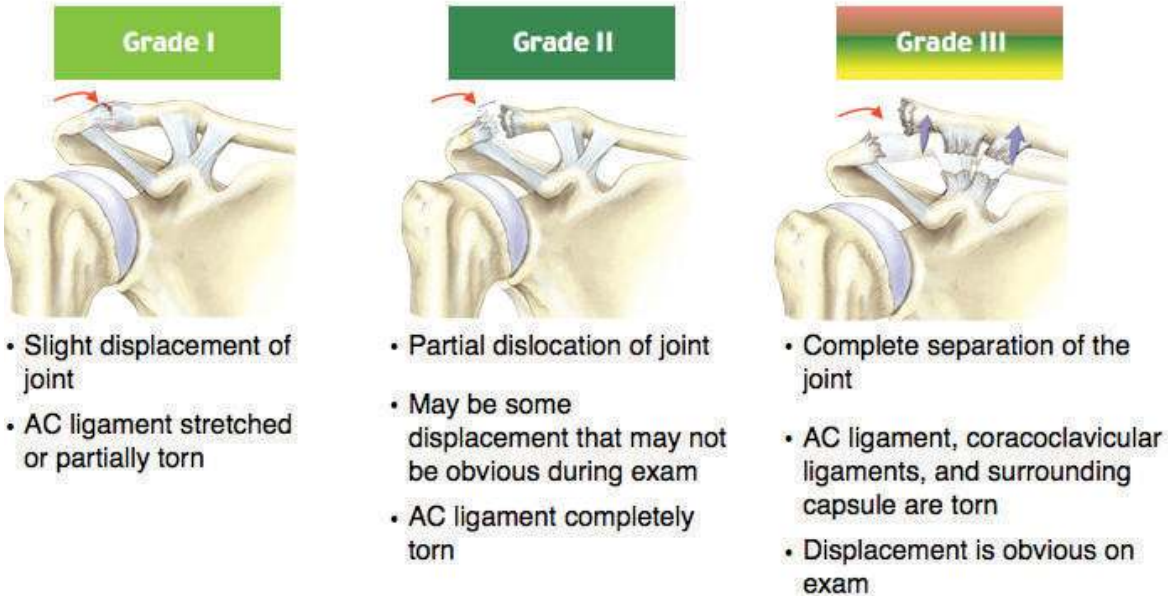
### C. Acromioclavicular (AC) Joint Separation

Pathophysiology
- Grades:
  - I: AC ligament sprain
  - II: AC ligament disruption + coracoclavicular (CC) ligament sprain
  - III: AC and CC disrupted, clavicle displaced
  - IV: clavicle displaced posteriorly
  - V: clavicle displaced superiorly and anteriorly
  - VI: clavicle displaced inferiorly

Diagnostic Studies
- **Bilateral** AP X-rays are diagnostic

Management
- Type I—III: sling, orthopedic referral
- Type IV—VI: sling, orthopedic referral, likely **surgery**




Notes


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### D. Adhesive Capsulitis (Frozen Shoulder)

Presentation
• **Diffuse** shoulder pain, with **progressive stiffness** that results in **severe ROM limitations**

Management
• Physical therapy and intra-articular corticosteroid injections




1. **Initial**: Pain, worse at night, increasing stiffness (2 to 9 months)

2. **Intermediate**: Stiffness and severe loss of motion, less pain (4 to 12 months)

3. **Recovery**: Gradual return of range of motion (5 to 24 months)

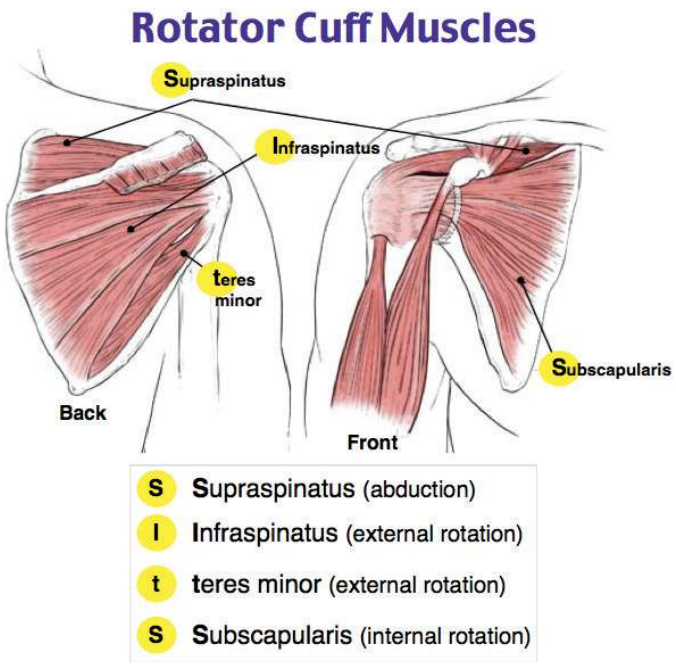
**Risk Factors**
• Diabetes
• Thyroid disease
• Prolonged immobilization
• Stroke
• Autoimmune disease

**Management**
• Physical therapy
• Corticosteroids (oral and intra-articular)

Notes


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**E. Rotator Cuff Impingement and Tear**




Pathophysiology
- Rotator Cuff made up of **SITS** muscles:
  - **S**upraspinatus (abduction)
  - **I**nfraspinatus (external rotation)
  - **T**eres minor (external rotation)
  - **S**ubscapularis (internal rotation)

Patient
- History of **repetitive overhead activity**

Presentation
- Pain with brushing hair or teeth, pain at night when rolling onto shoulder

Physical Exam
- Positive **Neer** and **Hawkins** tests (see next page)

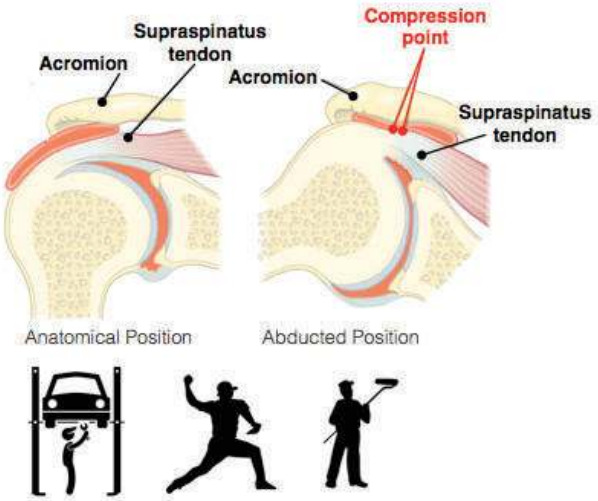
Notes


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## F. Shoulder Impingement Syndrome

↳ Also known as subacromial impingement

**Most common cause of shoulder pain in primary care**




**Risk Factor**
- Repetitive activity at or above the shoulder

**Clinical**
- Pain with overhead activity
- Pain localized to deltoid area or lateral arm
- Often worse at night or when lying on affected shoulder



**Hawkins Kennedy test**
1. Stabilize shoulder
2. Flex elbow 90 degrees
3. Internally rotate shoulder

**Pain with internal rotation = positive test**



**Neer test (Passive painful arc maneuver)**
1. Start with arm lowered
2. Passively flex the glenohumeral joint
3. Prevent shoulder from shrugging

**Pain in anterior, lateral shoulder = positive**

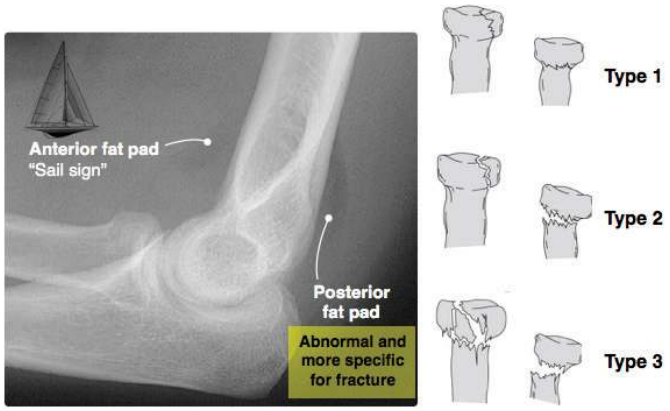
**Management**
- Rest, ice, NSAIDs
- Physical therapy

Notes


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## 2. FOREARM/WRIST/HAND

### A. Radial Head Fracture




Patient
• Adult with a history of a **fall on an outstretched hand**

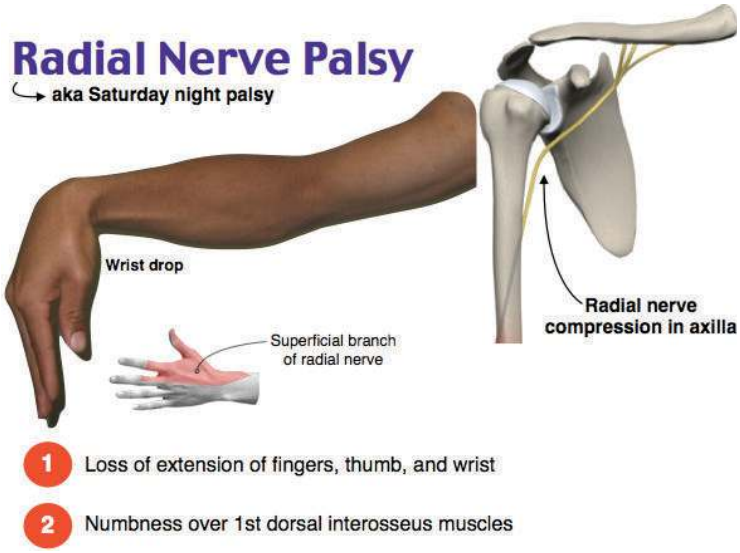
Physical Exam
• Localized swelling, tenderness, and decreased motion

Diagnostic Studies
• X-ray will reveal **fat pad** or **"sail" sign**

Notes


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**B. Radial Nerve Palsy (Saturday Night Palsy)**




Patient
• History of a night of heavy drinking

Physical Exam
• Weakness of the wrist extensors, **wrist drop**, and weakness of the finger extensors

Management
• Wrist splint, physical therapy, and pain management

Comments
• Radial nerve is formed from nerve roots **C6** through **C8**

Notes


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### C. Radial Head Subluxation (Nursemaid Elbow)

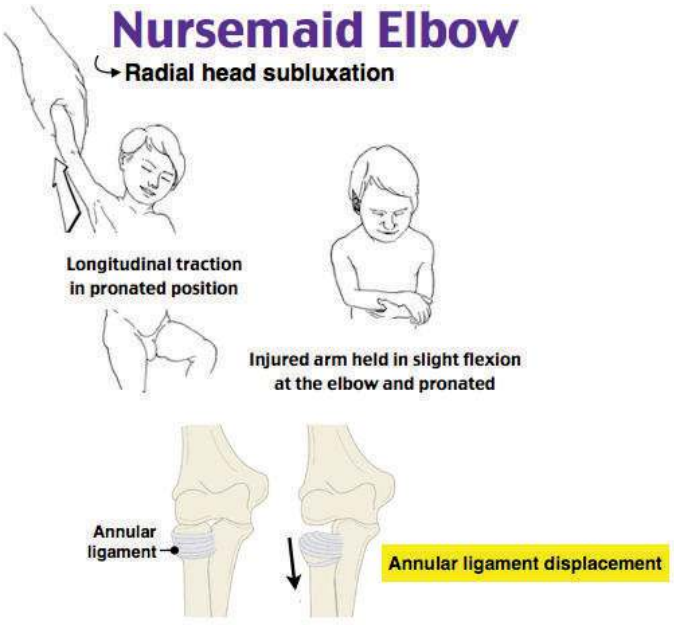
Pathophysiology
• Caused by longitudinal traction on a pronated forearm while the elbow is extended, subluxing the **annular ligament**

Patient
• Child, **1–4 years old** with a history of being pulled up by the wrist

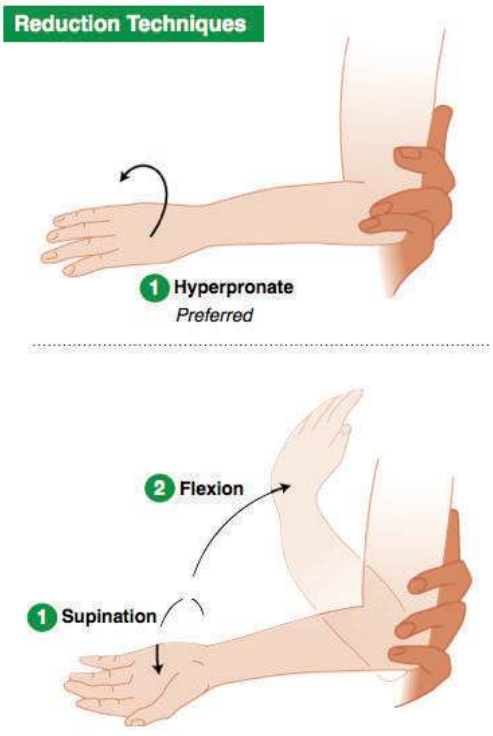
Physical Exam
• Affected arm is held close to the body in a slightly **flexed** and **pronated position**

Management
• **Hyperpronation method**: apply **pressure** to the radial head and **hyper-pronate** the forearm
• **Supination/flexion method**: **supinate** and fully **flex** the elbow while applying **pressure** to the radial head and pulling with gentle traction

Notes




**Diagnosis**
• Clinical
• Radiographs not required



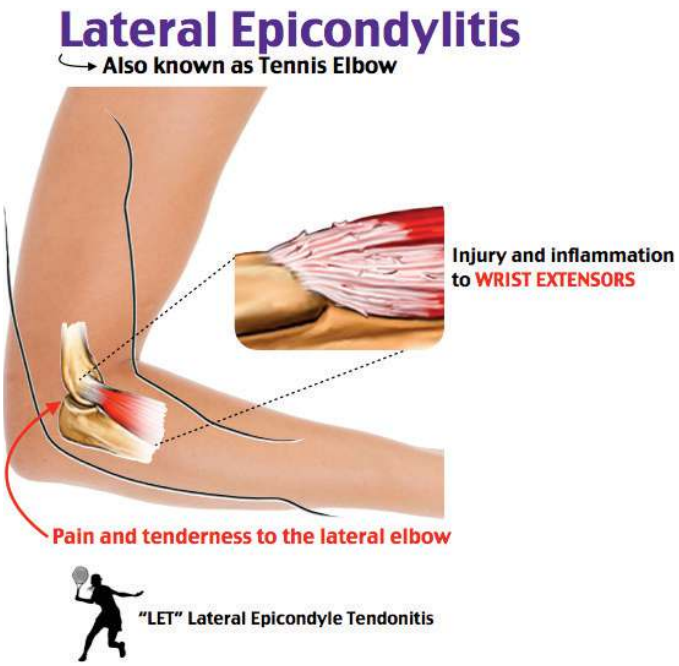


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**D. Lateral Epicondylitis (Tennis Elbow)**

Presentation
• Pain when **lifting** objects, primarily when the arm is **pronated**

Physical Exam
• Tenderness over the proximal **extensor** muscle group
• Pain when elbow is extended, when the forearm is in the pronated position, and while extending the fingers and wrist against resistance




Notes


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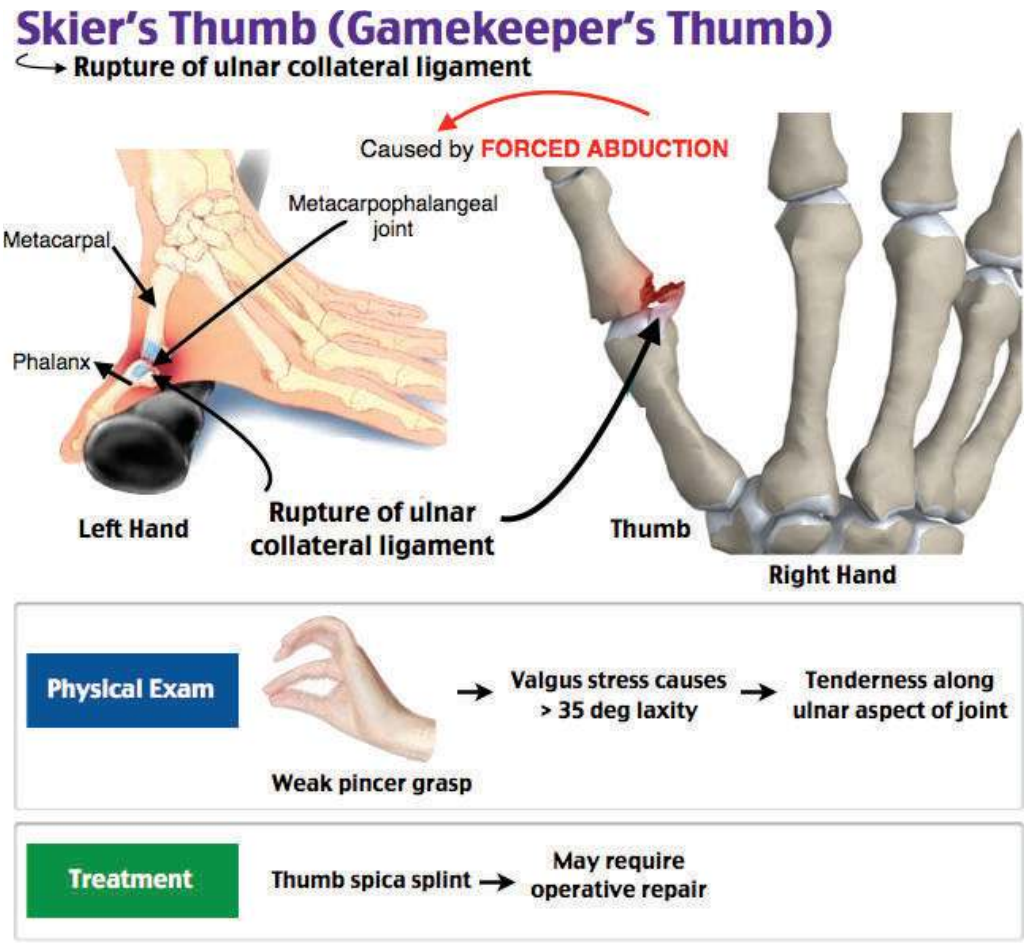
### E. Skier's Thumb (Gamekeeper's Thumb)

Pathophysiology
• Caused by forceful **radial deviation** of the thumb

Patient
• History of skiing

Presentation
• Pain, swelling, and tenderness on the **ulnar** side of the **first MCP joint**

Management
• **Thumb spica splint**




Notes


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**F. Boxer Fracture**

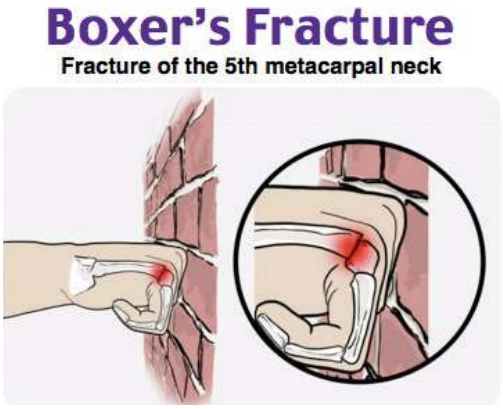
Pathophysiology
• Fracture of the **fifth metacarpal neck**

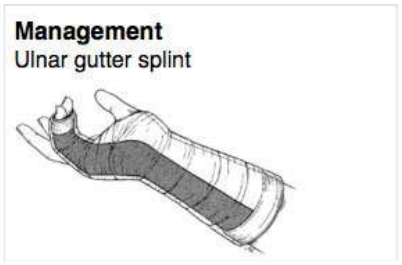
Patient
• History of **punching** an object with a closed fist

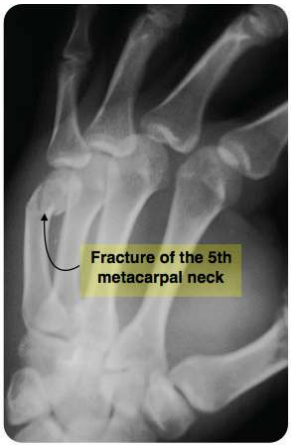
Physical Exam
• Evaluate for "fight bite"

Diagnostic Studies
• X-rays to measure angulation

Management
• **Ulnar gutter splint**
• Hand surgery consult for open fractures, malrotation, neurovascular injury




Notes


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## G. Monteggia and Galeazzi Fractures

Pathophysiology
- **Galeazzi**: **Fracture** of the **distal third** of the **radius** and **dislocation** of the **distal radioulnar joint**
- **Monteggia**: **Fracture** of the **proximal third** of the **ulna** with **dislocation** of the **proximal radial head**

Management
- Immediate ORIF

Comments
- Risk of **anterior interosseous nerve** injury
- Mnemonic: "**GRU**esome **MUR**der"
  - **G**: Galeazzi **R**: radius fracture **U**: ulna dislocation
  - **M**: Monteggia **U**: ulna fracture **R**: radial head dislocation

Notes








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## H. Scaphoid Fracture

Patient
• History of **fall on an outstretched hand**

Presentation
• Dorsal, radial wrist pain with decreased ROM

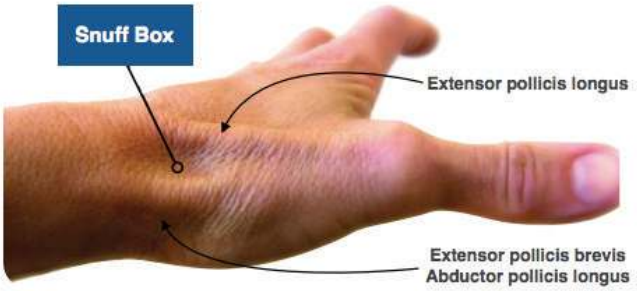
Physical Exam
• **Anatomical snuffbox tenderness**

Management
• **Thumb spica splint**

Comments
• Negative X-ray does not rule out occult fracture
• **Always apply splint if snuffbox tenderness is present**; complications of an untreated fracture include **avascular necrosis** based on unique blood supply to the distal pole of bone



Fall On Outstretched Hand (FOOSH)



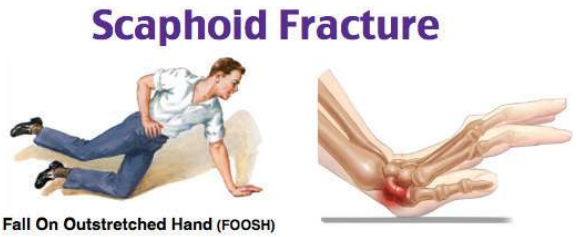

**Snuff box tenderness** = Must rule out **Scaphoid fracture**

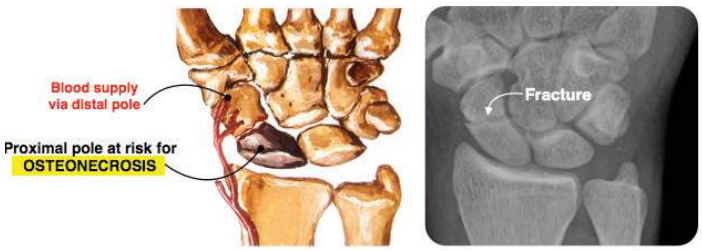
**Management**
If no radiographic evidence of fracture at time of injury...
Apply **thumb spica splint**
**Repeat X-ray (or MRI)** in **10-14 days**



Thumb spica splint

Notes





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## I. Colles/Smith Fracture

Patient
• History of a **fall** onto an **extended wrist**

Presentation
• Pain, swelling over dorsal aspect of wrist

Diagnostic Studies
• Colles: X-ray reveals a **dorsal displacement** and **angulation** of the **distal radius** (**"dinner fork"** deformity)
• Smith: X-ray reveals a **volar displacement** and **angulation** of the **distal radius** (**"garden spade"** deformity)
• Note: displacement always refers to the **distal** fragment

Management
• Reduction, long-arm or sugar-tong splint

Comments
• **Median nerve** at risk
• **Colles is most common** distal radius fracture

Notes


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# Distal Radius Fractures

## Colles' Fracture

**Dorsal** displacement of distal radius fragment








Dinner fork deformity





Extension

## Smith Fracture

**Volar** displacement of distal radius fragment








Garden spade deformity





Flexion

Notes


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3. HIP

## A. Hip Dislocation

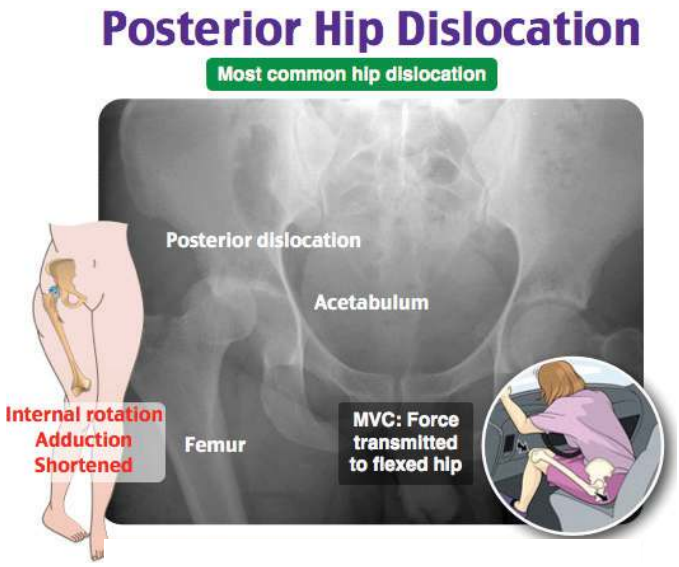
Pathophysiology
• **Most common** type of dislocation is **posterior** based on supporting musculature

Physical Exam
• Posterior dislocation: **internally** rotated; sciatic nerve injury
• Anterior dislocation: **externally** rotated; femoral artery, vein, and nerve injury

**Complications**
• Sciatic nerve injury
  • Loss of sensation in posterior leg and foot
  • Loss of dorsiflexion or plantar flexion
  • Loss of deep tendon reflex at ankle
• Femoral nerve injury
  • Loss of sensation over thigh
  • Weakness of quadriceps
  • Loss of deep tendon reflexes at knee
• Femoral artery injury
  • Hematoma
  • Loss of pulses
  • Pallor

Management
• Emergent reduction under conscious sedation

Comments
• Complications: **avascular necrosis** of the femoral head




Notes

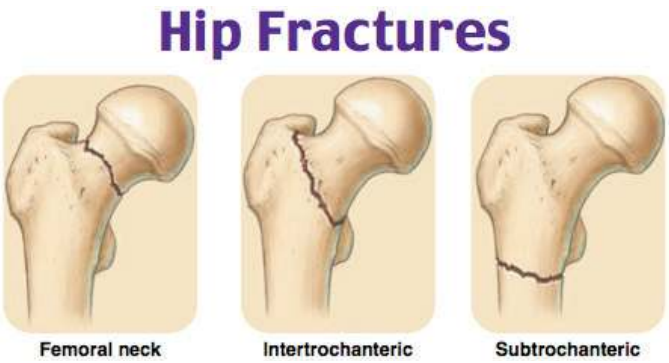

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### B. Hip Fractures: Femoral Neck

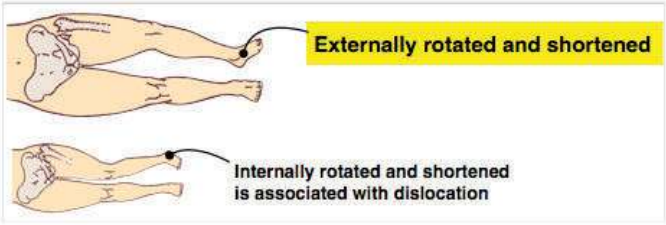
Physical Exam
- **Shortened** and **externally** rotated leg

Diagnostic Studies
- **Gold standard** diagnosis is MRI

Comments
- Negative radiographs do not rule out occult fracture




**Risk factors in elderly**
- Osteoporosis
- Falls




**Management**
- Supportive care
- Orthopedic management

**Complications**
- Infection
- Thromboembolism
- Avascular necrosis
- Nonunion

Notes


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4. KNEE

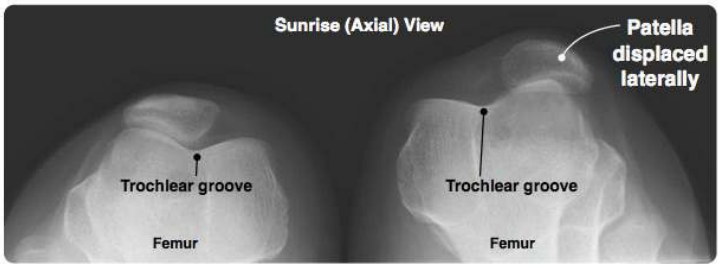
### A. Patellar Dislocation




Pathophysiology
• **Most commonly** caused by **twisting** injury on an **extended** knee
• **Most common** type is **lateral** dislocation

Patient
• Adolescent female, dancer, or athlete

Physical Exam
• Patella displaced over femoral condyle




Diagnostic Studies
• Clinical exam identifies dislocation and X-ray rules out fracture

Management
• **Reduction** and **knee immobilizer**

Notes


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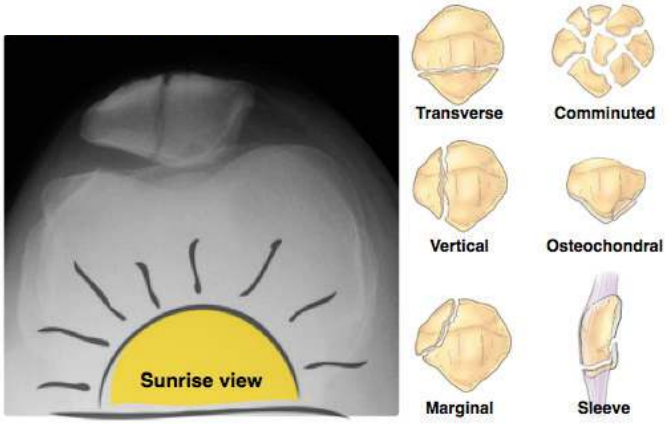
## B. Patella Fracture

Pathophysiology
• **Most commonly** caused by **direct trauma** to the knee
• **Most common** type is **transverse**

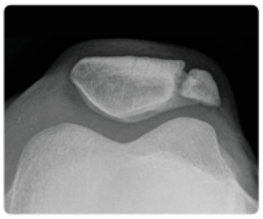
Diagnostic Studies
• X-ray (**sunrise view**) is diagnostic, revealing **sharp** fragment edges

Management
• Surgery if more than 3 mm dislocation or with extensor mechanism loss

Comments
• Compare to **bipartite patella** which has **smooth** fragment edges and is not related to trauma




**Mechanism**
• Direct force (fall onto flexed knee, dashboard strike)
• Indirect force (sudden, forceful contraction of the quadriceps



**Bipartite Patella**
• Both pieces have smooth, well-corticated borders
• Minimal separation between pieces

Notes


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### C. Knee Dislocation

Pathophysiology
• Anterior dislocation (**most common**): **hyperextension** of the knee
• Posterior dislocation: **direct blow** to the **anterior tibia** with the knee flexed

Patient
• History of violent trauma

Physical Exam
•**"Dimple"** sign with **posterolateral** dislocation

Diagnostic Studies
• Clinical exam is diagnostic, X-ray (if needed using **Ottawa Knee Rules**)

Management
• Reduction, knee immobilizer, emergent ortho consult and ABI or duplex post-reduction to evaluate for vascular injury

Comments
• Complications involve **popliteal artery** and **peroneal nerve** involvement

### D. Posterolateral Knee Dislocation



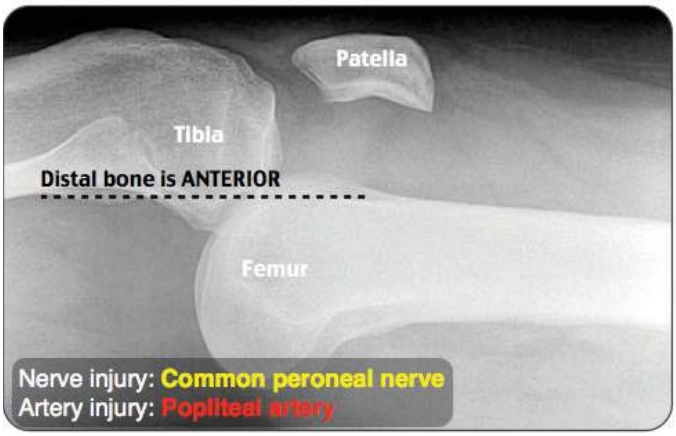

**"Dimple" or "Puker" sign** is pathognomonic

Notes


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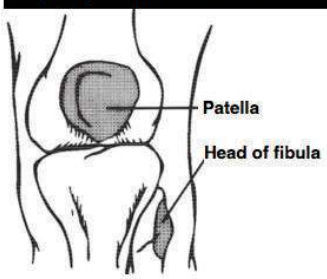
## E. Anterior Knee Dislocation

↳ Most common knee dislocation




## F. Ottawa Knee Rule

**Radiographs of the knee should be obtained after acute injury only for patients who meet one or more of the following criteria**




1. Age ≥ 55 years
2. Isolated tenderness of patella (with no other bony tenderness of the knee)
3. Tenderness at the head of the fibula
4. Inability to flex the knee to 90 degrees
5. Inability to bear weight both immediately and in the emergency department for four steps, regardless of limp (i.e. unable to transfer weight onto each lower limb two times)

Notes


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## G. Meniscus Injuries

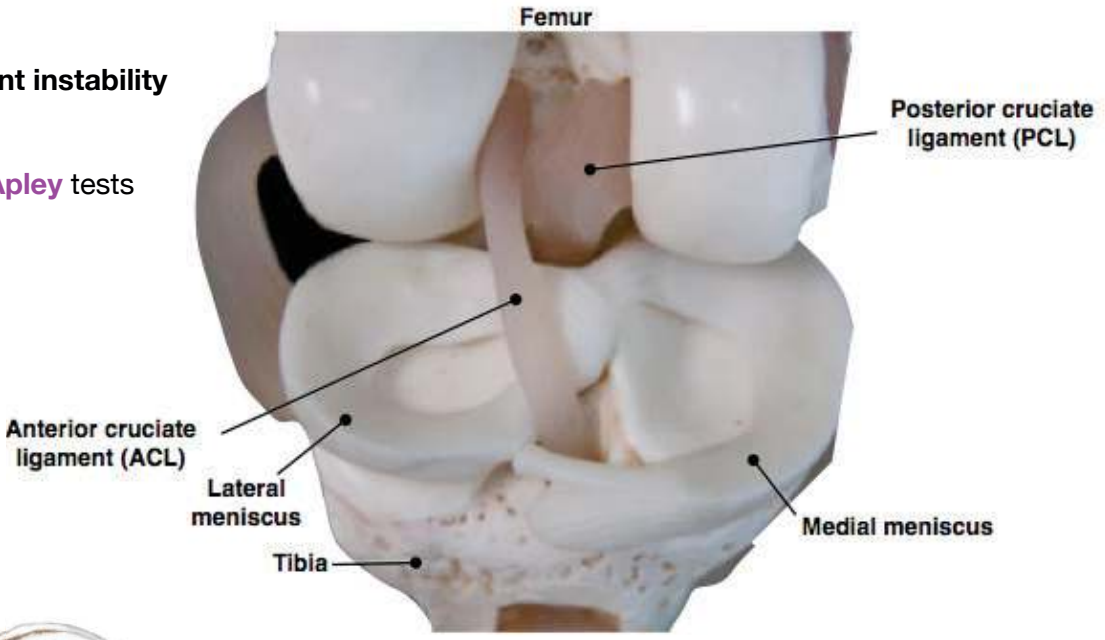
Patient
• History of a **"twist" injury**, rapidly changing directions

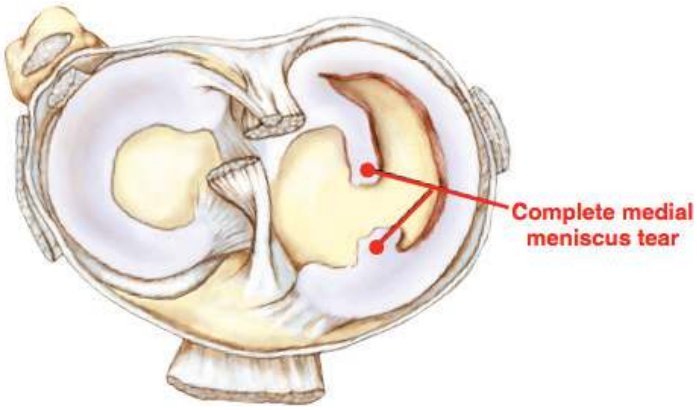
Presentation
• **Knee "locking"** and **joint instability**

Physical Exam
• Positive **McMurray** or **Apley** tests

Diagnostic Studies
• **MRI** confirms diagnosis

Management
• NSAIDs and physical therapy, surgery if severe




Notes


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### H. Medial Collateral Ligament Injury

Pathophysiology
• **Most commonly** caused by a direct blow to the lateral portion of the knee

Presentation
• Hearing or feeling a pop over the **medial knee**

Physical Exam
• Tenderness and swelling to medial knee and ligament

Diagnostic Studies
• History and physical is diagnostic; degree of injury is evaluated by applying **valgus** stress

Management
• Rest, ice, compression and elevation (**RICE**)

### I. Lateral Collateral Ligament Injury

Pathophysiology
• **Most commonly** caused by a direct blow to the medial knee with the knee flexed and foot planted

Physical Exam
• Tenderness, stiffness, and localized swelling
• **Varus** stressing of the knee reveals increased laxity

Management
• Surgical repair

Notes


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# Valgus Stress Test

**Used to determine integrity of the medial collateral ligament (MCL)**





1. Brace the knee by placing one hand along the lateral aspect of the joint
2. With the other hand, hold the ankle and apply a valgus force to the knee, while keeping the ankle in neutral position
3. Perform with the knee at 0 and approximately 30 degrees of flexion

| Grade | |
|---|---|
| 1 | Stretch injury without dissociation (0 to 5 mm opening on valgus stress) |
| 2 | Partial ligament disruption (6 to 10 mm opening on valgus stress) |
| 3 | Complete ligament disruption (> 10 mm opening on valgus stress or lack of any firm endpoint) |

Notes


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# Varus Stress Test

**Used to determine integrity of the lateral collateral ligament (LCL)**





1. Brace the knee by placing one hand along the medial aspect of the joint
2. With the other hand, hold the ankle and apply a varus force to the knee, while keeping the ankle in neutral position
3. Perform with the knee at 0 and approximately 30 degrees of flexion

| Grade | |
|---|---|
| 1 | Stretch injury without dissociation (0 to 5 mm opening on varus stress) |
| 2 | Partial ligament disruption (6 to 10 mm opening on varus stress) |
| 3 | Complete ligament disruption (> 10 mm opening on varus stress or lack of any firm endpoint) |

Notes


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**J. Anterior Cruciate Ligament (ACL) Tear**

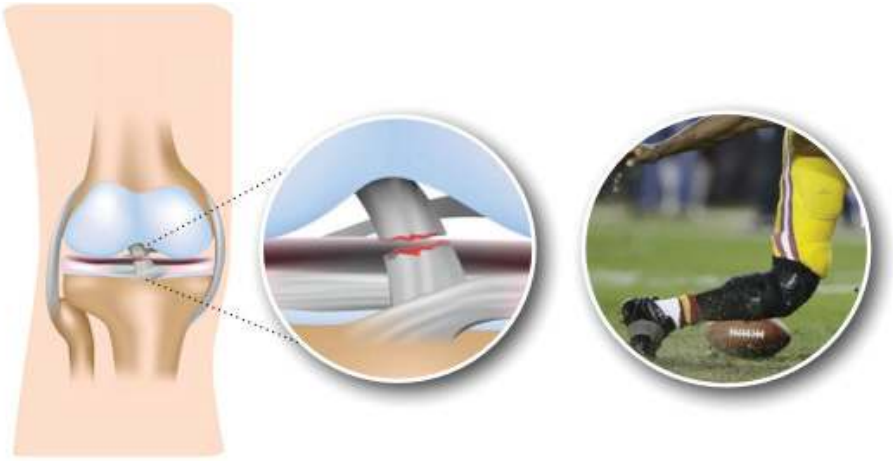
Patient
• History of a **quick stop of movement** and **change of direction** while running

Presentation
• **Hearing a pop** with associated **swelling**

Physical Exam
• Positive **Lachman** test (most **sensitive**) and **anterior drawer test**

Management
• Surgical repair

• **"Pop"** is heard or felt and is **reliable indicator** of tear
• Rapid development of **hemarthrosis** causing significant sweeling
• **Instability** of the knee
• Positive **Lachman** test
• Positive **anterior drawer** test



Notes


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**K. Posterior Cruciate Ligament Injury**

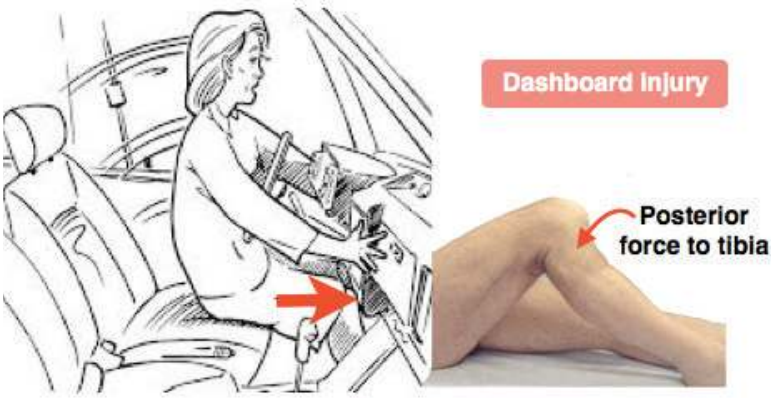
Pathophysiology
• **Most commonly** caused by hyperextending the knee or a direct blow to the anterior proximal tibia

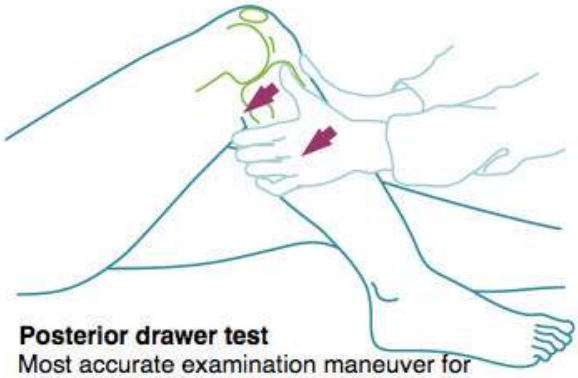
Patient
• History of involvement in a car accident (**dashboard injury**)

Presentation
• Dull aching pain and stiffness

Physical Exam
• Positive **posterior drawer test**

Management
• Conservative management with physical therapy, consider surgical intervention



**Posterior drawer test**
Most accurate examination maneuver for detecting PCL ligament injury

Notes


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## 5. ANKLE/FOOT

### A. Ankle Sprain

Pathophysiology
• **Most commonly** injured **anterior talofibular ligament** (ATFL)

Patient
• History of **ankle inversion**

Presentation
• **Tenderness** and **swelling** to lateral aspect of ankle

Diagnostic Studies
• **Ottawa Ankle Rules** determine imaging protocol

Management
• Protection, rest, ice, compression, elevation (**PRICE**)

### B. Lateral Ankle Sprain

**Immediate Therapy**

1. Limit inflammation and swelling
2. Maintain range of motion




**Protect**
Functional bracing

**Rest**
Limiting weightbearing
Patients use crutches until able to walk with normal gait
Early mobilization

**Ice (cryotherapy) or cold water**
15 to 20 minutes every 2 to 3 hrs for first 48 hrs or until swelling is improved

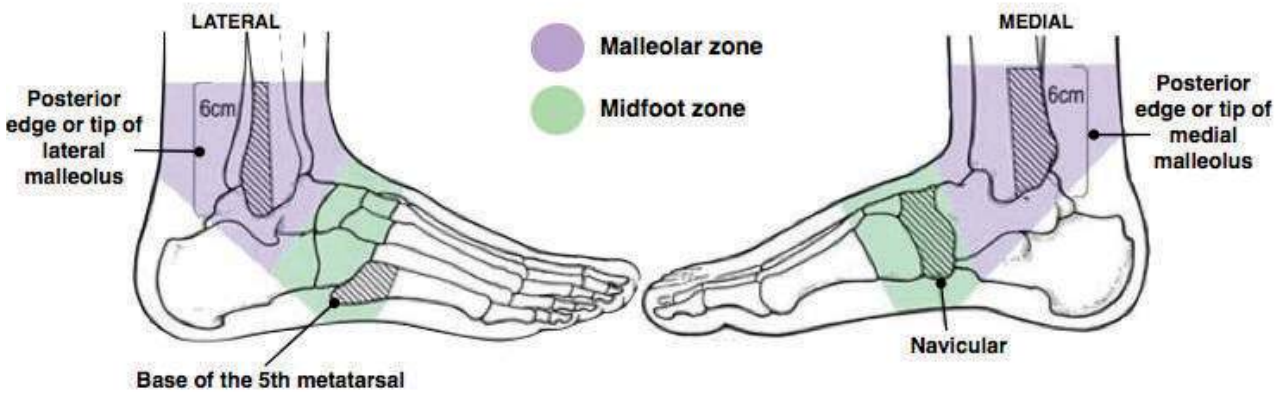
**Compression**
Elastic bandage to minimize swelling

**Elevation**
Keep elevated above the level of the heart to further alleviate swelling

Notes


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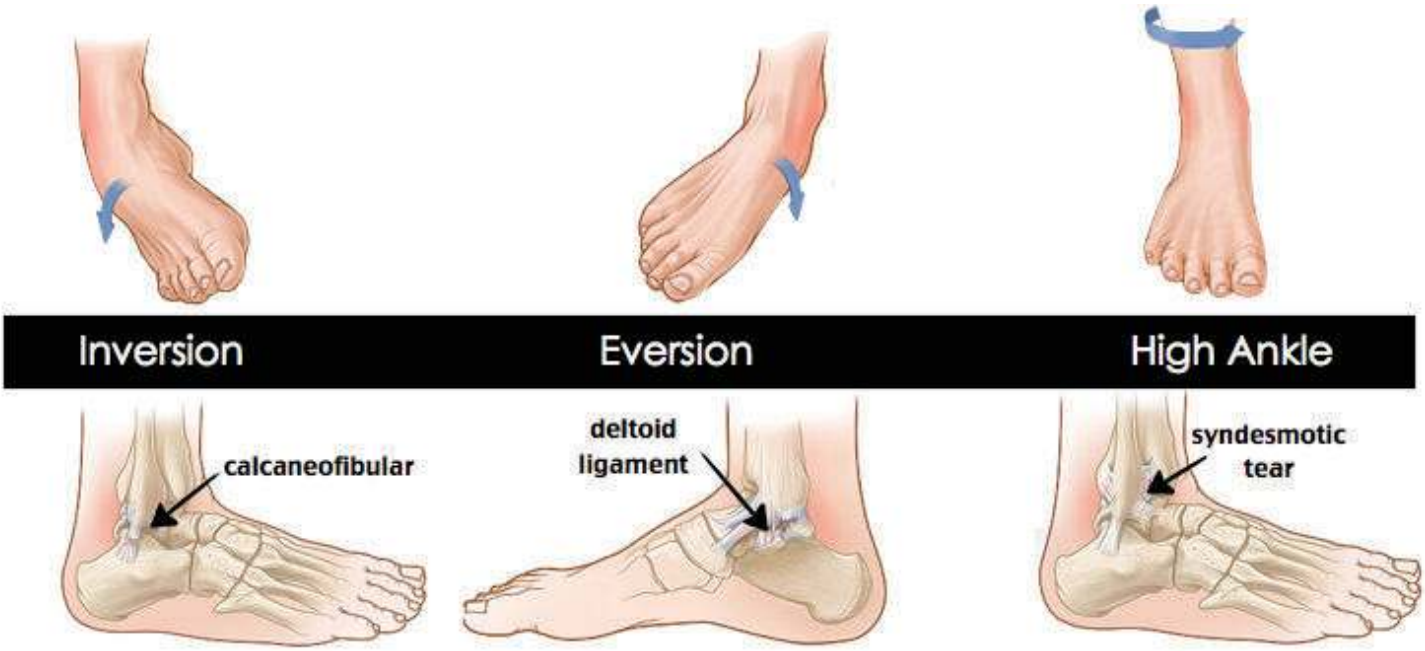
## C. Ottawa Ankle Rules




| An ANKLE radiograph should be performed if there is pain in the malleolar region with any of the following: |
|---|
| Bone tenderness at the posterior edge of the distal 6 cm or the tip of the lateral malleolus |
| Bone tenderness at the posterior edge of the distal 6 cm or the tip of the medial malleolus |
| Inability to bear weight for at least 4 steps both immediately after injury and at the time of evaluation |
| **A FOOT radiographic series should be performed if there is pain in the midfoot region with any one of the following:** |
| Bone tenderness at the navicular bone |
| Bone tenderness at the base of the fifth metatarsal |
| Inability to bear weight for at least 4 steps both immediately after injury and at the time of evaluation |

Notes


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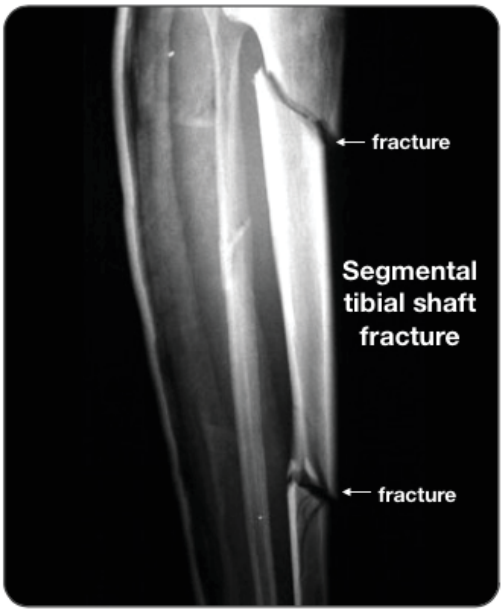

**Ankle Sprain Classification by Grade**

| Severity | Physical Exam Findings | Impairment | Pathophysiology | Treatment |
|---|---|---|---|---|
| Grade 1 | • Minimal tenderness and swelling | Minimal | Microscopic tearing of collagen fibers | • Weight bear as tolerated, physical therapy |
| Grade 2 | • Moderate tenderness and swelling<br>• Decreased range of motion<br>• Possible instability | Moderate | Complete tears of some but not all collagen fibers in the ligament | • Immobilize with air splint<br>• Physical therapy |
| Grade 3 | • Significant swelling and tenderness<br>• Instability | Severe | Complete tear or rupture of ligament | • Immobilization<br>• Physical therapy<br>• Possible surgical reconstruction |

Notes

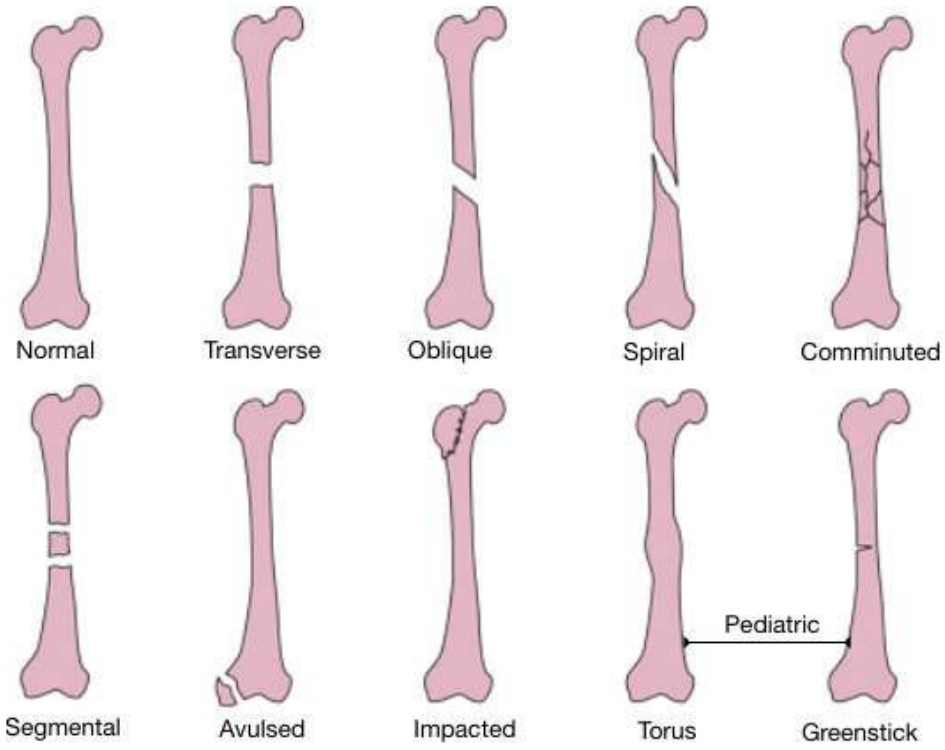

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## 6. FRACTURES




Fracture Descriptors
- **Closed fracture**: skin and soft tissue overlying the fracture are intact
- **Open fracture**: fracture exposed to the outside environment via break in the skin

Types of Fractures
- **Transverse**: fracture occurs at a right angle to the long axis of the bone
- **Oblique**: fracture runs oblique to the long axis of the bone
- **Spiral**: fracture encircles the shaft of the bone in a spiral fashion
- **Comminuted**: fracture where there are more than two fragments present




Notes

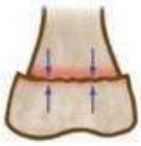

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### A. Salter-Harris Fractures (SALTR)

Classification
- I: **S**lipped epiphysis
- II: fracture **A**bove physis; most common
- III: fracture be**L**ow physis
- IV: fracture **T**hrough physis
- V: **R**ammed physis

Management
- I–II management: nonoperative
- III–V management: surgery required
- Negative radiographs do not rule out a Salter I fracture

**Salter-Harris Fracture Classification**

| | Type | Characteristics |
|---|---|---|
|  | I | **S – Slipped**<br>Epiphyseal slip, separation through the physis (growth plate)<br>Excellent prognosis, managed non-operatively |
|  | II | **A – Above growth plate**<br>Fracture through a portion of the physis (growth plate) that extends through the metaphysis<br>Excellent prognosis, managed non-operatively |
|  | III | **L – Lower than growth plate**<br>Fracture through a portion of the physis (growth plate) that extends through the epiphysis and into the joint<br>Often unstable |
|  | IV | **T – Through the growth plate**<br>Fracture across the metaphysis, physis (growth plate), and epiphysis<br>Prone to limb length discrepancies |
|  | V | **ER – ERasure of growth plate**<br>Crush injury to the physis (growth plate)<br>Prone to limb length discrepancies |

Notes


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### B. Lisfranc Injuries

Patient
• History of a motor vehicle accident, fall, or playing sports

Presentation
• Severe foot pain and an inability to bear weight

Physical Exam
• Tenderness of the **tarsometatarsal joint**

Management
• Immobilization or surgical repair

Comments
• Post-traumatic arthritis is the most common complication




**Mechanism**
• Severe plantar flexion of the foot
• Sports-related injuries, MVCs, fall from height

**Clinical**
• Pain at tarsal-metatarsal joints
• Ecchymosis, instability

**Management**
• Immobilization or surgical repair

Notes


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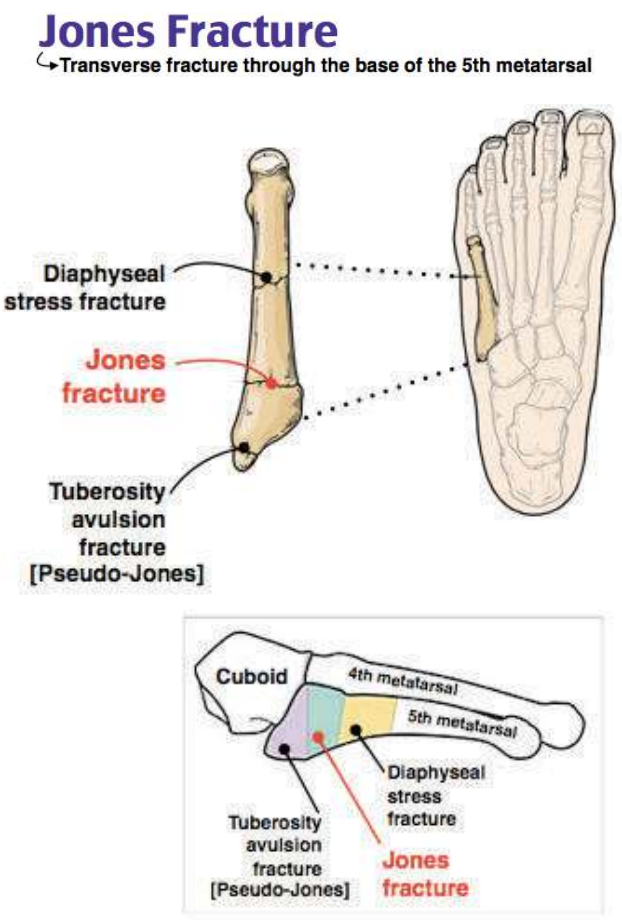
### C. Jones and Pseudo-Jones Fractures

Jones
- **Fifth metatarsal diaphysis** fracture ≥ 1.5 cm distal to base
- Complications include malunion/nonunion
- Management: cast or splint, non-weightbearing

Pseudo-Jones
- Fifth metatarsal base **avulsion** fracture
- Bulky dressing or hard sole shoe

**Jones Fracture**
- **Increased risk for MALUNION**
- A transverse fracture at the base of the fifth metatarsal
- 1.5 to 3 cm distal to the proximal tuberosity at the metadiaphyseal junction
- Without distal extension and no articular involvement

**Pseudo-Jones Fracture**
- A fracture of the proximal 5th metatarsal reaching the articular surface




Notes


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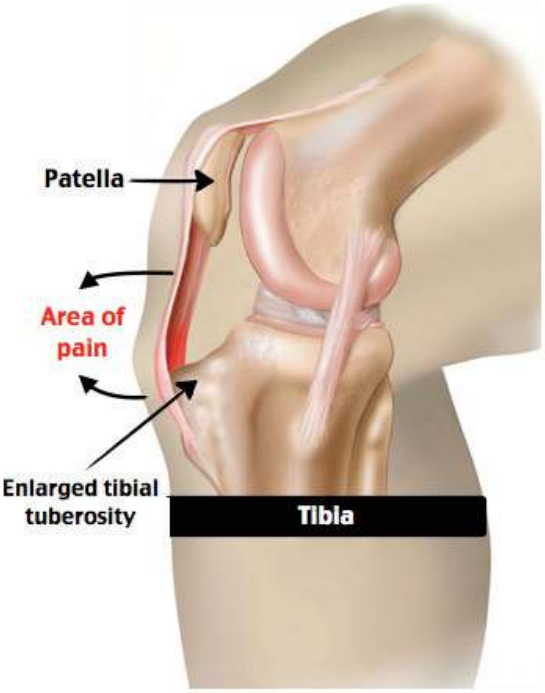
### D. Osgood-Schlatter Disease

Patient
• Young, athletic **boy** aged **10–15 years old**

Presentation
• Knee pain while **running**

Physical Exam
• **Tenderness** over the **tibial tubercle**

Management
• Ice, NSAIDs, quadriceps stretching
• Surgical intervention if refractory to conservative management




- **Rupture of growth plate** at the tibial tuberosity
- **Rapidly growing** adolescents
- More common in **athletes**
- Pain and tenderness at **tibial tubercle**

Notes

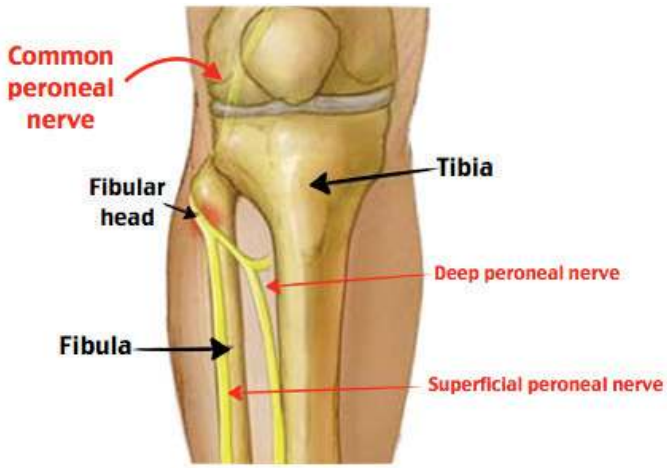

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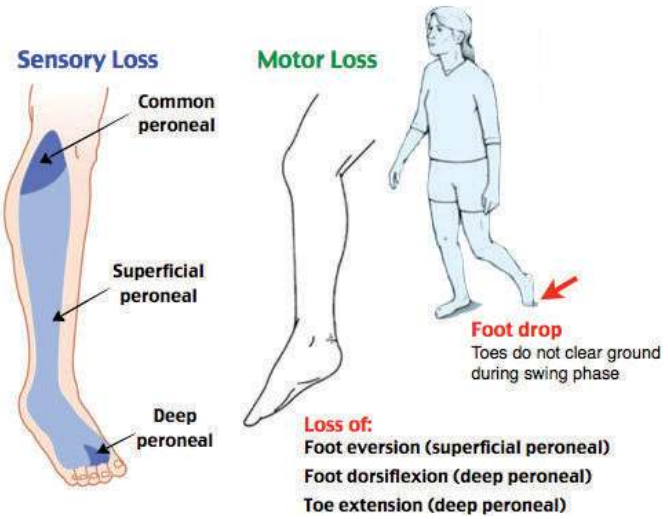
### E. Common Peroneal Neuropathy

Pathophysiology
- **Most commonly** caused by proximal fibula injury
- Can occur after poor positioning during surgery

Physical Exam
- **Foot drop** with **numbness** in web space between **first and second toes**

Management
- Ankle splint




Notes


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## F. Slipped Capital Femoral Epiphysis (SCFE)

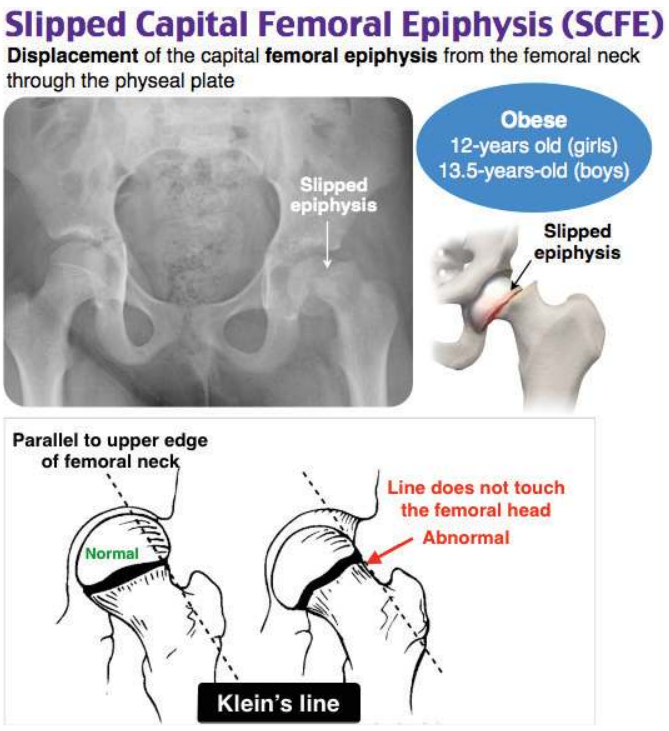
Patient
• **Obese boy** aged **12–16 years old**

Presentation
• **Progressive limp** with **hip** and **knee pain**

Physical Exam
• Decreased range of **internal rotation**

Diagnostic Studies
• X-rays (AP and frog leg lateral views) revealing **"scoop of ice cream slipping off an ice cream cone"**

Management
• Non-weightbearing status and urgent orthopedic consultation




**Diagnosis**
• Plain film (AP, frog-leg/cross-table lateral)

**Management**
• Operative stabilization

Notes

**Clinical**

*Preslip*
• Pain, but no discernible displacement of epiphysis

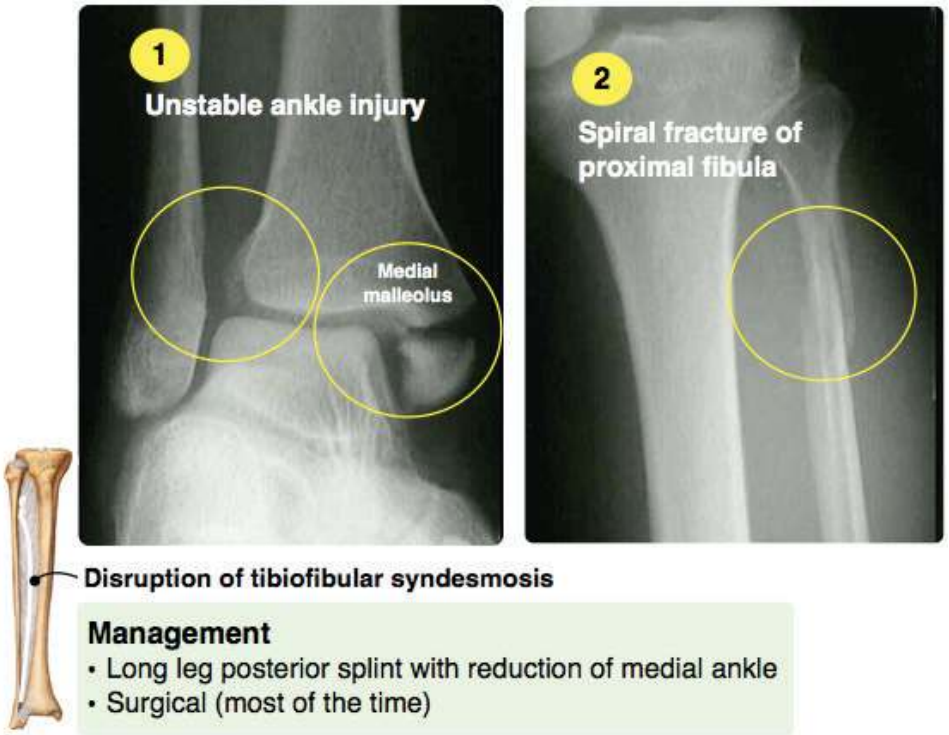
*Acute*
• Severe pain, external rotation deformity
• Limited range of motion of hip
• Inability to bear weight
• Joint effusion
• No metaphyseal remodeling

*Acute-on-chronic*
• Acute pain in chronic SCFE

*Chronic*
• Most common pattern of presentation
• Nonradiating, dull, aching pain in the hip, groin, thigh, or knee
• No history of preceding trauma
• Remodeling present


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### D. Maisonneuve Fracture




**Management**
- Long leg posterior splint with reduction of medial ankle
- Surgical (most of the time)

Notes


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# VI. Gout

**A. Gout**

Pathophysiology
• Caused by **uric acid** crystals
• **Most commonly** affects the **first MTP (podagra)**

Patient
• Middle-aged man with a history of consuming alcohol and processed meats

Presentation
• Acute onset of joint pain

Physical Exam
• Warmth, erythema, swelling, and tenderness to affected joint
• Nodular accumulation of uric acid (**tophi**)

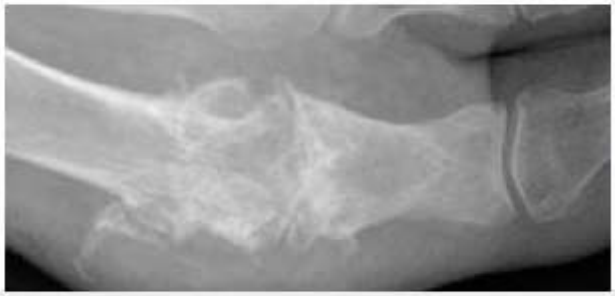
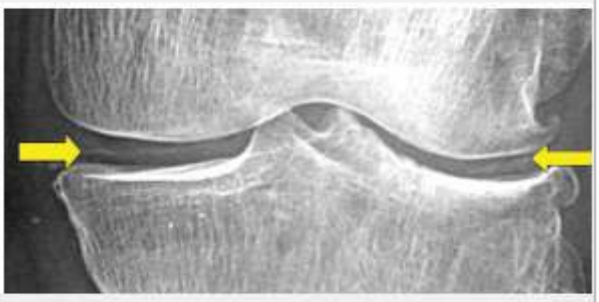
Diagnostic Studies
• Joint aspiration reveals **needle-shaped**, **negatively birefringent** crystals
• Serum uric acid **not clinically reliable** in acute setting

Management
• **Acute**: NSAIDs, steroids, **colchicine**
• **Chronic**: **allopurinol** or **colchicine**

Comments
• Can be triggered by **loop** and **thiazide diuretics**

Notes

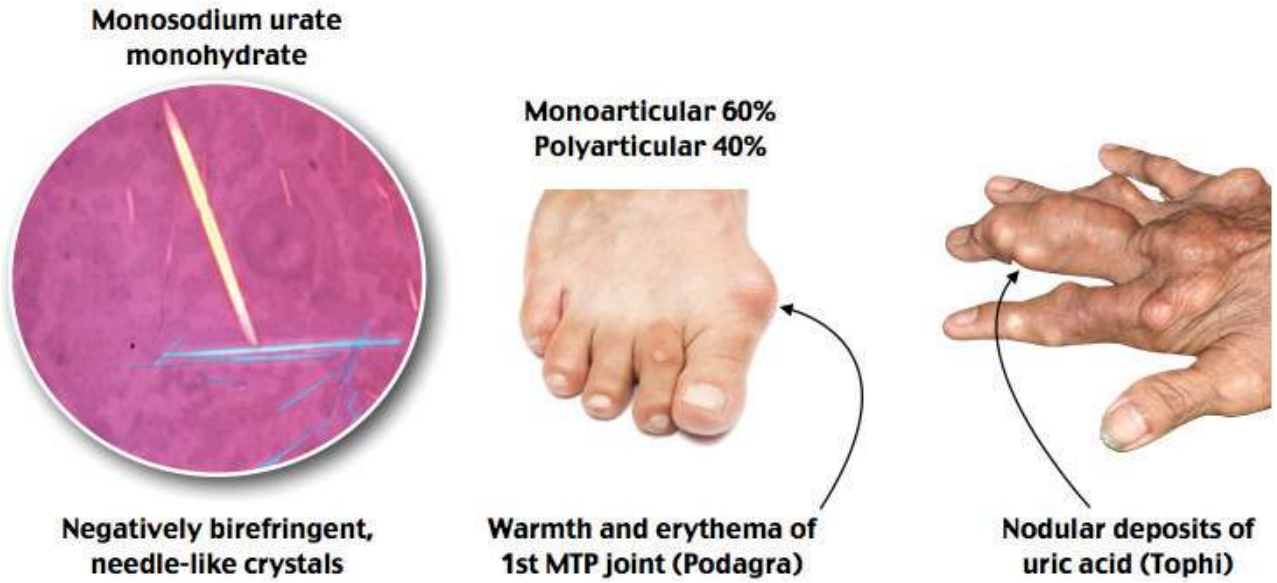

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| Characteristic | Gout | Pseudogout |
|---|---|---|
| Crystal composition | Uric acid | Calcium pyrophosphate |
| |  |  |
| Crystal shape | Needle-like | Rhomboid |
| Birefringent | Negative | Weakly positive |
| Most common joint affected | 1st MTP | Knee |
| Radiography | "Rat-bite" erosions | White lines of chondrocalcinosis |
| |  |  |
| First line treatment | NSAIDs | NSAIDs |

Notes


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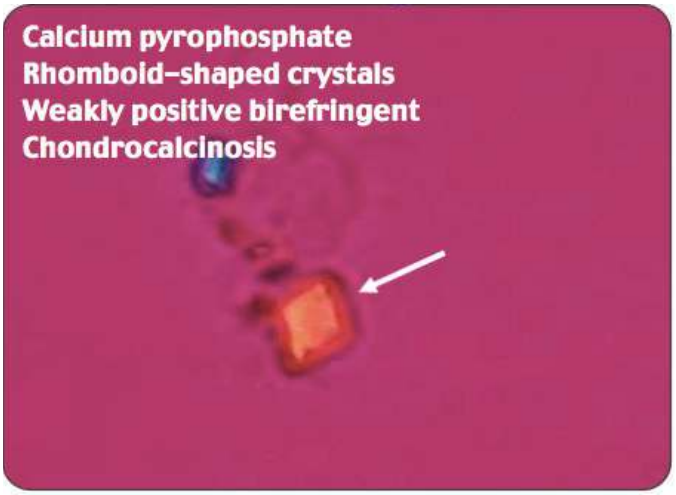
**B. Gouty Arthritis**




Notes


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### C. Pseudogout




Pathophysiology
• Caused by **calcium pyrophosphate crystals**
• **Most commonly** affects the **knee** joint

Patient
• Middle-aged man

Presentation
• **Monoarticular** joint pain

Diagnostic Studies
• Joint aspiration reveals **rhomboid-shaped**, **weakly positive birefringent** crystals

Management
• NSAIDs

Notes


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# VII. Herniated Disk

### A. Herniated Disk Pulposus

Pathophysiology
- Caused by herniation of the nucleus pulposus into the lumbar spinal canal
- **Most commonly** affected locations: L4–L5 and L5–S1

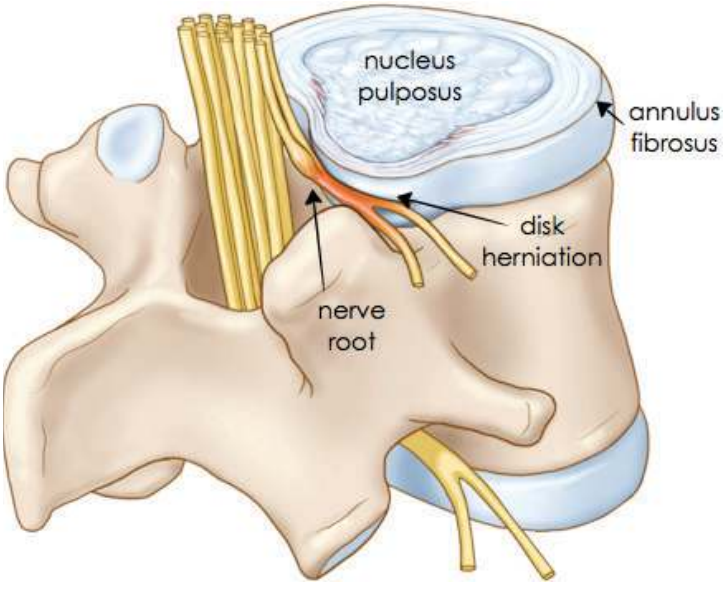
Presentation
- Abrupt onset of pain that is **severe** and exaggerated by sitting, walking, standing, or coughing
- Pain radiates from the buttock down the **posterior** or **posterolateral** leg to the **ankle** or **foot**

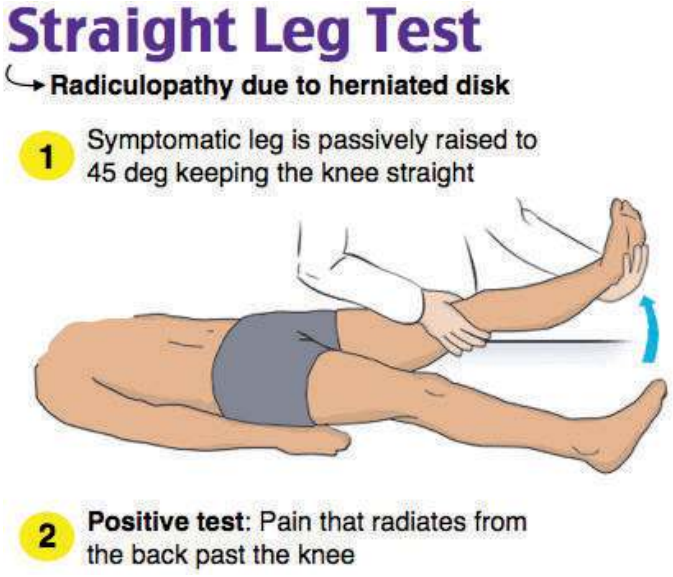
Physical Exam
- **Positive straight leg raise test**, tenderness in the sciatic notch, and limited ROM

Diagnostic Studies
- MRI is diagnostic

Management
- Physical therapy, surgical intervention if refractory

Notes





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# VIII. Osteomyelitis

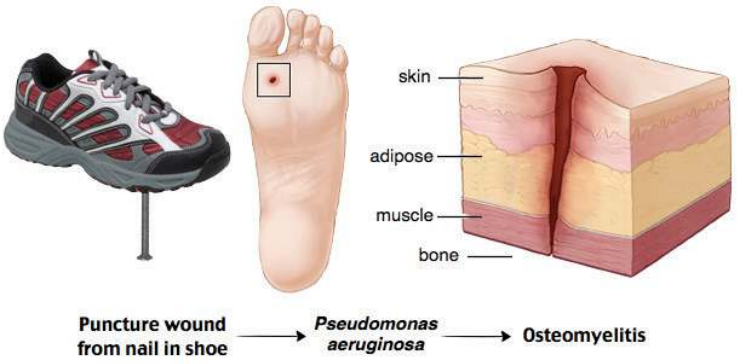
### A. Osteomyelitis

Pathophysiology
- **Most common** causative organisms:
  - General: ***S. aureus***
  - Sickle cell: ***Salmonella***
  - Cat/dog bites: ***Pasteurella multocida***
  - Puncture wound in shoe: ***Pseudomonas aeruginosa***

Diagnostic Studies
- X-ray will show periosteal elevation or bony erosions
- Diagnosis is made by **X-ray** (may be normal early), bone scan, or MRI

Management
- Long-term antibiotics

Comments
- Adults: **contiguous** spread **most common**
- Children: **hematogenous** spread **most common**







Notes


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# IX. Septic Arthritis

### A. Septic Arthritis




Pathophysiology
• **Most commonly** caused by ***S. aureus*** overall (***N. gonorrhoeae*** if younger than 35 years old)

Presentation
• Fever, monoarticular pain with decreased range of motion

Diagnostic Studies
• Diagnosis is made by **arthrocentesis**
• Joint fluid: **WBC > 50,000** with **> 75% PMNs**

Management
• IV antibiotics, surgical washout

Notes


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**Septic Arthritis cont.**

| Organism | Clinical Clues |
|---|---|
| *Staphylococcus aureus* | • Healthy adults **(most common overall)**<br>• Skin breakdown<br>• Previously damaged joint<br>• Prosthetic joint |
| Streptococcal sp. | • Healthy adults<br>• Splenic dysfunction |
| *Neisseria gonorrhoeae* | • Healthy, sexually active young adults<br>• Associated tenosynovitis, vesicular pustules<br>• Late complement deficiency<br>• Negative synovial fluid culture & Gram-stain |
| Aerobic/Anaerobic Gram-negative bacteria | • Immunocompromised hosts<br>• GI infection |
| Brucellosis | • Zoonosis |
| Mycobacterial sp. | • Immunocompromised hosts |
| Fungal sp. | • Immunocompromised hosts |
| Spirochete (*Borellia burgdorferi*) | • Exposure to ticks<br>• Antecedent rash |
| *Mycoplasma hominis* | • Immunocompromised hosts<br>• Prior urinary tract manipulation |

| | WBC | Color | Viscosity |
|---|---|---|---|
| **Normal** | < 150 | Colorless/Straw | High |
| **Noninflammatory** | < 3,000 | Straw/Yellow | High |
| **Inflammatory** | > 3,000 | Yellow | Low |
| **Septic (purulent)** | > 50,000 | Pus/Mixed | Mixed |
| **Hemorrhagic** | Similar to blood | Red | Low |

Notes


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| Kocher Criteria to Determine Risk for Pediatric Septic Joint | |
|---|---|
| Non-weight bearing on affected side | *Probability of Septic Arthritis*<br>• 4/4 = 99%<br>• 3/4 = 93%<br>• 2/4 = 40%<br>• 1/4 = 3% |
| ESR > 40 mm/hr | |
| Fever ≥ 38.5°C | |
| WBC > 12,000 | |

Notes


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CHAPTER 3

# Gastrointestinal/ Nutritional

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CHAPTER 3

# Gastrointestinal/Nutritional


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180 **OBSTRUCTION (SMALL BOWEL, LARGE BOWEL, VOLVULUS)**

186 **PEPTIC ULCER DISEASE**



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# I. Abdominal Pain

### A. Age Differences in Abdominal Pain

| | Age Differences in Abdominal Pain | | |
|---|---|---|---|
| | 2-12 yrs old | 13-49 yrs old | > 50 yrs old |
| Nonspecific abdominal pain | ++ | +++ | ++ |
| Appendicitis | + | +++ | ++ |
| Cholecystitis | + | ++ | +++ |
| Bowel obstruction | + | + | +++ |
| Non-abdominal causes | +++ | ++ | ++ |

Notes


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### B. Rapid Onset of Abdominal Pain

**R-I-P-O**

**RUPTURE**
- Abdominal aortic aneurysm (AAA)
- Ectopic pregnancy
- Splenic rupture
- Esophagus (Boerhaave syndrome)

**ISCHEMIA**
- Mesenteric ischemia
- Testicular torsion
- Ovarian torsion
- Incarceration of hernia
- Myocardial infarction

**PERFORATION**
- Peptic/duodenal ulcer
- Diverticulitis
- Toxic megacolon
- Caustic ingestion
- Foreign body

**OBSTRUCTION**
- Small/Large bowel
- Volvulus
- Intussusception
- Gallbladder
- Ureter (urolithiasis)

Notes


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# II. Acute Appendicitis

**A. Appendicitis**

Pathophysiology
• **Most commonly** caused by a **fecalith**

Presentation
• Fever, pain that began **periumbilically** then moved **to RLQ**, nausea, and anorexia

Physical Exam
• **Psoas sign** (RLQ pain on extension of right hip), **obturator sign** (RLQ pain on internal rotation of flexed right hip), **Rovsing sign** (RLQ pain when the LLQ is palpated)

Diagnostic Studies
• Ultrasound, CT

Management
• Surgery

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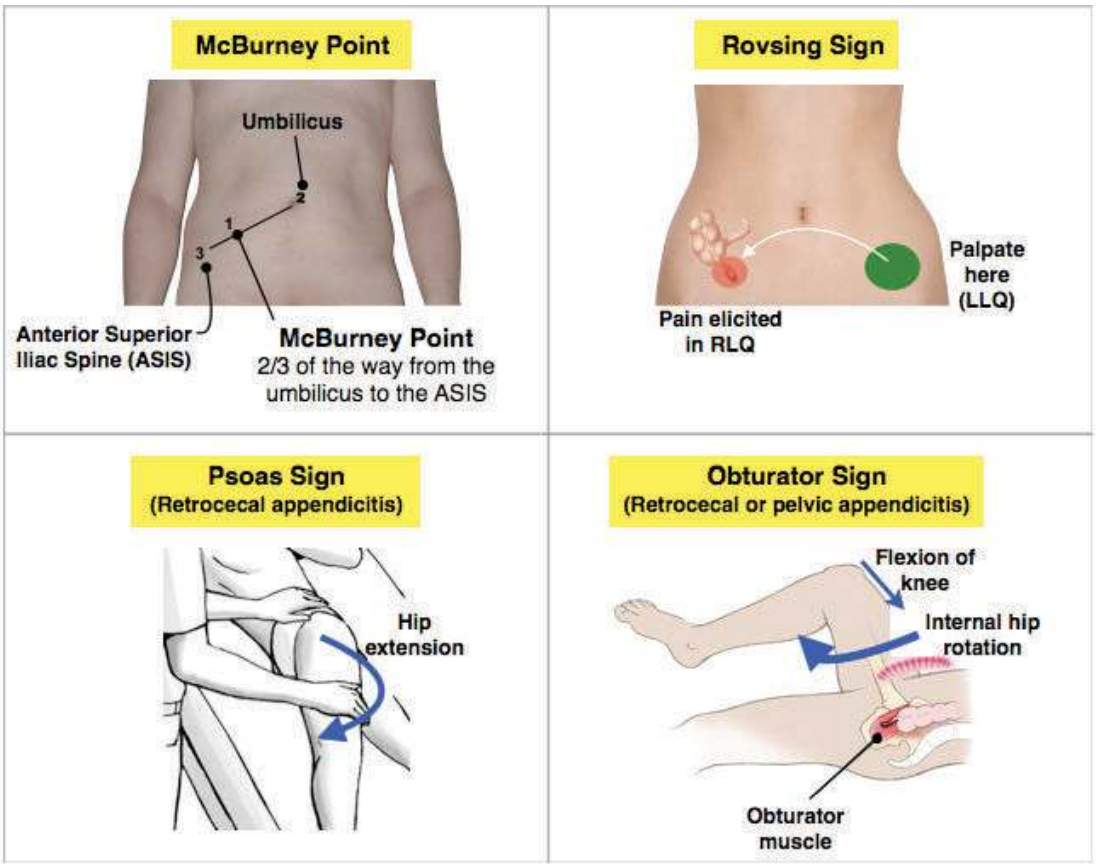
## B. Acute Appendicitis

**Epidemiology**
- Most frequent in the second and third decades of life
- Most common in 10-to-19-year-old group
- More common in men

**Management**
- Appendectomy
- Antibiotics only (reserved for cases of nonperforated, uncomplicated appendicitis

**Clinical**
- Right lower quadrant abdominal pain
- Anorexia
- Nausea and vomiting
- Periumbilical pain that migrates to RLQ

**Atypical features**
- Dyspepsia
- Flatulence
- Bowel irregularity
- Diarrhea
- Generalized malaise




Notes


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# III. Acute Cholecystitis

**Acute Cholecystitis**

Pathophysiology
• **Most commonly** caused by an **obstructive gallstone**

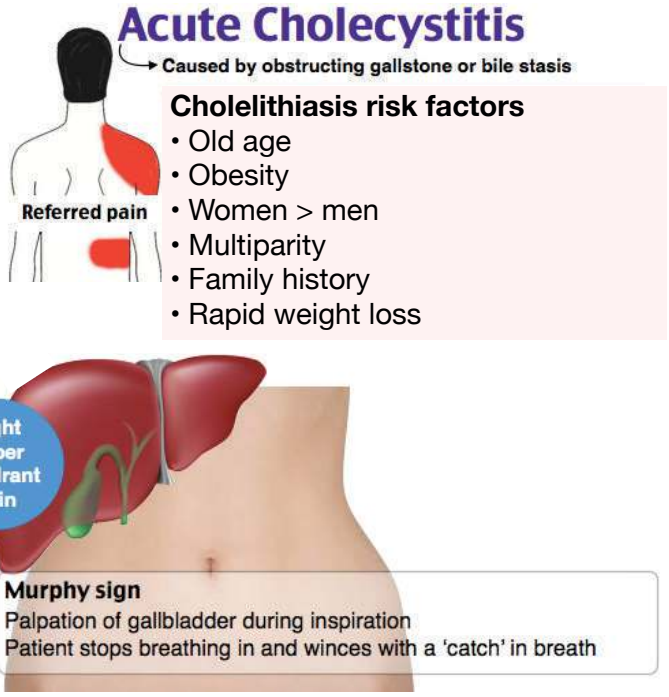
Presentation
• **Colicky**, steadily increasing right upper quadrant (RUQ) or epigastric pain after eating **fatty foods**
• Fever, nausea, vomiting

Physical Exam
• Positive **Murphy sign** and **Boas sign** (hyperaesthesia (increased or altered sensitivity) below the right scapula)

Diagnostic Studies
• Initially: RUQ ultrasound
• **Gold standard**: **HIDA scan**

Management
• Cholecystectomy

**Clinical**
• Right upper quadrant or epigastric pain
• Steady pain
• Fever, nausea, vomiting, anorexia




**Cholelithiasis risk factors**
• Old age
• Obesity
• Women > men
• Multiparity
• Family history
• Rapid weight loss

**Diagnosis**
• Ultrasound
• Hepatobiliary iminodiacetic acid scan (HIDA)
• CT scan

**Management**
• Supportive care
• Cholecystectomy

Notes


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# IV. Acute Hepatitis

### A. Acute Hepatitis

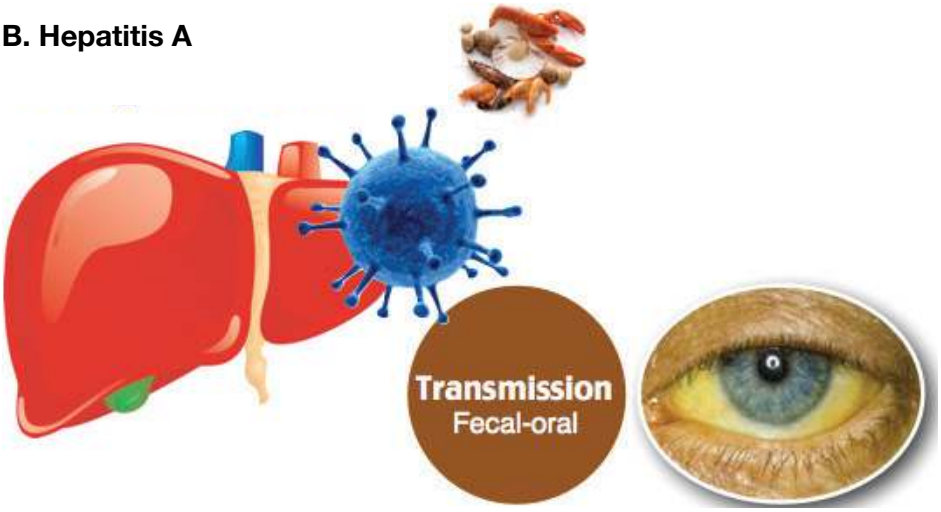
Presentation
• Fatigue, flu-like symptoms, dark urine, pale stool, abdominal pain, loss of appetite, unexplained weight loss, or jaundice

Pathophysiology
• HAV: fecal-oral, shellfish, alone (no carrier)
• HBV:
  • HBsAg: active infection
  • Anti-HBs: recovered or immunized
  • Anti-HBc IgM: early marker of infection, positive in window period
  • Anti-HBc IgG: best marker for prior HBV
  • HBeAg: high infectivity
  • Anti-HBeAb: low infectivity
• H**C**V: IVDA, chronic, cirrhosis, carcinoma, carrier
• **HDV**: **dependent on HBV coinfection**
• HEV: fecal-oral (enteric), high mortality rate among pregnant (expectant) patients, epidemics
• HAV and HEV are fecal-oral: **"The vowels hit your bowels"**
• HAV and HBV have preventative vaccine available
• Autoimmune hepatitis: young females
• Alcoholic hepatitis: moderate transaminase elevation, **AST > ALT (2:1)**
• Supportive care

Notes


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**B. Hepatitis A**




**Clinical**
*Acute*
• Symptoms develop 2-3 weeks after infection and last 8 weeks
• Nausea, vomiting, diarrhea
• Fever, jaundice, abdominal pain

**Diagnosis**
• Anti-HAV IgM is positive in acute infection
• Anti-HAV IgG is positive in prior infection or immunization

**Prevention**
• Vaccination (formalin-killed)

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### C. Hepatitis B

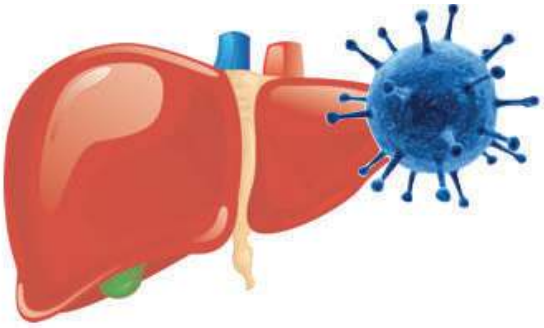
| Hepatitis B | | | | | |
|---|---|---|---|---|---|
| **Clinical Status** | **HBsAg** | **Anti-HBs** | **Anti-HBc** | **HBeAg** | **Anti-HBe** |
| **Acute** hepatitis B infection | + | - | IgM | + | - |
| **Chronic** hepatitis B with **active viral replication** | + | - | IgG | + | - |
| **Chronic** hepatitis B with **low viral replication** | + | - | IgG | - | + |
| **Recovery** from hepatitis B (immunity) | - | + | IgG | - | + or - |
| **Immunity** from **previous vaccination** | - | + | - | - | - |
| (1) Remote infection and anti-HBs waned<br>(2) Window period<br>(3) Low level chronic infection | - | - | IgM | - | - |

| Hepatitis B Virus | | |
|---|---|---|
| **Test** | | **Use** |
| **Hepatitis B surface antigen** | **HBsAG** | • Screen for, detect, and diagnose acute and chronic HBV infections<br>• Earliest routine indicator of acute HBV<br>• Identifies infected people before symptoms appear<br>• Undetectable during recovery<br>• Identifies those with chronic infections and HBV carrier |
| **Hepatitis B surface antibody** | **Anti-HBs** | • Used to detect previous exposure to HBV<br>• Can develop from successful vaccination<br>• Determines the need for vaccination<br>• If present, person is immune to future HBV infection |
| **Total anti-hepatitis B core** | **Anti-HBc, IgM and IgG** | • Helps to detect acute and chronic HBV infection<br>• IgM first antibody produced after HBV infection<br>• IgG produced in response to core antigen later in course and persists for life |
| **Anti-hepatitis B core, IgM** | **Anti-HBc** | • Used to detect acute infections |
| **Hepatitis B e antigen** | **HBeAG** | • Marker of infectivity<br>• Monitor effectiveness of treatment |
| **Anti-hepatitis B e antibody** | **Anti-HBe** | • Used to monitor acute infections |

Notes


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**D. Hepatitis C**



**Transmission** **Clinical**

Blood-borne

**Acute** - most individuals are **asymptomatic**

→ **80%** go on to **chronic** infection

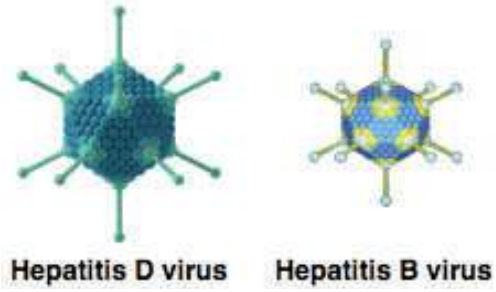
→ **20%** of chronic infections go on to develop **cirrhosis** in 20 yrs



Notes


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### E. Hepatitis D




- Simultaneous presence of HBV is required for complete viron assembly and secretion
- Individuals with hepatitis D are always dually infected with HDV and HBV
- HBV replication, however, is suppressed

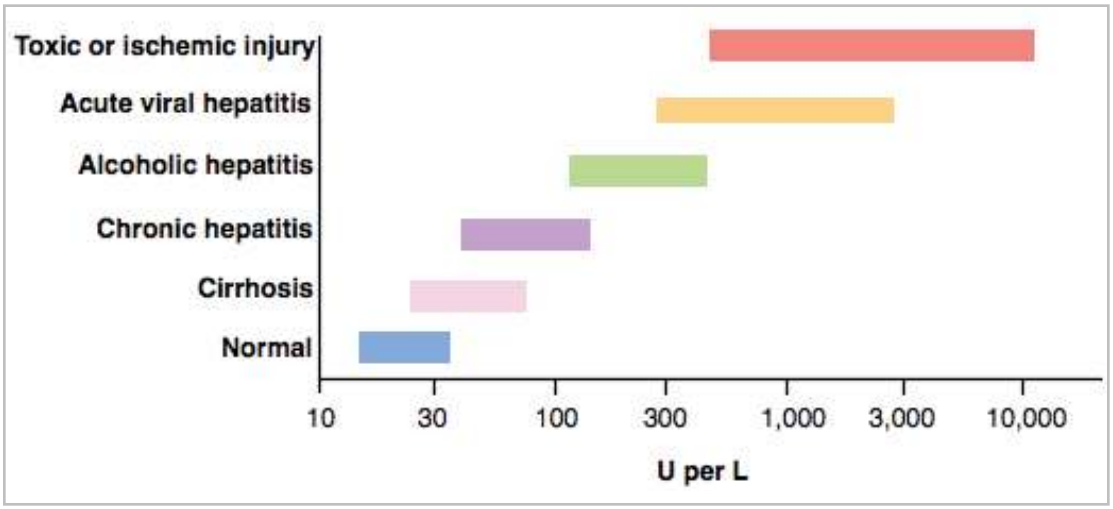
### F. Alcoholic Hepatitis




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### G. Typical AST or ALT Values in Selected Diseases




Notes


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# V. Acute Pancreatitis

### A. Acute Pancreatitis

Pathophysiology
• **Most commonly** caused by **gallstones**, then alcohol

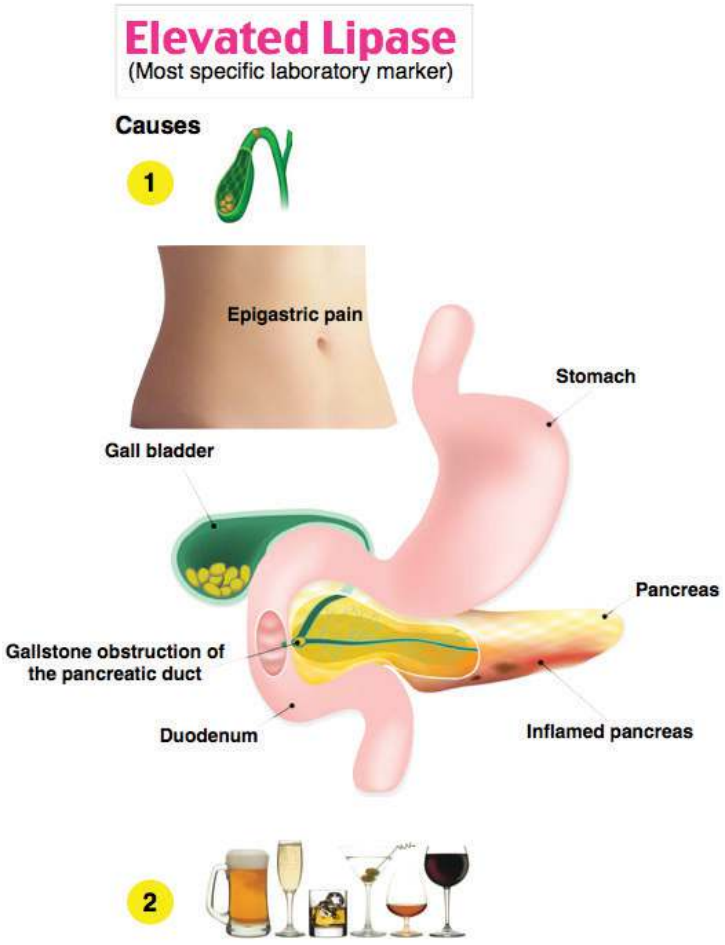
Presentation
• **Epigastric pain radiating to the back**, nausea, and vomiting

Physical Exam
• Ecchymosis of left flank (**Grey-Turner sign**) and umbilical ecchymosis (**Cullen sign**) are seen in **hemorrhagic** pancreatitis

Diagnostic Studies
• **Elevated lipase** (best) and amylase
• Ultrasound

Management
• **IV fluids**
• **Ranson criteria** and APACHE are used to predict the severity (Note: they are difficult to apply and have not been found to be that reliable)

Comments
• Pancreatic pseudocyst is a complication in adults




Notes


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**Acute Pancreatitis cont.**

| Ranson's Criteria |
|---|
| *At Admission* |
| Age > 55 years |
| WBCs > 16,000/mm$^3$ |
| Glucose > 200 mg/dl |
| LDH > 350 IU/L |
| AST > 250 SF units |
| *48 hours After Admission* |
| Hematocrit fall >10% |
| BUN rise > 5 mg/dl |
| Calcium < 8 mg/dl |
| $PO_2$ < 60 mm Hg |
| Base deficit > 4 mEq/L |
| Fluid sequestration > 6 L |

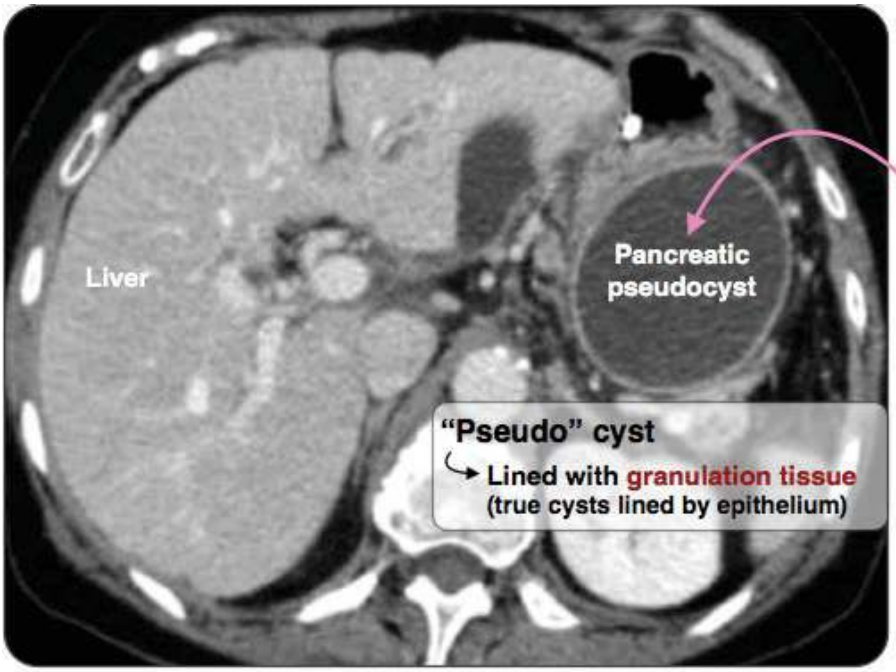
| Score | Associated Mortality Rate |
|---|---|
| 0-2 | 1% |
| 3-4 | 15% |
| 5-6 | 40% |
| > 7 | 100% |



Notes


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## B. Pancreatic Pseudocyst




### Etiology

- Adult: Complication of acute pancreatitis
- Pediatric: Following abdominal trauma

| | |
|---|---|
| **Early (< 4 weeks)**<br>Infection is rare<br>Drainage not recommended | • Acute peripancreatic collection<br>• Acute necrotic collection |
| **Late (> 4 weeks)**<br>Drainage is reasonable if symptomatic | • Pseudocyst<br>• Walled-off necrosis |

Notes


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# VI. Anal Fissure/Fistula/Abscess

## A. Anal Fissure

Presentation
• **Rectal pain** and **bleeding**, onset with or shortly after **defecation**

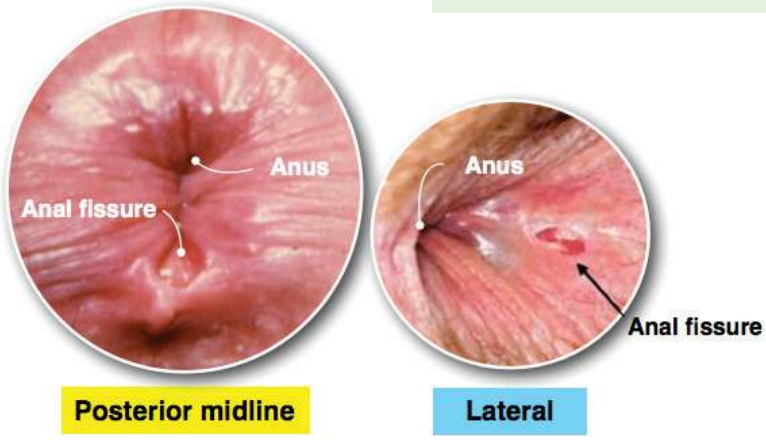
Physical Exam
• **Most common** location of fissure is **posterior midline**

Management
• Stool softeners, protective ointments, sitz baths

Comments
• Fissures oriented laterally should raise concern for pathologic etiology

| **Primary Anal Fissures** | **Secondary Anal Fissures** |
|---|---|
| • Local trauma | • Crohn disease |
| • Constipation | • Other granulomatous diseases |
| • Diarrhea | • Malignancy |
| • Vaginal delivery | • Communicable disease |
| • Anal sex | |

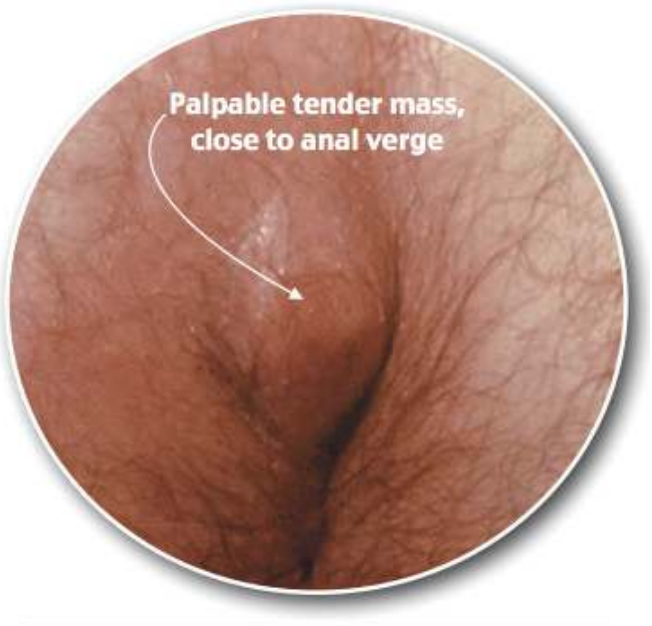
**Management**
• Topical nifedipine or nitroglycerin
• Topical analgesic
• Stool softener
• Sitz bath
• Fiber




Notes


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**B. Perianal Abscess**




Presentation
• Dull, achy pain in rectum

Physical Exam
• Careful rectal exam

Management
• Operative drainage

Comments
• Complication: **fistula**

**Clinical**
• Severe pain in the anal and rectal area
• Constant pain
• Constitutional symptoms (fever, malaise)
• Can result in fistula formation
• Incision and drainage

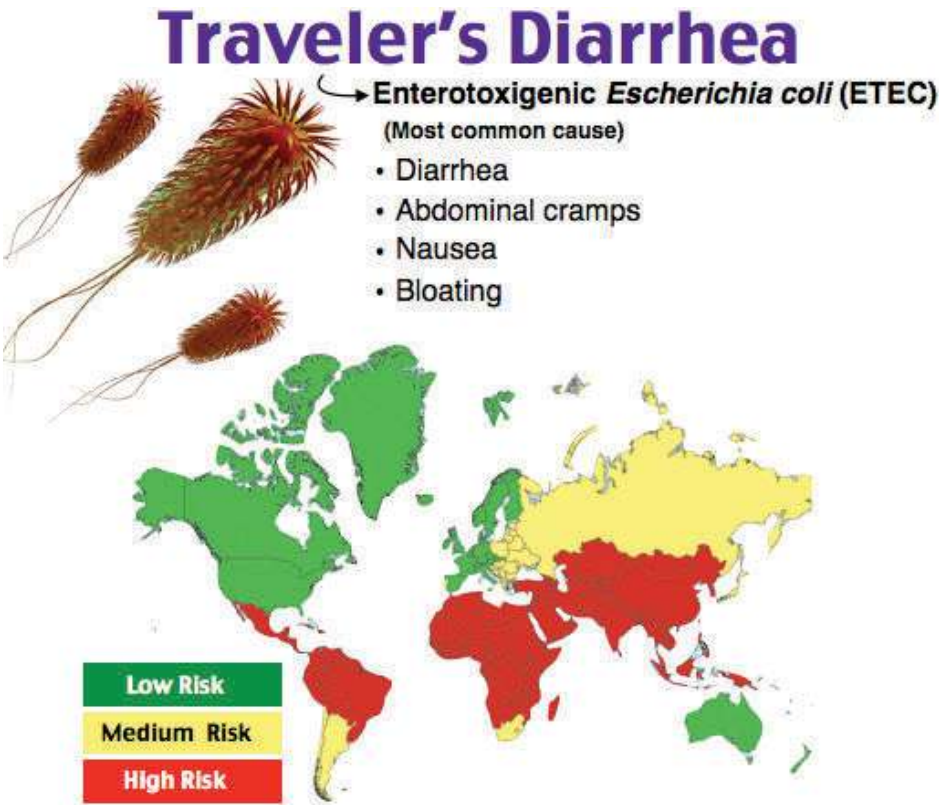
Notes


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# VII. Change in Bowel Habits/Diarrhea/Constipation

## 1. DIARHHEA

### A. Traveler's Diarrhea




Notes


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## 2. CONSTIPATION

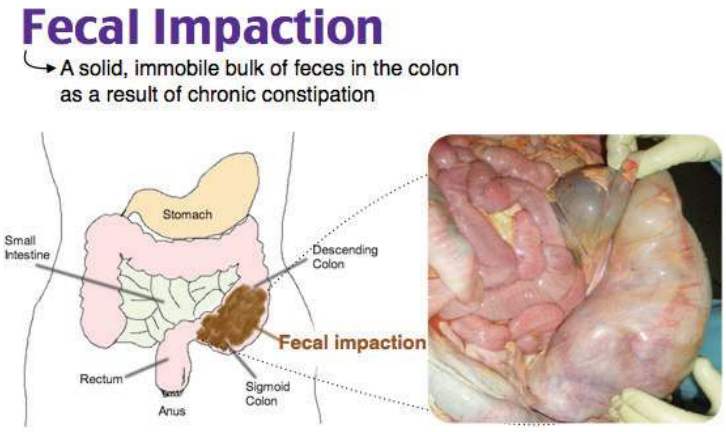
### A. Fecal Impaction

Patient
• Elderly bed-bound patient

Presentation
• Abdominal cramping and bloating, small amount of **stool leakage** and rectal discomfort

**Etiology**
• Lack of fiber
• Opioids
• Irritable bowel syndrome
• Diabetes
• Hypothyroidism

**Etiology**
• Abdominal distension and pain
• Chronic constipation
• Fecal incontinence/overflow diarrhea




**Complications**
• Rectal necrosis and ulcers

**Management**
• Manual disimpaction
• Stool softening
• Osmotic laxatives
• Surgery

Notes


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**B. Common Laxatives**

| | |
|---|---|
| **Bulk formers** | Psyllium, methylcellulose |
| **Softeners** | Docusate |
| **Stimulants** | Senna, bisacodyl, castor oil |
| **Saline preps** | Magnesium hydroxide, magnesium citrate, magnesium sulfate |
| **Lubricants** | Mineral oil |
| **Osmotics** | Lactulose, sorbitol, polyethylene glycol |
| **Prokinetics** | Tegaserod, metoclopramide |

Notes


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# VIII. Cholangitis

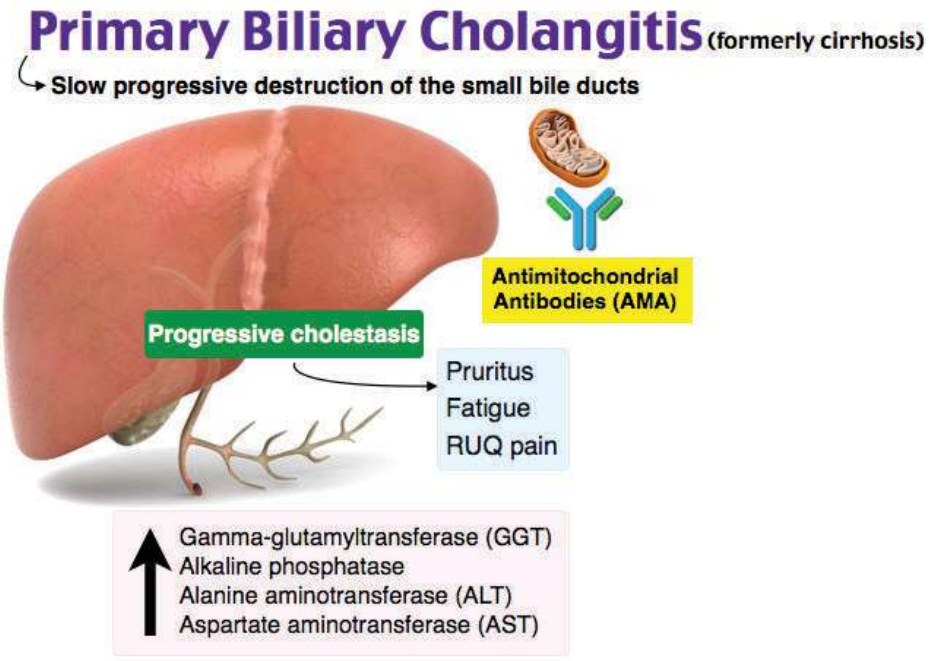
### A. Primary Biliary Cholangitis

Pathophysiology
• **Most commonly** caused by an autoimmune **T cell-mediated** attack on the **interlobular bile ducts** causing a slow, progressive destruction

Patient
• Woman **40–50 years old**

Presentation
• Fatigue, jaundice, pruritus, RUQ discomfort, weight loss

Diagnostic Studies
• Serum **antimitochondrial antibodies** (**AMA**)




Notes


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### B. Primary Sclerosing Cholangitis

Patient
• Man with a history of **ulcerative colitis**

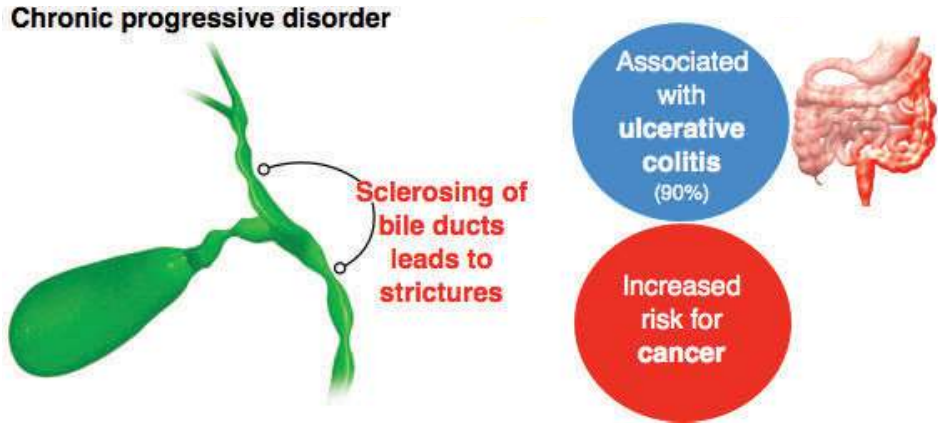
Presentation
• Pruritus, jaundice, fatigue, malaise

Diagnostic Studies
• Lab evaluation reveals a **cholestatic pattern** with **elevation** of **alkaline phosphatase**
• Cholangiogram via magnetic resonance cholangiopancreatography (**MRCP**) or endoscopic retrograde cholangiopancreatography (**ERCP**)

**Clinical**
• Asymtpomatic (50%)
• Pruritus
• Fatigue

**Complications**
• Cholestasis
• Liver failure

**Management**
• Drug therapy (e.g. immunosuppressive & anti inflammatory)
• Endoscopic therapy
• Surgical therapy
• Liver transplant




Notes


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### C. Acute Cholangitis

Pathophysiology
• **Most commonly** caused by **choledocholithiasis** leading to **bacterial infection** (***E. coli***)

Presentation
• RUQ pain, jaundice, fever (**Charcot triad**)

Diagnostic Studies
• Initially: RUQ ultrasound or CT scan
• **Gold standard**: **ERCP**

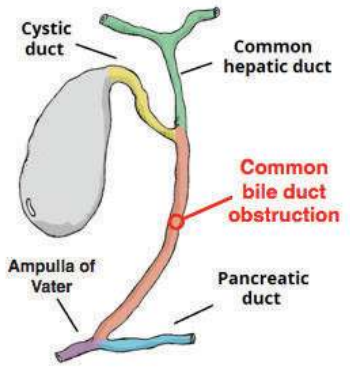
Management
• Antibiotics

Comments
• **Charcot triad** with hypotension and AMS makeup (**Reynolds pentad**)

## Acute (Ascending) Cholangitis

A clinical syndrome characterized by **fever**, **jaundice**, and **abdominal pain** that develops as a result of **stasis** and **infection** in the **biliary tract**.

**Bacterial infection** in a patient with **biliary obstruction**




**Charcot triad**
Fever
Abdominal pain
Jaundice

**Reynolds pentad**
Fever
Abdominal pain
Jaundice
+
Confusion
Hypotension

**Management**
• Broad-spectrum antibiotics (e.g., pipericillin tazobactam)
• Biliary drainage (e.g., ERCP)

Notes


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# IX. Cirrhosis

### A. Cirrhosis

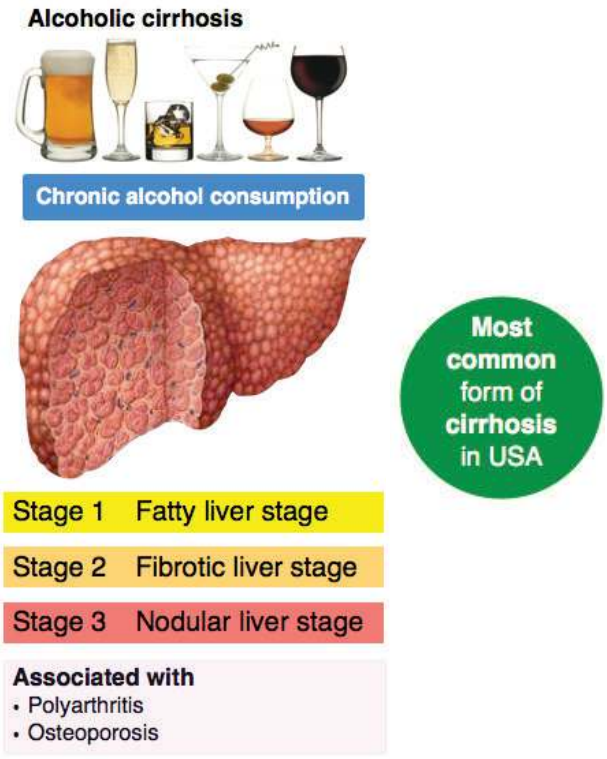
Pathophysiology
• **Most commonly** caused by **alcohol**, then hepatitis and autoimmune disorders

Physical Exam
• Gynecomastia, palmar erythema, delayed clotting
• **Hepatic encephalopathy**: asterixis, confusion
• **Portal hypertension**: esophageal varices, anorectal hemorrhoids, caput medusae (**the gut**, **the butt**, and **the caput**), splenomegaly, ascites

Management
• **Lactulose** for hepatic encephalopathy

Comments
• **Increased** risk for hepatocellular carcinoma

### B. Laennecs Cirrhosis




Notes


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## C. Chronic Liver Disease



**Hepatomegaly** and **Ascites**



**Cirrhosis**



**Caput medusae**
(Dilated veins around the umbilicus due to portal htn)



**Gynecomastia**
(Impaired breakdown of estrogens)



**Icterus**
(Increased bilirubin due to dysfunction of bilirubin metabolism)



**Palmar erythema**
(Impaired breakdown of sex hormones)



**Spider nevi**
(Isolated telangiectasias)



**Ecchymosis**
(Defective coagulation)



**Leukonychia**
(Hypoalbuminemia)



**Finger clubbing**



**Asterixis**
(Abnormal motor fct due to faulty metabolism)

**FETOR HEPATICUS**

(Characteristic odor due to volatile aromatic compounds)

Notes


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# X. Diverticular Disease

### A. Diverticulosis

Patient
• Obese, sedentary, cigarette smoker with a diet **high** in red meat and **low** in fiber

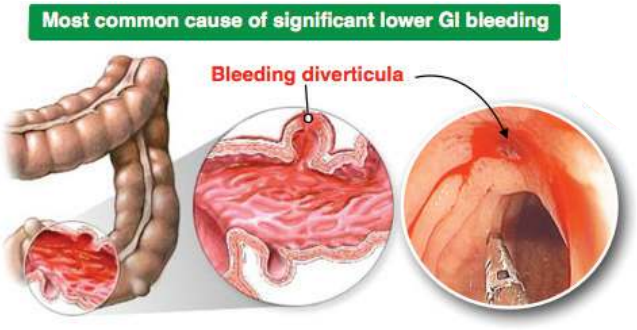
Presentation
• **Painless hematochezia**

Painless rectal bleeding

Diagnostic Studies
• **Colonoscopy**

Management
• Supportive care

Comments
• **Most common** cause of **significant lower gastrointestinal bleeding**




**Clinical**
• Often asymptomatic
• May have bloating, crampy abdominal pain
• **Painless rectal bleeding**

**Management**
• Supportive care
• Most bleeding stops spontaneously
• Surgery for persistent bleeding

**Diagnosis**
• CT scan
• Colonoscopy
• Nuclear scan or angiogram

Notes


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### B. Diverticulitis

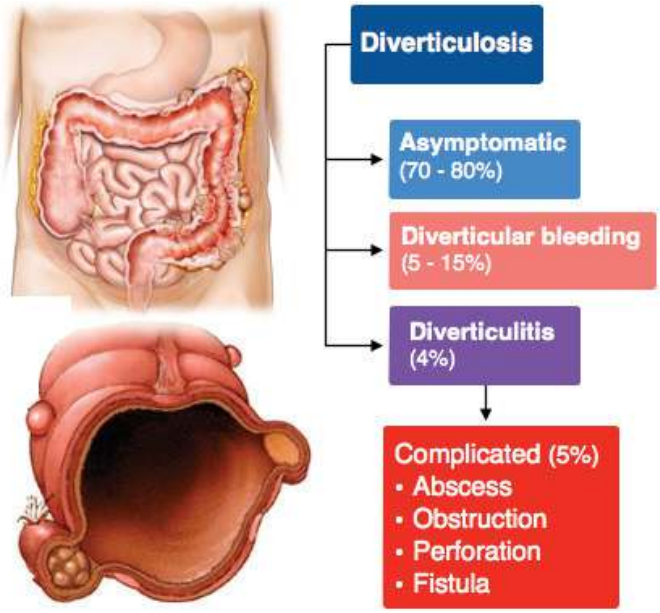
Presentation
• **Left lower quadrant** (LLQ) abdominal pain, **fever**, nausea, vomiting, and change in bowel habits

Physical Exam
• LLQ tenderness, guarding, rigidity, and rebound tenderness

Diagnostic Studies
• **CT scan**

Management
• Antibiotics (inpatient vs outpatient)
• If complicated or recurrent, consider **surgical management**

### C. Acute Diverticulitis




**Clinical**
• Abdominal pain (most common complaint)
• Nausea and vomiting
• Fever
• Change in bowel habits
• Hematochezia is rare

**Diagnosis**
• CT Scan (95% sensitive, 99% specific)
  - Localized bowel wall thickening ( > 4mm)
  - Pericolonic fat stranding
  - Colonic diverticula
• Ultrasound
• MRI

**Management**
• Inpatient vs Outpatient
• Antibiotics
• Surgical (complicated)

Notes


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# XI. Esophagitis

### A. Infectious Esophagitis

- Infectious **Candida** (**Esophageal Candidiasis**)
  - **Most common** in patients with **HIV**
  - **Odynophagia** (pain on swallowing) present
  - Linear **yellow-white plaques** seen on EGD
  - Treat with **fluconazole**

- **HSV**: **shallow** ulcers noted on EGD, treat with **acyclovir**

- **CMV**: **deep** ulcers on EGD, treat with **ganciclovir**




**Etiology**
- Candida (Esophageal candidiasis)
- Herpes simplex (Herpes esophagitis)
- Cytomegalovirus (CMV esophagitis)

**Clinical**
- Dyspepsia
- Nausea
- Postprandial worsening of symptoms

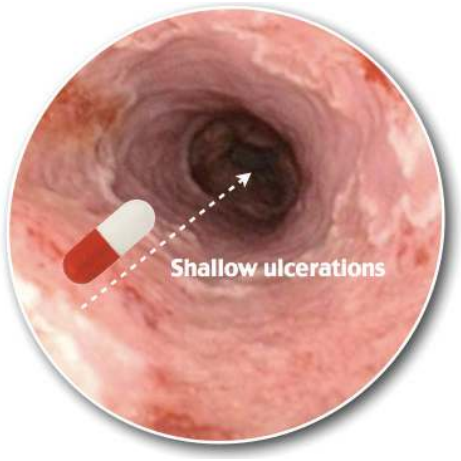
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**B. Pill Esophagitis**

• Caused **most commonly** by NSAIDs or **bisphosphonates**

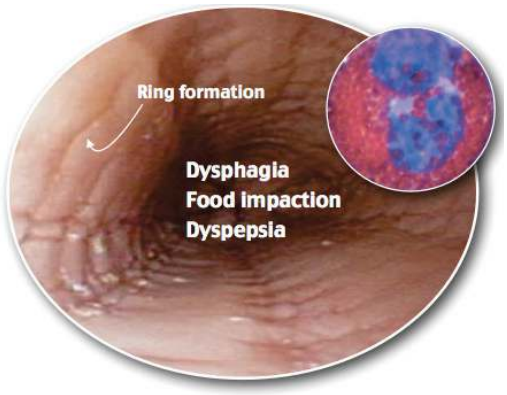
**Causes**
• Antibiotics (tetracycline, doxycycline, clindamycin)
• Anti-inflammatories (NSAIDs, aspirin)
• Bisphosphonates (alendronate, risedronate)
• Ferrous sulfate (iron)
• Ascorbic acid (vitamin C)
• Potassium chloride




Notes


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### C. Eosinophilic Esophagitis




Pathophysiology
• Allergic, **eosinophilic** infiltration of the esophagus

Presentation
• **Asthma symptoms** and **GERD** not responsive to antacids

Diagnostic Studies
• Barium swallow will show **multiple corrugated rings**

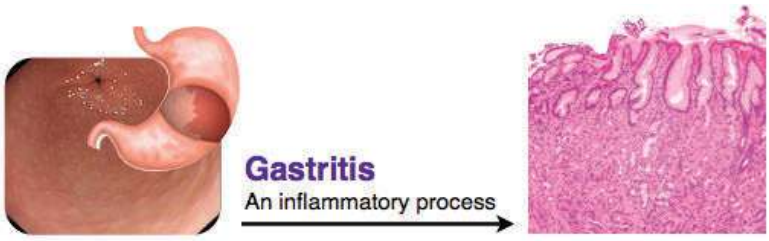
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# XII. Gastritis

### A. Gastritis

- Acute: **NSAIDs more common** than alcohol
- Type A chronic: **pernicious anemia**
- Type B chronic : ***H. pylori***




**Etiology-Based Classification of Gastritis and Gastropathy**

| | | |
|---|---|---|
| **Gastritis** (An inflammatory process) | Infectious gastritis | • *Helicobacter pylori*<br>• Phlegmonous gastritis<br>• Mycobacterial<br>• Syphilitic<br>• Viral<br>• Parasitic<br>• Fungal |
| | Autoimmune gastritis | • Autoimmune gastritis |
| | Granulomatous disease | • Crohn disease<br>• Sarcoidosis |
| | Uncertain etiology | • Lymphocytic gastritis<br>• Collagenous gastritis<br>• Eosinophilic gastritis |
| **Gastropathy** (A gastric mucosal disorder with minimal to no inflammation) | Reactive (chemical) gastropathy | • Bile reflux<br>• Alcohol<br>• NSAIDs<br>• Iron salts |
| | Vascular gastropathy | • Portal hyertensive (congestive)<br>• Gastric antral vascular ectasia |
| | Ischemic gastropathy | • Trauma<br>• Burns<br>• Sepsis<br>• Hypovolemia<br>• Cocaine |

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# XIII. Gastroenteritis

### A. Gastroenteritis

Pathophysiology
• **Most commonly** caused by a **viral** pathogen

Presentation
• Frequent loose stools, with or without fever and vomiting, lasting from one day to two weeks

Management
• Supportive care

Notes


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### B. Non-Invasive Gastroenteritis

| Non-Invasive Gastroenteritis | | | | |
|---|---|---|---|---|
| Causative agent | Incubation | Transmission | Preformed toxin | Description |
| **Viral** | 11-72 hrs | • Person-to-person<br>• Fecal-oral<br>• Contaminated food or water | No | • Norwalk and Rotavirus most common agents<br>• N/V, watery diarrhea<br>• Mild abdominal cramps, myalgia |
| ***Giardia lamblia*** | 1-4 wks | • Contaminated food or water<br>• Person-to-person<br>• Fecal-oral | No | • Backpackers, campers<br>• Flatus, bloating<br>• Most common intestinal parasite in the US |
| ***Staph. aureus*** | 1-6 hrs | • Previously cooked foods (ham, egg salad, potato salad) | Yes | • Symptoms caused by preformed enterotoxin<br>• Nausea, severe vomiting, diarrhea, mild abdominal cramping |
| ***Bacillus cereus*** | 1-6 hrs | • Previously cooked meats, fried rice, vegetables, and dried fruits | Yes | • Symptoms caused by preformed enterotoxin<br>• Abrupt onset of nausea, vomiting, and mild diarrhea |
| ***Clostridium perfringens*** | 8-24 hrs | • Previously cooked or reheated meats and poultry | Yes | • Symptoms caused by preformed enterotoxin<br>• Abdominal cramps, nausea, min vomiting, watery diarrhea |
| ***Vibrio cholerae*** | 11-72 hrs | • Raw or undercooked seafood<br>• Fecal-oral<br>• Contaminated water | No | • Enterotoxins are formed before and after bacterial colonization<br>• Explosive rice-water diarrhea<br>• Vomiting, fever, abdominal cramps |

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### C. Invasive Gastroenteritis

| Invasive Gastroenteritis | | | |
|---|---|---|---|
| **Causative agent** | **Incubation** | **Transmission** | **Description** |
| ***Salmonella* sp.** | 8–72 hrs | • Contaminated food (eggs and poultry) or water, pet turtles, chicks, lizards | • Fever, abdominal pain, myalgia, headache<br>• Risk of sepsis in very young/old, immunocompromised |
| ***Shigella sp.*** | 1–3 days | • Contaminated food<br>• Person-to-person<br>• Fecal-oral | • Fever, myalgia, headache, diarrhea, little vomiting<br>• Common in kids 1–5 yrs old |
| ***Campylobacter sp.*** | 1–7 days | • Contaminated water, food (poultry, eggs), animals, pets | • Low-grade fever, abdominal pain, and vomiting may precede diarrhea<br>• Common in children and young adults |
| ***Yersinia enterocolitica*** | 1–5 days | • Contaminated water, food (milk, pork), pets, or wild animals<br>• Fecal-oral<br>• Person-to-person | • May mimic appendicitis, RLQ pain, fever, vomiting precedes diarrhea<br>• Children and young adults |
| ***Clostridia difficile*** | 1–11 wks | • Recent ABX use (PCN, clindamycin, cephalosporins) | • Fever, abdominal pain, copious foul-smelling diarrhea<br>• Rarely vomiting |
| ***Entamoeba histolytica*** | 1–11 wks | • Contaminated food or water<br>• Poor sanitation<br>• Travel to developing countries | • May mimic appendicitis<br>• Abrupt onset of nausea, vomiting, and mild diarrhea |
| ***Enterohemorrhagic E. coli 0157:H7*** | 3–8 days | • Contaminated food or water<br>• Undercooked meats<br>• Fecal-oral | • Fever, abdominal pain, vomiting<br>• Grossly bloody diarrhea<br>• Hemolytic uremic syndrome (HUS)<br>• Fecal WBCs |

Notes


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### D. Acute Viral Gastroenteritis

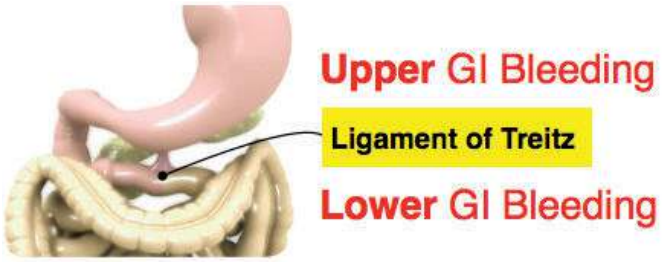
| Acute Viral Gastroenteritis | | | | | |
|---|---|---|---|---|---|
| **Virus** | **Predominant season** | **Common modes of transmission** | **Age** | **Duration** | **Other features** |
| **Rotavirus** | • Fall/winter | • Fecal-oral | • 6-24 m | • 5 to 7 d | • Causes severe childhood diarrhea<br>• Endemic |
| **Norovirus** | • All year<br>• Highest in winter | • Fecal-oral<br>• Water<br>• Shellfish<br>• Other foods | • All | • 1 to 4 d | • Vomiting is prominent symptom<br>• Causes most outbreaks of nonbacterial gastroenteritis<br>• Endemic<br>• Epidemic |
| **Sapovirus** | • All year | • Fecal-oral | • Infants<br>• Toddlers | • 3 to 4 d | • Endemic<br>• Epidemic |
| **Astrovirus** | • Winter | • Fecal-oral<br>• Water | • All | • 5 to 6 d | • Endemic<br>• Epidemic |
| **Enteric adenovirus** *Types 40 and 41* | • Summer | • Fecal-oral | • Children | • 6 to 9 d | • Endemic |

Notes

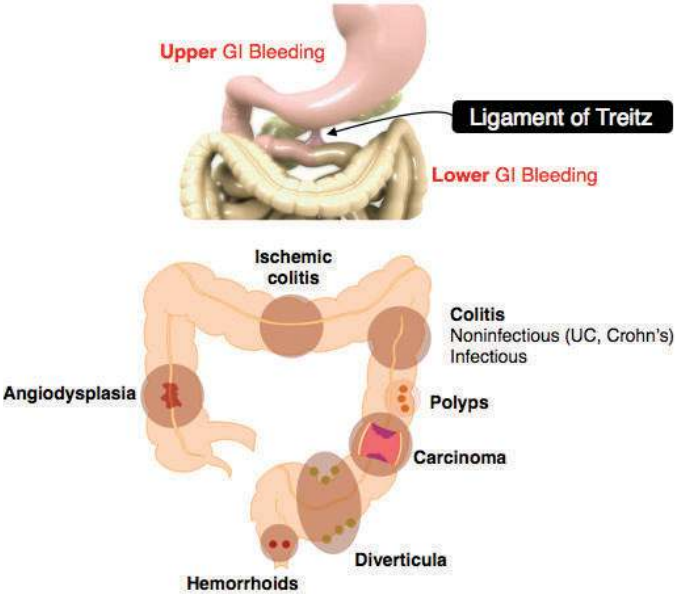

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# XIV. Gastrointestinal Bleeding

## A. Gastrointestinal Bleeding




## B. Lower GI Bleeding




Notes


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# XV. Giardiasis and Other Parasitic Infections

**A. Giardiasis**

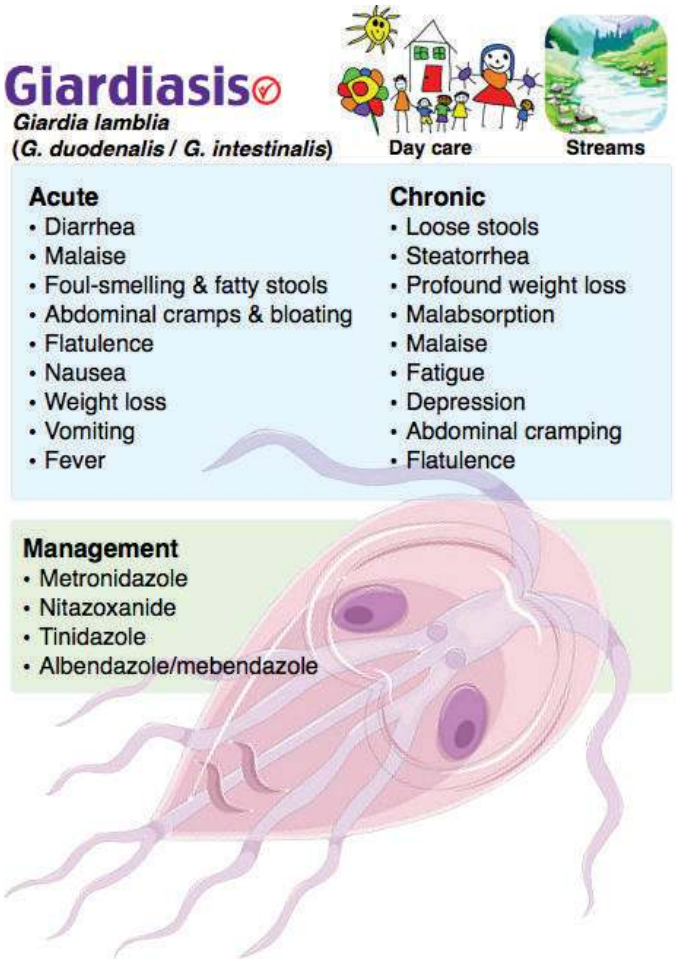
Pathophysiology
• **Most commonly** caused by ***Giardia lamblia***

Patient
• Recent camping and drinking lake water

Presentation
• Sudden onset explosive, **foul-smelling**, nonbloody diarrhea

Diagnostic Studies
• **Flagellated protozoan** in stool sample

Management
• **Metronidazole**




Notes


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# XVI. Heartburn

### A. Gastroesophageal Reflux Disease (GERD)

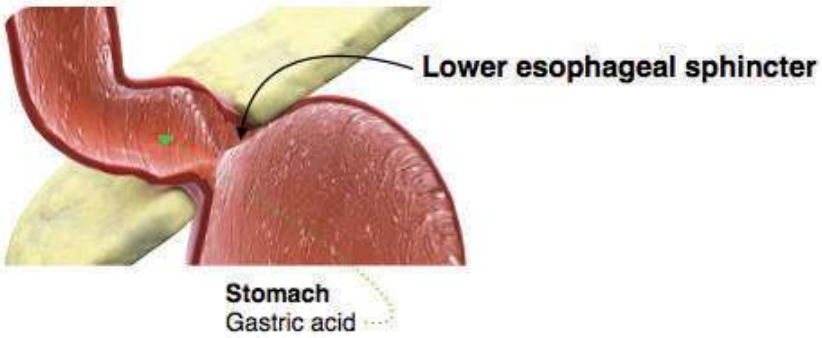
Pathophysiology
• **Most commonly** caused by **lower esophageal sphincter** (LES) dysfunction

Patient
• History of nocturnal cough or asthma

Presentation
• **Retrosternal burning sensation** radiating upward (**"heartburn"**), usually after eating

Diagnostic Studies
• Therapeutic trial of **proton-pump inhibitors** (**PPIs**)

Management
• Weight loss, **elevate head of bed** during sleep, **avoidance** of certain foods (**caffeine**, **alcohol**, **peppermint**, **acidic foods**)




**The 3 Dominant Pathophysiologic Mechanisms causing GERD**

1. Transient lower esophageal sphincter relaxations
2. A hypotensive lower esophageal sphincter
3. Anatomic disruption of the gastroesophageal junction

Notes


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**B. Barrett Esophagus**

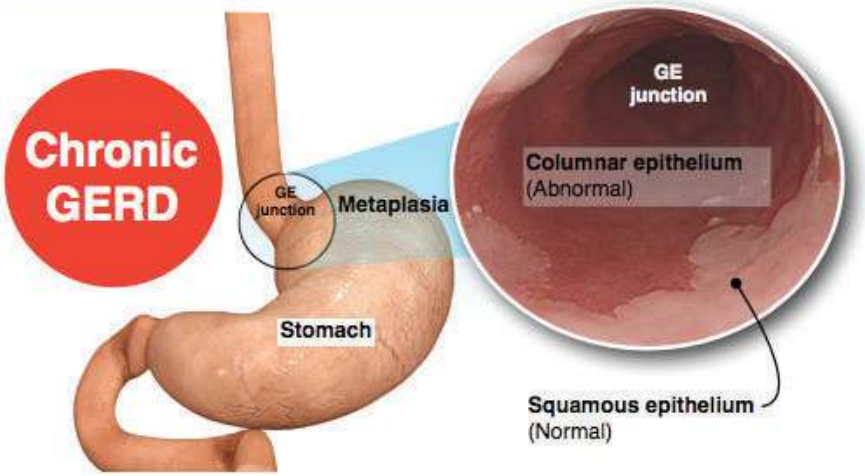
Patient
• History of **chronic GERD**

Diagnostic Studies
• Upper endoscopy
• **Biopsy** reveals transformation from squamous to **columnar** epithelium and **proximal shift** in squamocolumnar junction

Management
• PPIs

Comments
• **Increased** risk of **adenocarcinoma**

**Increased risk for adenocarcinoma**




| Feature | Management | Cancer Risk* |
|---|---|---|
| **No dysplasia** | Upper endoscopy every 3 to 5 years | 0.2% per year |
| **Low-grade dysplasia** | Upper endoscopy annually OR endoscopic treatment with radiofrequency ablation | 0.7% per year, but may be as high as 8% |
| **High-grade dysplasia** | Radiofrequency ablation or surgical esophagectomy | 7 to 19% per year |

*Risk varies depending on source

Notes


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# XVII. Hemorrhoids (Thrombosed)

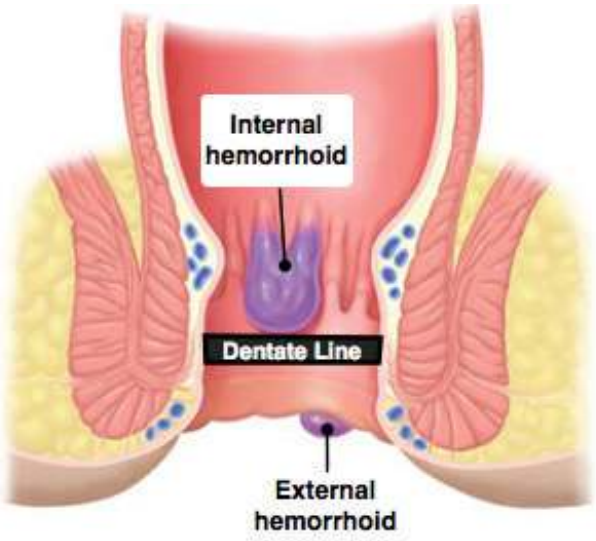
### A. Hemorrhoids

External
- **Lower 1/3** of anus (below **dentate line**)
- Significant **pain** and pruritus
- **Excision** for thrombosed external hemorrhoids

Internal
- **Upper 1/3** of anus (above **dentate line**)
- No pain, bright red blood per rectum, pruritus, and rectal discomfort
- Fiber, **sitz baths**, reduction if needed

### B. Classification of Internal Hemorrhoids

| Degree of prolapse | Clinical description |
|---|---|
| 1st | Hemorrhoids **do not protrude** through the anus |
| 2nd | Hemorrhoids **prolapse** but **reduce spontaneously** |
| 3rd | Hemorrhoids **prolapse** and **require manual reduction** |
| 4th | Hemorrhoids **cannot be reduced** and **may strangulate** |




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# XVIII. Hernia (Incarcerated/Strangulated)

### B. Hernias

| Hernias | | | |
|---|---|---|---|
| **Type** | **Pathophysiology** | **Location** | **Characteristics** |
| **Direct inguinal** | Defect of the transversalis fascia in Hesselbach triangle | Groin, **medial** to the inferior epigastric vessels | Middle-aged or **elderly men** |
| **Indirect inguinal** | Persistent processus | Groin, **lateral** to the inferior epigastric vessels | Congenital, trauma, can descend into **scrotum** |
| **Femoral** | Through femoral canal | **Upper thigh**, medial to the femoral vein | Most common in **women**, risk of incarceration and strangulation |
| **Spigelian (lateral ventral)** | Defect through the spigelian fascia (aponeurotic layer b/w rectus abdominus (medially), and semilunar line (laterally) | **Lateral** to rectus abdominus | Rare, small, high strangulation risk, right side |
| **Incisional/ ventral** | Breakdown of fascial closure from prior surgery | Site of **previous surgery** | Usually **asymptomatic**, increase in size with straining |
| **Umbilical** | Through the fibromuscular umbilical ring | Umbilicus | Repair if persists beyond 5 years |
| **Obturator** | Through the large obturator canal | Deep structures, not visualized externally | **Female** > male |
| **Epigastric** | Through **defects in aponeurosis of rectus sheath** | Midline between umbilicus and xiphoid process | Middle-aged and young children |

Notes


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## B. Inguinal Hernias

Pathophysiology
- Indirect:
  - **Most common**
  - Protrudes through internal ring, lateral to the inferior epigastric artery (**IEA**)
- Direct:
  - Protrudes **directly** through **Hesselbach triangle** and medial to the IEA
  - Bulge **improves** upon reclining

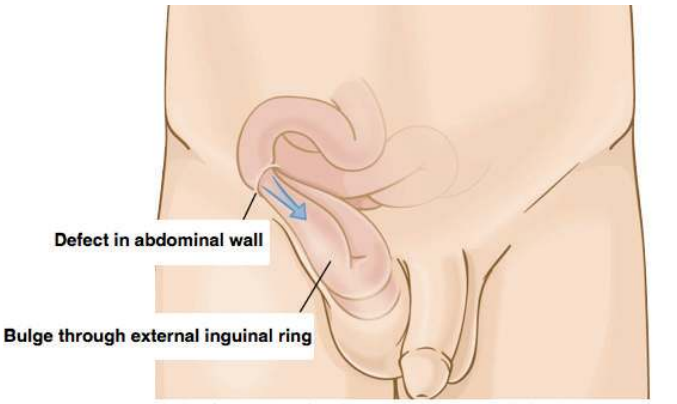
Patient
- **Bimodal** distribution ( < 1 year old, > 40 years old)

Management
- Nonreducible: **emergent** surgical consultation

Comments
- Strangulation risk **higher** with indirect hernia versus direct hernia

**Indirect**
Abdominal contents pass **lateral** to the epigastric artery, through the inguinal canal, into scrotum or labia through external inguinal ring

**Direct**
Abdominal contents pass **medial** to the epigastric artery, directly behind the superficial inguinal ring and do not extend into scrotum




Bulge **increases in size** with increased abdominal pressure (e.g. **straining, crying)**

| Characteristics of Hernias | |
|---|---|
| Reducible | Soft, easy to place back into defect |
| Incarcerated | Firm and painful, non-reducible by direct manual pressure |
| Strangulated | Painful due to impaired blood flow, ischemia, and necrosis, overlying skin changes |

Notes


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# XIX. Inflammatory Bowel Disease/ Toxic Megacolon

## A. Ulcerative Colitis

Patient
• **15–30 years old**

Presentation
• **Bloody** diarrhea, crampy abdominal pain, **tenesmus**

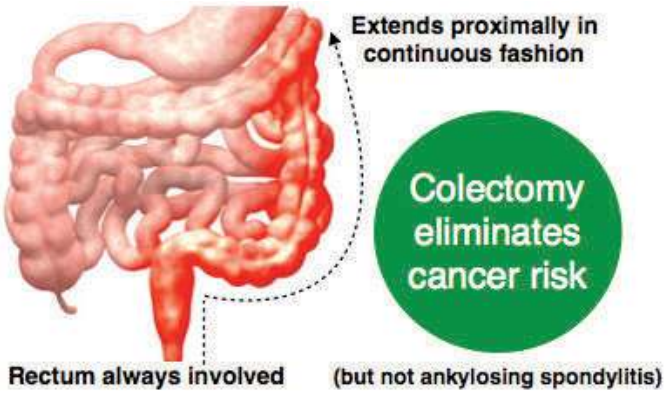
Diagnostic Studies
• Colonoscopy will show **continuous mucosal** inflammation **always involving the rectum**

Management
• **Sulfasalazine**
• Surgery is curative

**Clinical**
• Chronic bloody diarrhea
• Crampy abdominal pain
• Tenesmus
• Fecal urgency

**Treatment**
• 5-aminosalicylate (5-ASA)
• Corticosteroids
• 6-mercaptopurine, azathioprine, methotrexate

Comments
• Complications: toxic megacolon, **increased** risk of colon cancer
• **Smoking** is a protective factor




**Extraintestinal manifestations**
• Pyoderma gangrenosum
• Erythema nodosum
• Ankylosing spondylitis or sacroileitis
• Arthritis
• Uveitis
• Liver disease
• Renal stones
• Primary sclerosing cholangitis

Notes

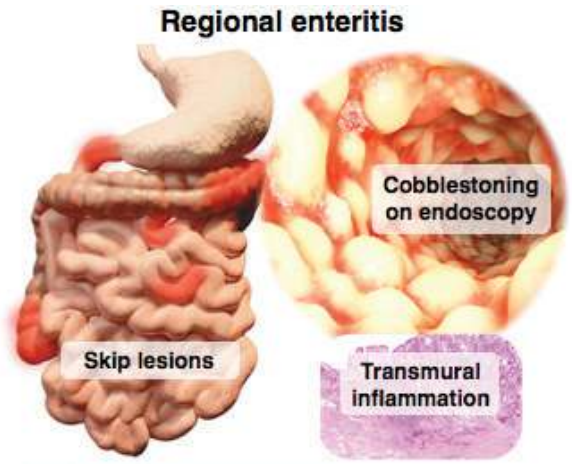

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**B. Crohn Disease**

Presentation
• Chronic **nonbloody diarrhea**, crampy abdominal pain, and weight loss

Physical Exam
• Aphthous ulcers, anal fissures, perirectal abscesses, anorectal fistulas

Diagnostic Studies
• Serum **ASCA positive**, **p-ANCA negative**
• Colonoscopy will reveal **skip lesions**, cobblestone mucosa, **transmural** involvement




Management
• **Mesalamine**
• Surgery is **not** curative

Comments
• Can affect any part of GI tract from mouth to anus

**Clinical**
• Chronic diarrhea
• Crampy abdominal pain
• Fever
• Weight loss
• Strictures
• Fistulization

**Extraintestinal manifestations**
• Pyoderma gangrenosum
• Erythema nodosum
• Ankylosing spondylitis or sacroileitis
• Arthritis
• Uveitis
• Liver disease
• Renal stones

Notes


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### C. Crohn Disease VS. Ulcerative Colitis

| | Crohn Disease | Ulcerative Colitis |
|---|---|---|
| Symptoms | • Chronic diarrhea, crampy abdominal pain, fever, weight loss, fatigue<br>• **Strictures** leading to obstructive symptoms<br>• **Fistulization** causing recurrent UTIs, pneumaturia, psoas abscess | • Chronic bloody diarrhea, abdominal pain, cramps, **tenesmus**, and fecal urgency |
| GI tract involvement | • **Transmucosal** inflammation<br>• **Skip** lesions<br>• May affect **any part of GI tract** (small bowel and colon are most common) | • Mucosal inflammation<br>• **Rectum** is always involved<br>• **Extends proximal** in a continuous fashion |
| GI bleeding manifestations | • Guaiac positive<br>• **No gross blood** | • **Grossly bloody** stools |
| Extraintestinal manifestation | • Same manifestations as UC, but more common | • **Uveitis**, **episcleritis**, **erythema nodosum**, **pyoderma gangrenosum**, arthritis, ankylosing spondylitis, thromboembolism, sclerosing cholangitis |

Notes


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## D. Toxic Megacolon

Pathophysiology
• **Most commonly** caused by inflammatory bowel disease or *C. diff*

Patient
• History of **ulcerative colitis**

Physical Exam
• Systemic toxicity

Diagnostic Studies
• Abdominal X-ray will show the colon **dilated > 6 cm**

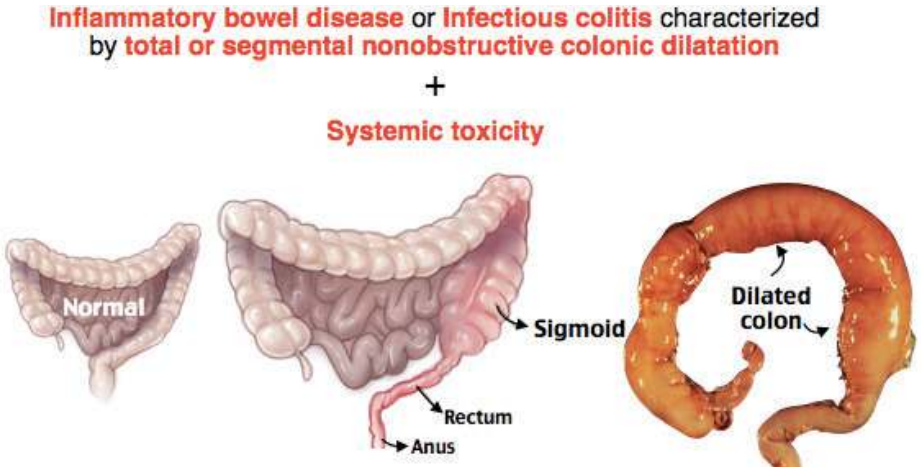
Management
• IV fluids, antibiotics, IV corticosteroids (if cause is not infectious), emergent surgical consultation

**Clinical**

Radiographic evidence of colonic distension
*PLUS* at least 3 of the following:
• Fever > 38°C
• HR > 120
• WBC > 10.5
• Anemia

*PLUS* at least 1 of the following:
• Dehydration
• Altered sensorium
• Electrolyte disturbances
• Hypotension

**Management**
• Supportive care
• Treat underlying condition




Notes


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# XX. Ischemic Bowel Disease

### A. Ischemic Colitis

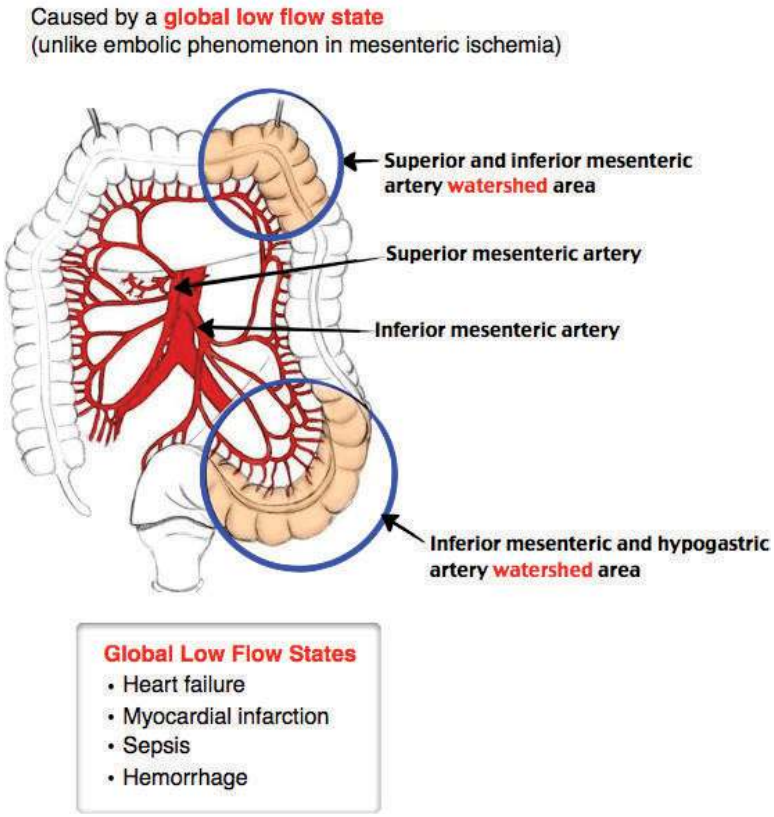
Pathophysiology
• Caused by **inadequate blood flow** through the mesenteric vessels

Patient
• History of **atherosclerotic disease**

Presentation
• Acute-onset crampy abdominal pain

Diagnostic Studies
• CT will show **bowel wall edema**

Management
• Most cases resolve with supportive care




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### B. Mesenteric Ischemia

Patient
• History of dysrhythmias (A-Fib), recent MI, or CAD

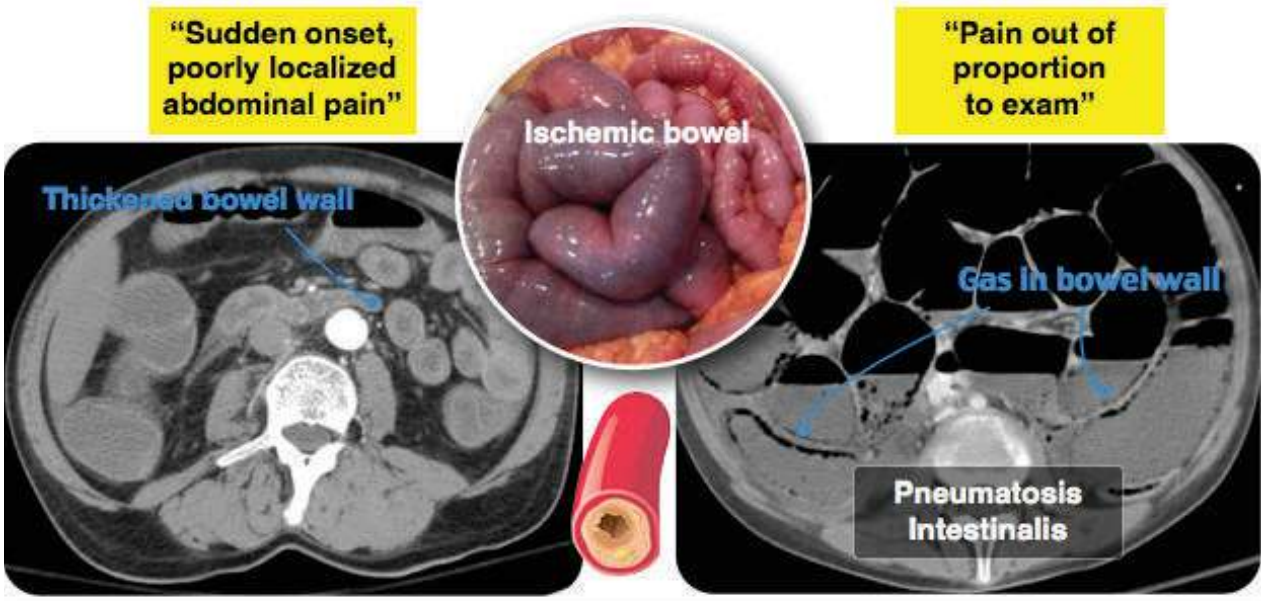
Presentation
• Abdominal pain **out of proportion** to benign abdominal exam

Diagnostic Studies
• **Lactic acidosis**
• Initially: CTA
• **Gold standard**: angiography

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### C. Acute Mesenteric Ischemia




| Mesenteric Ischemia | |
|---|---|
| **Type of occlusion** | **Predisposing factor** |
| Arterial occlusion | • Dysrhythmias (atrial fibrillation)<br>• Atherosclerotic heart disease<br>• Valvular heart disease<br>• Recent MI |
| Venous thrombosis | • History of prior thromboembolic events<br>• Hypercoagulable states |
| Nonocclusive ischemia | • Use of diuretics or vasoconstrictive medications<br>• Heart failure |

Notes


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# XXI. Jaundice

### A. Classification of Jaundice According to Mechanism

**Unconjugated hyperbilirubinemia**

**Increased bilirubin production***
- Extravascular hemolysis
- Extravasation of blood into tissues
- Intravascular hemolysis
- Dyserythropoiesis
- Wilson disease

**Impaired hepatic bilirubin uptake**
- Heart failure
- Portosystemic shunts
- Some patients with Gilbert syndrome
- Drugs (e.g. rifampin, probenecid)

**Impaired bilirubin conjugation**
- Crigler-Najjar syndrome types I and II
- Gilbert syndrome
- Neonates
- Hyperthyroidism
- Ethinyl estradiol
- Liver disease (chronic hepatitis, advanced cirrhosis)

Notes


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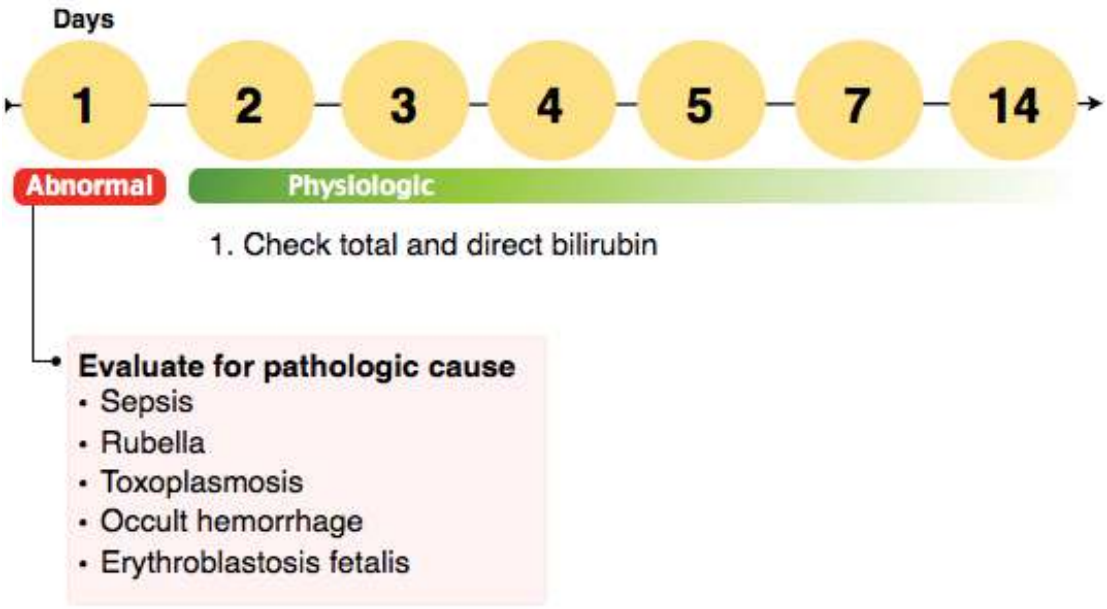
**Classification of Jaundice According to Mechanism cont.**

| Conjugated hyperbilirubinemia |
| --- |
| **Defect of canalicular anion transport**<br>• Dubin-Johnson syndrome |
| **Defect of sinusoidal reuptake of conjugated bilirubin**<br>• Rotor syndrome |
| **Extrahepatic cholestasis (biliary obstruction)**<br>• Choledocholithiasis<br>• Intrinsic and extrinsic tumors (e.g. cholangiocarcinoma)<br>• Primary sclerosing cholangitis<br>• AIDS cholangiopathy<br>• Acute and chronic pancreatitis<br>• Strictures after invasive procedures<br>• Parasitic infections (e.g. *Ascaris lumbricoides*) |
| **Intrahepatic cholestasis**<br>• Viral hepatitis<br>• Alcoholic hepatitis<br>• Nonalcoholic steatohepatitis<br>• Chronic hepatitis<br>• Primary biliary cholangitis<br>• Drugs (e.g. alkylated steroids, chlorpromazine)<br>• Sepsis and hypoperfusion states<br>• Infiltrative diseases (e.g. amyloidosis, lymphoma, sarcoidosis, TB)<br>• Total parenteral nutrition<br>• Pregnancy<br>• End-stage liver disease |
| *Serum bilirubin < 4 mg/dL in absence of underlying liver disease |

Notes


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**B. Neonatal Physiologic Jaundice**




Notes


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# XXII. Mallory-Weiss Tear

### A. Mallory-Weiss Syndrome

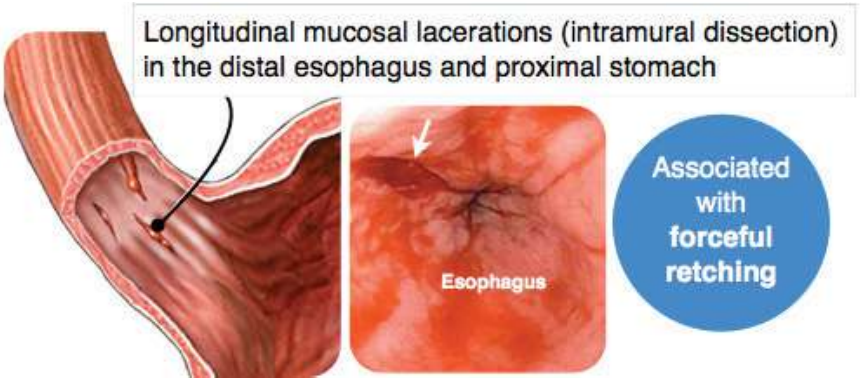
Pathophysiology
• **Incomplete longitudinal tear** in esophageal mucosa and proximal stomach

Patient
• History of **forceful vomiting**

Presentation
• **Hematemesis**

Diagnostic Studies
• Upper endoscopy

Management
• Supportive care




**Risk factors**
• Alcohol use
• Hiatal hernia
• Bulimia

**Clinical**
• Acute-onset GI bleeding (hematemesis)
• History of nonbloody emesis, retching, coughing prior to hematemesis

**Diagnosis**
• Suspected in patients with upper GI bleed and history of vomiting or retching
• Confirmed by upper endoscopy

**Management**
• Supportive
• Endoscopic therapy (active bleeding)
• Acid suppression (no active bleeding)

Notes


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# XXIII. Obstruction (Small Bowel, Large Bowel, Volvulus)

### A. Intussusception (Telescoping Bowel)

Pathophysiology
• **Most commonly** caused by a tumor or **Meckel diverticulum**

Patient
• Child **five months** to **three years old**

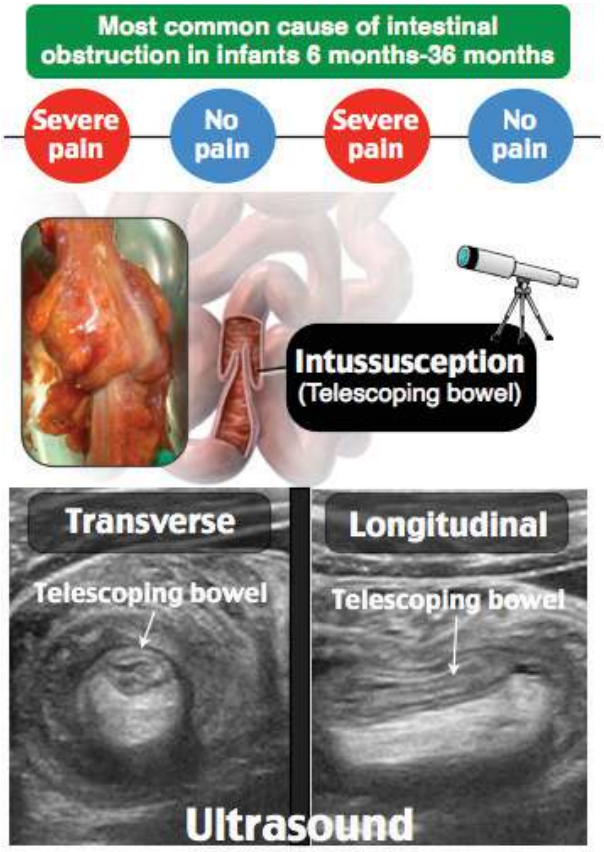
Presentation
• **Colicky abdominal pain**, vomiting, and bloody stools (**"currant jelly"**)

Diagnostic Studies
• **Ultrasound** (**"target sign"**)

Management
• **Air contrast enema**

**Abnormal lead point in children > 2 years**
• Tumor
• Meckel diverticulum
• Polyp

**Classic Triad** ▲
• Colicky abdominal pain
• Abdominal mass (sausage-shaped)
• Bloody stools (**currant jelly**)

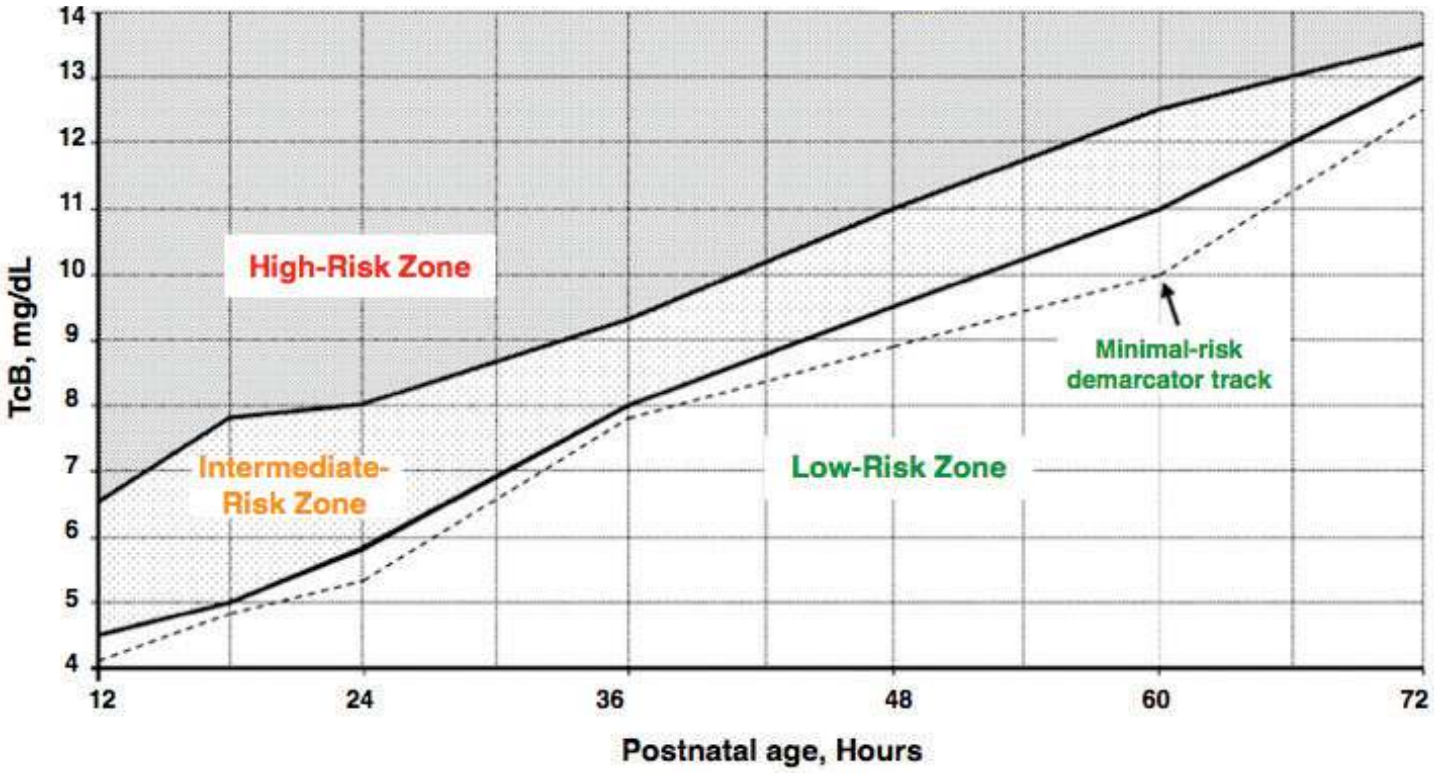



**Diagnosis & Treatment**
• Ultrasound
• Air/Contrast enema

Notes


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**B. Bilirubin Nomogram**




Notes


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## C. Small Bowel Obstruction

Pathophysiology
• **Most commonly** caused by adhesions

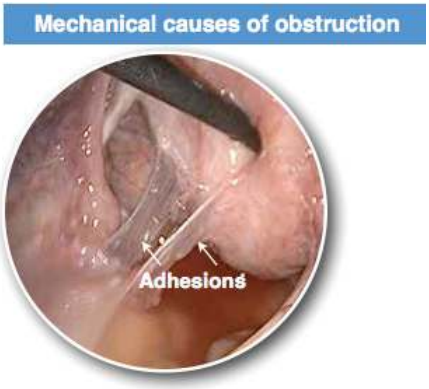
Patient
• History of prior **abdominal** or **pelvic surgery**

Presentation
• Bilious vomiting

Physical Exam
• Distended abdomen with **high-pitched** bowel tones

Diagnostic Studies
• X-ray will show **dilated loops of bowel**, **air-fluid levels**, **"stack of coins"** or **"string of pearls"** sign

Management
• NGT, surgery




**Extramural**
• **Adhesion (most common)**
• Neoplasm
• External hernia
• Abscess/phlegmon

**Mural**
• Volvulus
• Neoplasm **(most common for large bowel obstruction)**
• Crohns disease
• Radiation enteritis
• Stricture
• Hematoma
• Intussusception
• Meckels diverticulum

Notes


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## D. Large Bowel Obstruction

Pathophysiology
- **Most commonly** caused by **colorectal cancer**
- **Sigmoid colon** is **most common** location

Presentation
- Abdominal distension

Physical Exam
- High-pitched bowel tones

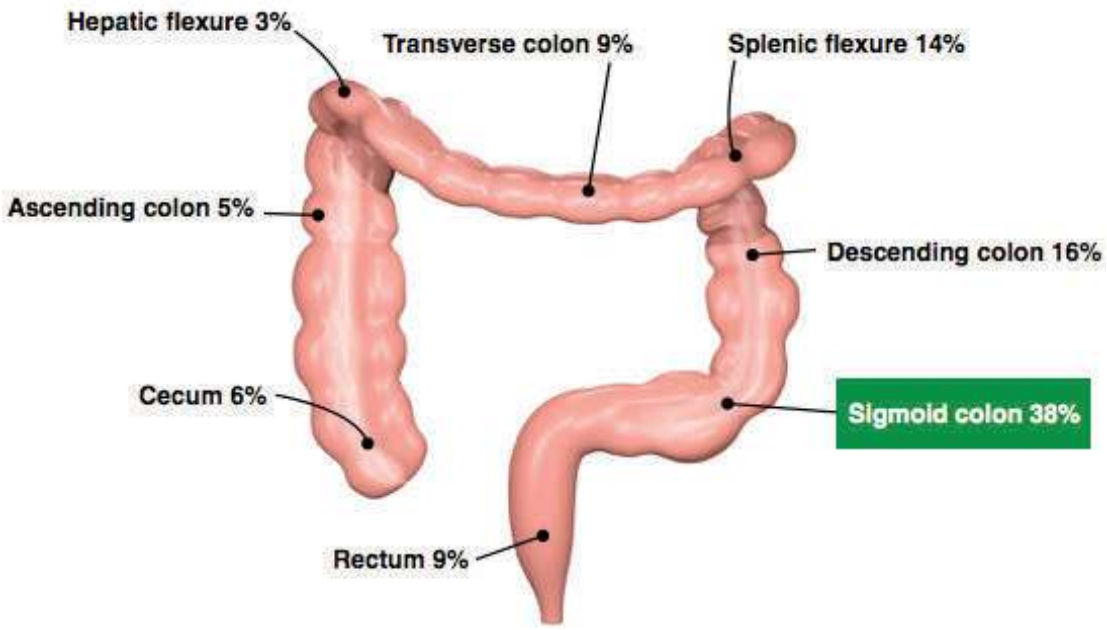
Diagnostic Studies
- Abdominal X-ray reveals **peripheral** distention and presence of **haustra**

Management
- NGT, surgery

**Etiology**
- Colorectal cancer
- Sigmoid volvulus
- Postoperative adhesions
- Strictures

**Clinical**
- Bloating
- Abdominal pain
- Obstipation

**Management**
- Supportive
- Decompression of sigmoid volvulus
- Stenting of malignant obstruction
- Surgical




Notes


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### E. Sigmoid Volvulus

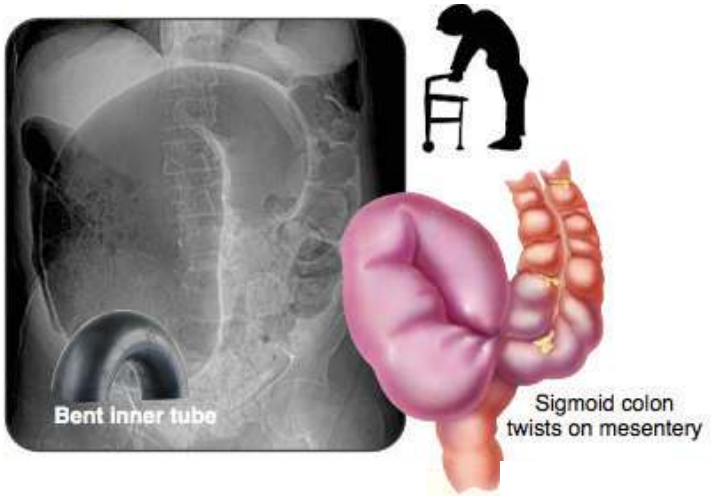
Patient
• Elderly, bedridden patient or patient with psychiatric and neurologic history
• History of constipation

Management
• Sigmoidoscopy (therapeutic **and** diagnostic)

**Management**
• Flexible sigmoidoscopy (to reduce volvulus)
• Surgery (to prevent recurrence)

**Diagnosis**
• Plain film (low specificity) [U-shaped, bent inner tube]
• Abdominal CT scan
• Contrast enema

**Risk factors**
• Nursing home patients
• Elderly
• Bedbound
• Chronic constipation

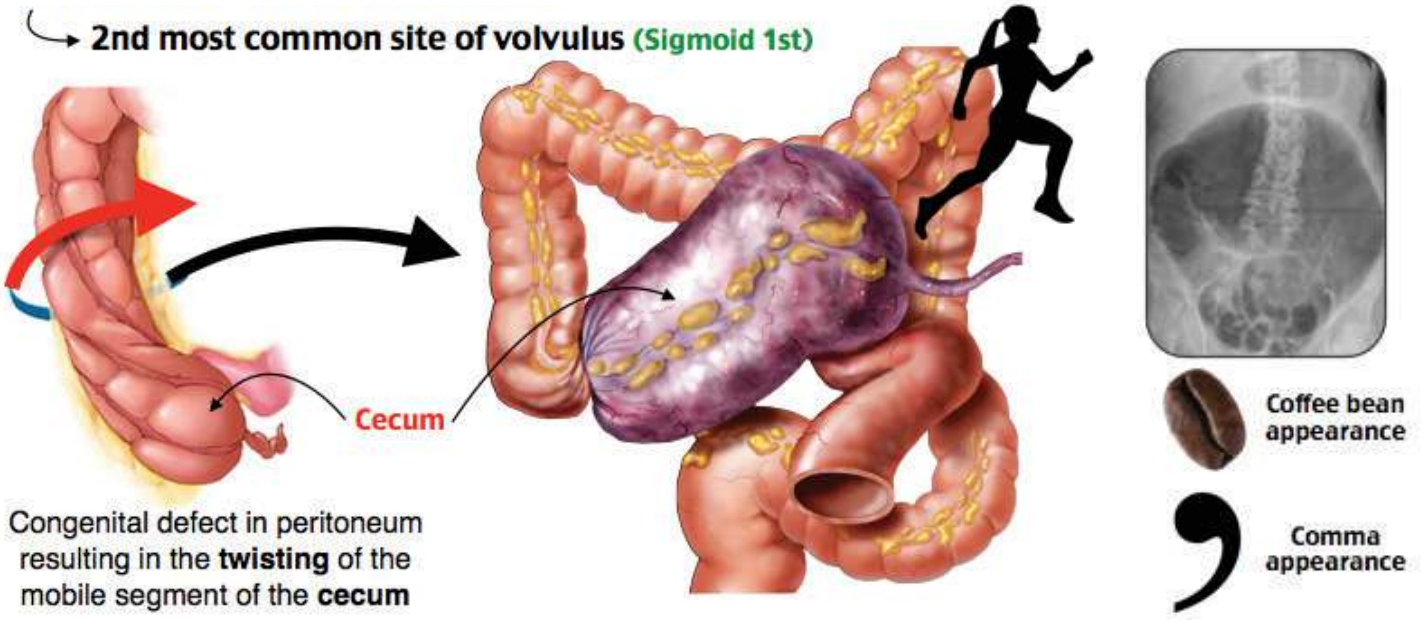



**Clinical**
• Insidious onset of slowly progressive abdominal pain
• Abdominal distension
• Nausea, constipation
• Vomiting (several days after pain onset)

Notes


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### F. Cecal Volvulus




**Risk factors**
- Relatively **younger** than sigmoid volvulus (30s-50s)
- Associated with **marathon runners**
- Increased in GI malignancy

**Diagnosis**
- Plain film (coffee-bean or comma cecum) [Low specificity]
- Abdominal CT (90% of patients) **[Whirl sign]**
- Surgical exploration (10% of patients)

**Management**
- Surgical

Notes


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# XXIV. Peptic Ulcer Disease

## A. Peptic Ulcer Disease

Pathophysiology
• **Most commonly** caused by ***Helicobacter pylori*** infection or NSAID use

Presentation
• Gnawing epigastric pain
  • Duodenal ulcer: pain is **alleviated** by ingesting food ("**DUD**e give me food")
  • Gastric ulcer: pain is **exacerbated** by ingesting food

Diagnostic Studies
• *H. pylori*: **fecal antigen** or **urea breath test**

Management
• Eradication of *H. pylori* with **triple therapy** (omeprazole, clarithromycin, amoxicillin)
• Greater than 20% clarithromycin-resistant *Helicobacter pylori*, **quadruple** therapy with tetracycline, metronidazole, a proton-pump inhibitor, and bismuth salts is indicated

Comments
• **Most common** cause of upper GI bleeding

**Complications**
• Bleeding
• Gastric outlet obstruction
• Penetration and fistulization
• Perforation

**Most common cause of upper GI bleed**



**Gastric ulcer**
• Early satiety
• Pain immediately after meals

**Duodenal ulcer**
• Awakens patient at night
• Pain relieved by food
• Postprandial pain (1 to 2 hr delay)
• More common than gastric

Notes


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CHAPTER 4

# Pulmonology

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CHAPTER 4

# Pulmonology


189 **ACUTE BRONCHIOLITIS**

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198 **CROUP**

199 **FOREIGN BODY ASPIRATION**

201 **HEMOPTYSIS**

202 **INFLUENZA**

203 **LUNG CANCER**

204 **PERTUSSIS**

205 **PLEURITIC CHEST PAIN**

207 **PNEUMONIA**
Bacterial
Viral
Fungal
Human immunodeficiency virus-related

216 **PULMONARY EMBOLISM**

218 **RESPIRATORY SYNCYTIAL VIRUS**

219 **SHORTNESS OF BREATH**

220 **TUBERCULOSIS**



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# I. Acute Bronchiolitis

**Acute Bronchiolitis**

Pathophysiology
• **Most commonly** caused by **respiratory syncytial virus** (**RSV**)

Patient
• Children **< 2 years old**

Presentation
• Difficulty breathing

Physical Exam
• Respiratory distress, **polyphonic wheezing**, and rales

Diagnostic Studies
• Diagnosis is established by history and physical exam

Management
• Supportive care




**Microbiology**
• Respiratory syncytial virus (RSV) [most common]
• Rhinovirus, parainfluenza virus, human metapneumovirus
• Influenza virus

**Clinical**
• Occurs primarily in children **≤ 2 years old**
• Often preceded by a 1 to 3 day URI prodrome
• Fever, cough
• Respiratory distress (tachypnea, retractions, wheezing, crackles)

**Complications**
• Dehydration
• Apnea (premature and < 2 months old)
• Aspiration pneumonia, respiratory failure

Notes

**Management**

*Nonsevere bronchiolitis*
• Anticipatory guidance, nasal suctioning, hydration

*Severe bronchiolitis*
• Trial of inhaled bronchodilator (controversial)
• Heated humidified high-flow nasal cannula
• Continuous positive airway pressure
• Endotracheal intubation


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# II. Acute Bronchitis

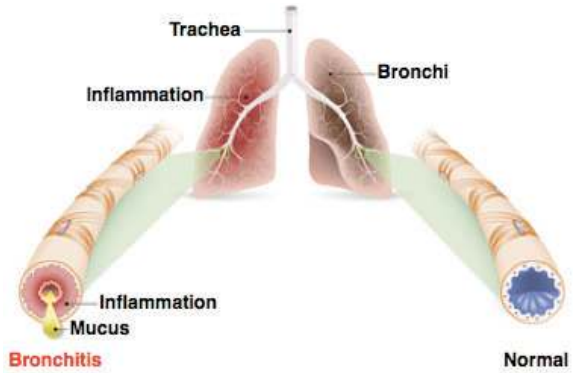
**Acute Bronchitis**

Pathophysiology
• **Most commonly** caused by viruses

Patient
• Productive **cough** for longer than five days

Management
• Symptomatic care and bronchodilators

Comments
• **Most common** cause of **minor hemoptysis**
• Routine antibiotic therapy not indicated




**Etiology**

*Viral (majority)*
• Influenza A and B
• Parainfluenza
• Coronavirus
• Rhinovirus
• Respiratory syncytial virus
• Human metapneumovirus

*Bacterial (minority)*
• *Bordetella pertussis*
• *Mycoplasma pneumoniae*
• *Chlamydia pneumoniae*

**Clinical**
• Cough (lasts ≥ 5 days)
• Purulent or nonpurulent sputum
• URI may precede onset of bronchitis

Notes


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# III. Acute Epiglottitis

**Epiglottitis**

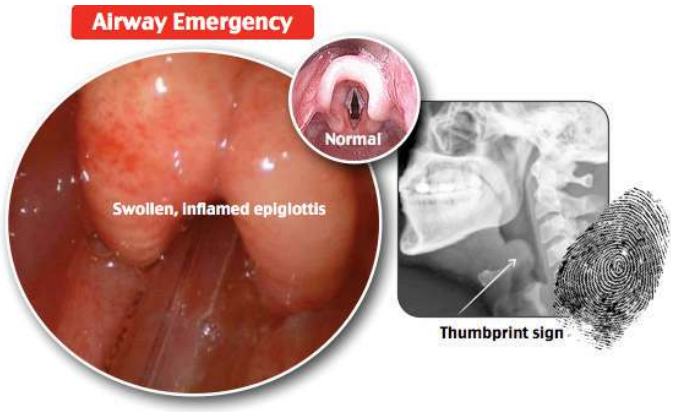
Pathophysiology
• **Most commonly** caused by *Strep.* and *Staph.* species, ***H. influenzae*** (prior to the Hib vaccine)

Presentation
• **Rapid onset** of **fever** and **dysphagia**

Physical Exam
• Patient **leaning forward**, **drooling**, **inspiratory stridor**

Diagnostic Studies
• **"Thumbprint sign"** on lateral neck X-ray

Management
• IV antibiotics and airway management in OR




**Etiologies**
• *Haemophilus influenzae* type B (pre HiB vaccine)
• *Streptococcus* spp.
• *Staph. aureus*
• *Moraxella catarrhalis*

**Clinical (Rapid onset)**
• Fever
• Sore throat
• Drooling
• Muffled voice
• Anxious
• Ill-appearing

Notes


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# IV. Acute Respiratory Distress Syndrome

### A. Acute Respiratory Distress Syndrome (ARDS)

Pathophysiology
- **Diffuse alveolar damage** leads to **increased alveolar capillary permeability**, causing **noncardiogenic pulmonary edema**
- **Sepsis** leading to ARDS is more common than gastric aspiration or shock

Physical Exam
- Hypoxemia refractory to $O_2$

Diagnostic Studies
- Chest X-ray reveals **bilateral** infiltrates
- PA wedge pressure < 18
- $PaO_2$: $FiO_2$ < 300 mm Hg
- **Increased** A-a gradient

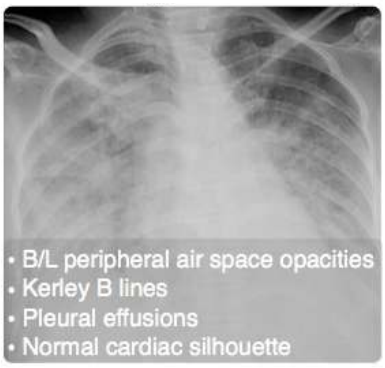
Management
- Mechanical ventilation: **PEEP**, low tidal volumes (6 mL/kg)

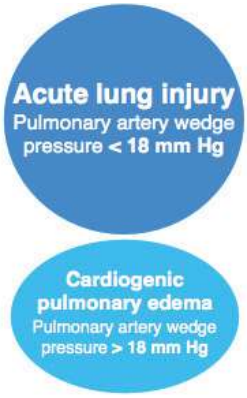
Notes


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### B. Noncardiogenic Pulmonary Edema

Radiographic evidence of **alveolar fluid accumulation** without hemodynamic evidence to suggest cardiac etiology




**Etiology**
- Acute respiratory distress syndrome (ARDS)
- High altitude (HAPE) and neurogenic pulmonary edema
- Opioid overdose
- Pulmonary embolism
- Eclampsia
- Transfusion-related lung injury (TRALI)

**Management**
- Treat underlying condition
- ABX for infection
- Supporitve care
- Mechanical ventilation
- Adequate nutrition
- Hemodynamic monitoring

Notes


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### C. Infant (Neonatal) Respiratory Distress Syndrome (Hyaline Membrane Disease)

Pathophysiology
• Caused by **surfactant deficiency**

Patient
• **Premature** infant

Presentation
• Respiratory difficulty within a few **hours** after birth

Physical Exam
• Tachypnea, tachycardia, cyanosis, nasal flaring, expiratory grunting, and chest wall retractions

Diagnostic Studies
• Chest X-ray will show **decreased lung volume**, air-bronchograms, and a **ground-glass appearance**

Management
• Oxygen, intubation, CPAP, **surfactant**

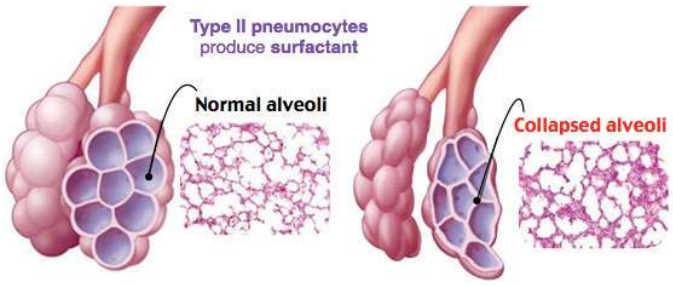
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## D. Respiratory Distress Syndrome

**Deficient surfactant in the lining of the alveoli (premature infants)**

Surfactant **GRADUALLY** increases until **33-36 weeks** gestation
**After 36 weeks,** there is a **SURGE** in surfactant




| INCREASED Risk | DECREASED Risk |
|---|---|
| • Diabetic mother<br>• C-section delivery<br>• Birth asphyxia | • Prolonged rupture of membranes<br>• Prenatally administered steroids |

**Clinical**
- Tachypnea
- Nasal flaring
- Expiratory grunting
- Retractions

**Management**
- Mechanical ventilation
- Exogenous surfactant

Infants with Respiratory Distress Syndrome that require **prolonged ventilator support** are at risk for **bronchopulmonary dysplasia**

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# V. Asthma

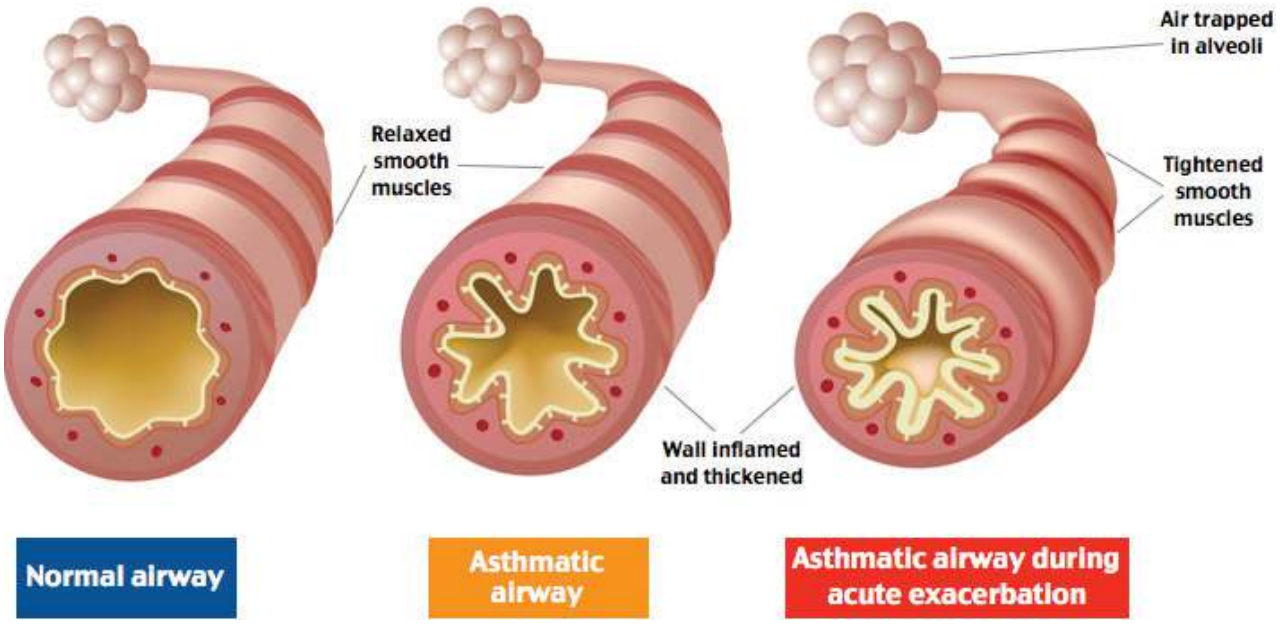
## A. Asthma

Pathophysiology
• Airway inflammation, bronchial hyper-responsiveness, and **reversible** airflow obstruction

Patient
• In exacerbations: recent exposure to allergens or cigarette smoke

Diagnostic Studies
• PEF < 50%: severe exacerbation

Management
• $O_2$: maintain $SpO_2$ > **88%**
• Beta-agonists: **increased** cAMP leads to bronchodilation
• Anticholinergics: **decrease** bronchoconstriction
• Corticosteroids: **decrease** inflammation, administer early
• Mg: severe exacerbations
• Noninvasive ventilation: **decrease** work of breathing
• Mechanical ventilation:
  • Objective: maximize expiratory time
  • **Low** respiratory rate
  • **High** inspiratory flow rate
  • Maintain plateau pressure < 30 cm $H_2O$
  • Permissive hypercapnia to avoid breath stacking




Notes


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### B. Pharmacologic Management of Acute Asthma Exacerbations

| | |
|---|---|
| **Beta-2 agonist bronchodilator** (nebulized or metered-dose inhaler) | • Albuterol by MDI 4 to 8 puffs every 20 minutes up to 1 hour, then every 1 to 4 hours, prn<br>• Albuterol by nebulizer 0.083 percent (2.5 mg/3 mL), 2.5 to 5 mg every 20 minutes for 3 doses and then 2.5 to 5 mg every 1 to 4 hours, prn<br>• Albuterol by continuous nebulization, administering 10 to 15 mg per hr |
| **Ipratropium** | • By nebulizer, 500 mcg every 20 minutes for 3 doses, then prn. Can be given simultaneously with beta-2 agonist.<br>• By MDI, 4 to 8 inhalations every 20 minutes for 3 doses, then prn |
| **Systemic glucocorticoids** (for those with a poor response to treatment after one hour or with initial therapy for patients on chronic oral glucocorticoids) | • For patients who can be managed at home: prednisone 40 to 60 mg per day in a single or divided dose<br>• For patients who require hospitalization: prednisone 40 to 80 mg daily in a single or divided dose (or the equivalent dose of methylprednisolone intravenously) until peak flow reaches 70% of predicted or personal best, and then taper as patient improves |
| **For patients not responding to above therapies, consider adjunct therapies** | • Intravenous magnesium sulfate 2 g infused over 20 minutes, in absence of renal insufficiency<br>• Subcutaneous terbutaline 0.25 mg every 20 minutes for up to 3 doses |

Notes


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# VI. Croup

**A. Laryngotracheitis (Croup)**

Pathophysiology
• **Most commonly** caused by ***Parainfluenza virus***

Patient
• Non-toxic appearing child, **six months** to **three years old**

Presentation
• URI symptoms with barky, **seal-like cough**, **inspiratory stridor**, low-grade fever
• Symptoms are improved when patient is brought outside into cool air

Diagnostic Studies
• X-ray will show **"steeple sign"** on PA view

Management
• **Dexamethasone**, **racemic epinephrine** (for severe cases)






**Clinical**
• URI-like symptoms
• Barking, Seal-like cough
• Stridor
• Symptoms typically worse at night
• Hypoxia is uncommon

**Management**
• Supportive care
• Corticosteroids (dexamethasone)
• Racemic epinephrine (for moderate to severe cases)

Notes





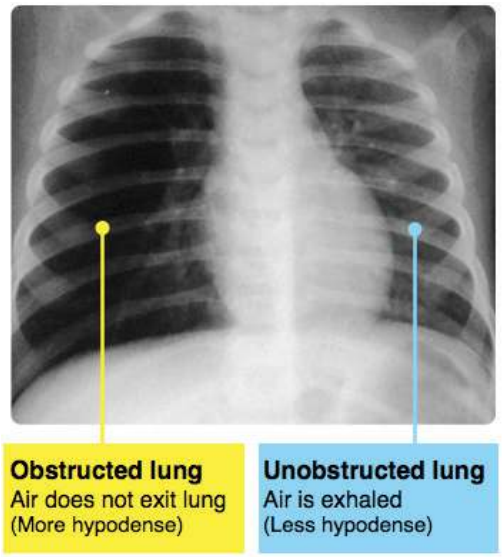
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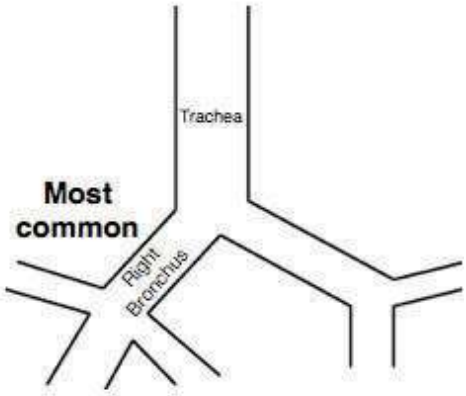
# VII. Foreign Body Aspiration

### A. Foreign Body Aspiration

Pathophysiology
• **Most common** site is the **right main bronchus**

**Foreign Body Aspiration**
**On expiration, foreign body obstructs bronchus (Decreased diameter of bronchi)**



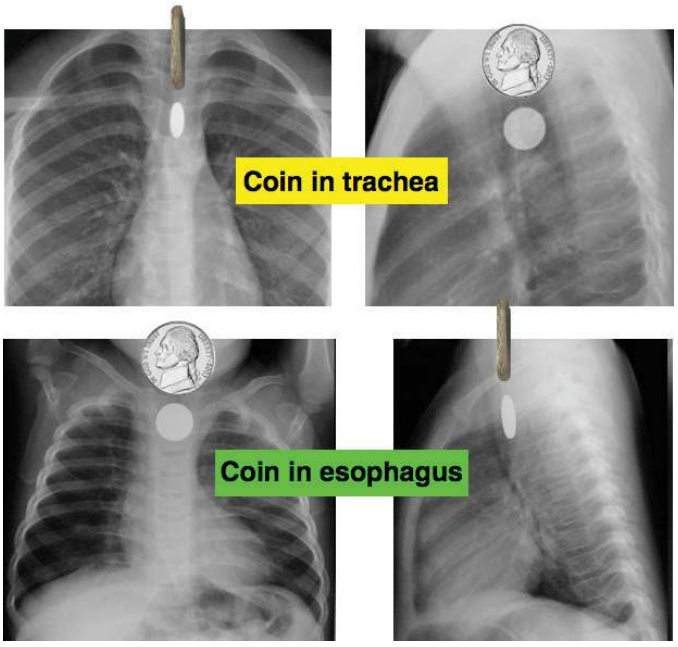




Patient
• Child, usually when not observed by caregiver

Presentation
• **Sudden** episode of coughing, wheezing, or stridor

**Clinical**
• Most children present with partial airway obstruction
• Cough, tachypnea, stridor
• Monophonic wheezing or decreased air entry
• Sudden episode of coughing or choking

**Management**
• Supportive
• Immediate removal
• Rigid or flexible bronchoscopy




Notes


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### B. Foreign Body Management

| Foreign Body Type | Endoscopic Removal | Surgical Removal | Imaging Follow-up |
|---|---|---|---|
| **Sharp foreign body** | • Emergency endoscopic removal if in esophagus<br>• Urgent endoscopic removal if in stomach | • If symptomatic and beyond the reach of endoscopy or too dangerous to remove endoscopically | • Daily radiograph up to 3 days |
| **Blunt foreign body** | Urgent endoscopic removal if:<br>• In esophagus<br>• FB is > 6 cm in length and proximal to duodenum<br>• FB is > 2.5 cm in width and proximal to duodenum<br>• FB fails to pass through pylorus after 3-4 wks | • If immobile and distal to the duodenum for > 1 wk<br>• If symptomatic and distal to duodenum | • Weekly radiograph up to 4 wks |
| **Coins** | • If asymptomatic, observe for 12-24 hrs before considering endoscopic removal | • If immobile and distal to duodenum for > 1 wk | • Weekly radiograph up to 4 wks |
| **Batteries** | • Emergency endoscopic removal if button battery in esophagus<br>• If > 2 cm battery remains in stomach for > 48 hrs | • If immobile or symptomatic and beyond reach by endoscopy | • Once past gastroesophageal jct., initial radiograph at 48 hrs<br>• Once past pylorus, repeated radiographs every 3-4 days |
| **Magnets** | • Urgent endoscopic removal of all magnets within endoscopic reach | • If immobile on serial radiographs and beyond reach of endoscopy<br>• If symptoms of obstruction or perforation | • Close follow up with frequent serial radiographs to ensure mobility |
| **Illicit drug packets** | • Not recommended due to risk of rupture | • If symptomatic from ruptured drug packet<br>• If failure to progress | • CT with IV contrast helpful if plain radiographs negative |

Notes


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# VIII. Hemoptysis

## A. Hemoptysis

Pathophysiology
- Expectoration of blood
- Can be **"massive"** (≥ 500 mL) or simply blood-tinged sputum

Diagnostic Studies
- **Chest X-ray** most important initial study

Comments
- **Most common** source is **bronchial arteries**

## B. Causes of Hemoptysis

BATTLE CAMP

**B**ronchitis

**B**ronchiectasis

**A**spergilloma

**T**umor

**T**uberculosis

**L**ung abscess

**E**mboli (pulmonary)

**C**oagulopathy

**A**utoimmune disorders

**A**V malformation

**A**lveolar hemorrhage

**M**itral stenosis

**P**neumonia

Notes


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# IX. Influenza

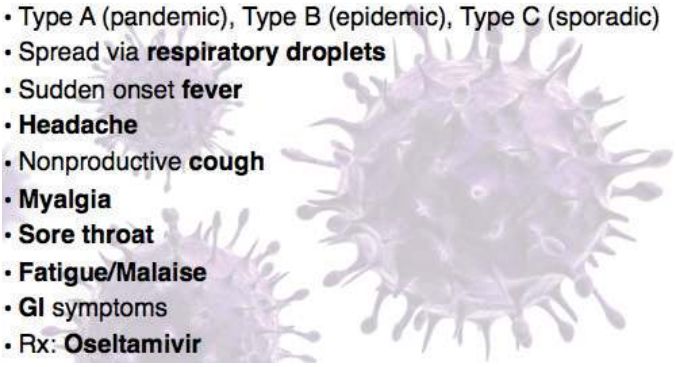
**Influenza**

Presentation
• **Sudden-onset fever**, headache, cough, **myalgia**, sore throat, **fatigue**

Diagnostic Studies
• Diagnosis can be made clinically and confirmed with reverse-transcription polymerase chain reaction (**RT-PCR**) or **viral culture**

Management
• Supportive care
• **Oseltamivir** for high-risk patients if diagnosis is made **within 48 hours** of symptom onset, otherwise this provides no clinical benefit

Comments
• **Most common** cause of viral pneumonia in adults




Notes


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# X. Lung Cancer

## A. Lung Cancer

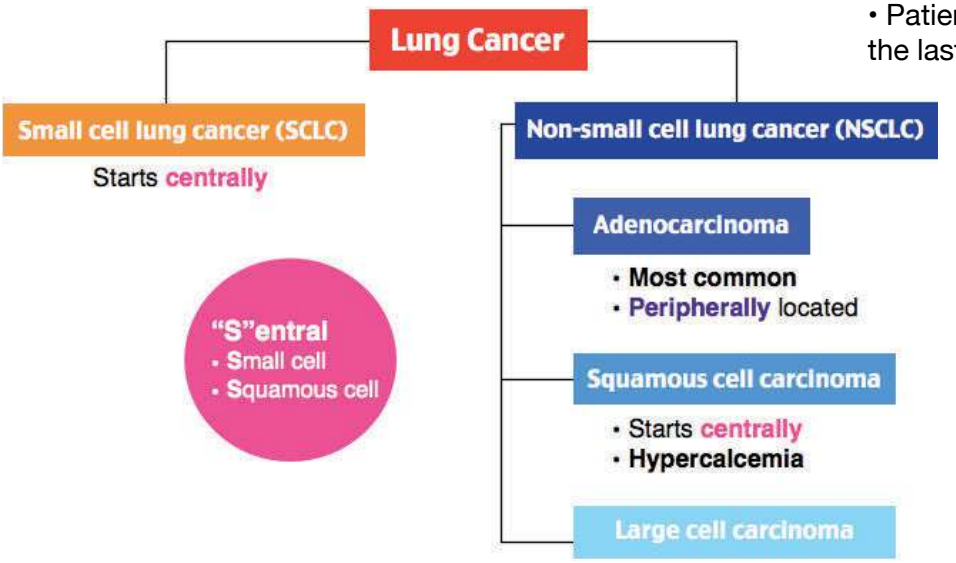
Pathophysiology
• **Most common** type is **non-small cell** (adenocarcinoma)

Patient
• History of **smoking**

Presentation
• **Most common** presenting symptom is **cough**
• Hemoptysis, dyspnea, chest pain
• Nonpulmonary symptoms that suggest metastases:
  • hip and back pain
  • **Horner syndrome** (ipsilateral ptosis, anhidrosis, and miosis)
  • neurologic symptoms
  • hypotension with tachycardia

Diagnostic Studies
• Chest X-ray prompts a CT scan, and **biopsy** will confirm
• **Hypercalcemia**, exudative effusion

Comments
• Leading cause of cancer-related death among men and women
• Screening with low-dose helical CT scan (American Cancer Society)
  • No screening recommended for normal risk patients
  • High-risk patients meet all of the following criteria:
    • 55–74 years of age, in good health
    • 30 pack-year smoking history (packs of cigarettes smoked per day x number of years the person has smoked)
    • Patient is currently smoking or quit within the last 15 years




Notes


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# XI. Pertussis

**Pertussis (Whooping Cough)**

Pathophysiology
• **Most commonly** caused by ***Bordetella pertussis***

Patient
• History of nasal congestion, cough, and low-grade fever

Presentation
• **"Rapid fire," repetitive coughing** followed by an **inspiratory "whoop"** and **post-tussive emesis**

Management
• Macrolide (**azithromycin**)



***Bordetella pertussis***

1. **Catarrhal phase (lasts 1 to 2 weeks)**
   - Mild fever, cough, rhinorrhea
   - Conjunctival injection
   - Excessive lacrimation
2. **Paroxysmal phase (lasts 2 to 6 weeks)**
   - Begins during second week of illness
   - Paroxysmal cough **("whooping" cough)**
   - Inspiratory stridor
3. **Convalescent phase (lasts 1 to 2 weeks)**
   - Gradual reduction in symptoms

**Treatment**
*Macrolides*
• Erythromycin
• Azithromycin (preferred in infants < 1 m)
• Clarithromycin

Notes


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# XII. Pleuritic Chest Pain

### A. Pleural Effusion

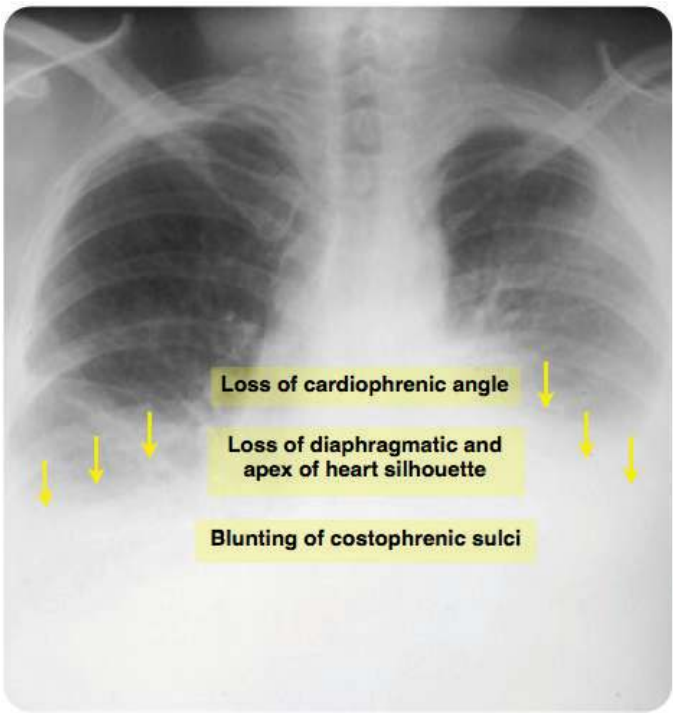
Pathophysiology
- **Most commonly** caused by:
  - Transudative: **CHF**
  - Exudative: **infection** (bacterial pneumonia) more often than malignancy and pulmonary embolism

Physical Exam
- **Decreased** breath sounds, dullness to percussion, **decreased** tactile fremitus

Diagnostic Studies
- Chest X-ray will show **blunting** of the **costophrenic angle**

Management
- Thoracentesis

Comments
- **Light criteria** are used to differentiate between transudative and exudative effusions




Notes


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**B. Light Criteria**

| | Transudate | Exudate |
|---|---|---|
| **Pleural:Serum Protein** | < 0.5 | ≥ 0.5 |
| **Pleural:Serum LDH** | < 0.6 | ≥ 0.6 |
| **Pleural fluid LDH** | < 2/3 upper limit of normal | > 2/3 upper limit of normal |
| **Main Causes** | • Heart failure<br>• Cirrhosis<br>• Nephrotic syndrome<br>• Pulmonary embolism | • Malignancy<br>• Bacterial/Viral pneumonia<br>• Tuberculosis<br>• Pulmonary embolism<br>• Pancreatitis<br>• Esophageal rupture<br>• Collagen vascular disease<br>• Chylothorax/Hemothorax |

Notes


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# XIII. Pneumonia

BACTERIAL • VIRAL • FUNGAL • HUMAN IMMUNODEFICIENCY • VIRUS-RELATED

## A. Bacterial Pneumonia

Pathophysiology
• ***S. pneumonia***: **most common**, rust-colored sputum, rigors, gram-positive paired lancets
• *Klebsiella*: **alcoholics**, **currant jelly sputum**, bulging fissures
• *S. aureus*: **IVDA**, post-influenza, elderly, gram-positive cocci in clusters
• *H. influenzae*: COPD, **gram-negative pleomorphic rods**
• *Pseudomonas*: **cystic fibrosis**, nursing home resident, and cyanosis
• Healthcare-associated pneumonia: *Pseudomonas*, MRSA

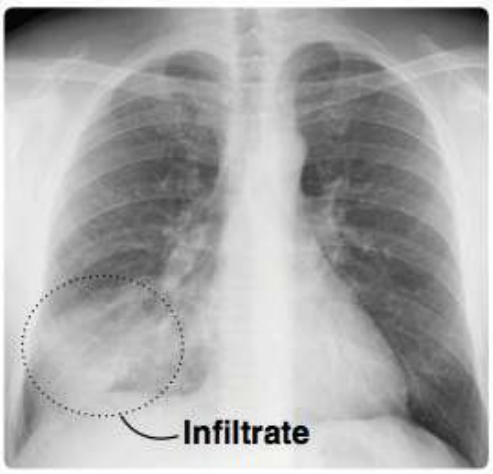
Management
• Outpatient, healthy: macrolide (**azithromycin**) or doxycycline
• Outpatient, comorbidity: respiratory tract fluoroquinolone (RTF)
• Inpatient: RTF
• ICU: antipneumococcal beta-lactam (ceftriaxone or cefotaxime) **and** either azithromycin or an RTF

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## B. Community-Acquired Pneumonia

An acute infection of the pulmonary parenchyma in a patient who has acquired the infection in the community




**Clinical**
- Cough, fever, pleuritic chest pain, sputum production
- Tachypnea, tachycardia
- Crackles, decreased bronchial breath sounds
- Dullness to percussion, tactile fremitus, egophony

**Microbiology**

Typical
- *Streptococcus pneumoniae*
- *Haemophilus influenzae*
- *Staphylococcus aureus*
- Group A streptococci
- *Moraxella catarrhalis*
- Anaerobes
- Aerobic gram-negative bacteria

Atypical
- *Legionella spp*
- *Mycoplasma pneumoniae*
- *Chlamydophila pneumoniae*
- *Chlamydia psittaci*

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## C. Atypical Pneumonia



**Atypical Pneumonia**
**(aka walking pneumonia)**

Pathophysiology
• ***Mycoplasma pneumoniae***: **most common**, young patients
• *Legionella*: smokers, **aerosolized water**, air travel, **diarrhea**, **hyponatremia**
• *Chlamydophila pneumoniae*: close quarters outbreaks, young, follows pharyngitis
• *Coxiella burnetii*: **livestock exposure**, increased LFTs
• *Chlamydophila psittaci*: **bird exposure**, hyperpyrexia, severe HA

**Organisms**

• *Chlamydophila pneumoniae* (Mild pneumonia)
• *Chlamydophila psittaci* (Psittacosis)
• *Coxiella burnetii* (Q fever)
• *Francisella tularensis* (Tularemia)
• *Legionella pneumophila* (Legionnaires disease)
• *Mycoplasma pneumoniae* (Pneumonia in young age groups)

Presentation
• Gradual onset of **dry cough**, dyspnea, and **extrapulmonary symptoms** such as headache, myalgias, fatigue, and GI disturbance

**Clinical**

• No response to beta-lactams and sulfonamides
• Patient "looks better" than clinical picture
• No signs of lobar consolidation
• Absence of leukocytosis
• Presence of extrapulmonary symptoms

Physical Exam
• **Rales**

Diagnostic Studies
• Chest X-ray will show **interstitial infiltrate**

**Treatment**

• Tetracyclines (doxycycline, tetracycline)
• Macrolides (azithromycin, clarithromycin)
• Fluoroquinolones

Management
• **Azithromycin**

Notes


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PNEUMONIAS: FUNGAL

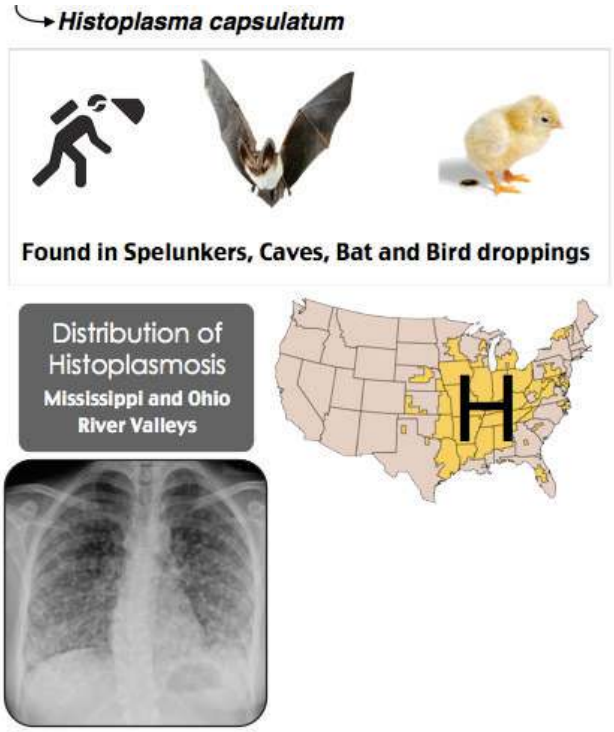
### A. Histoplasmosis

Patient
• History of travel to **Ohio** or **Mississippi river valleys** and exposure to **bird** or **bat droppings**

Diagnostic Studies
• Chest X-ray will show **solitary pulmonary calcification**, hilar and mediastinal adenopathy
• Sputum culture is diagnostic

Management
• **Itraconazole** or amphotericin B

**Histoplasmosis**



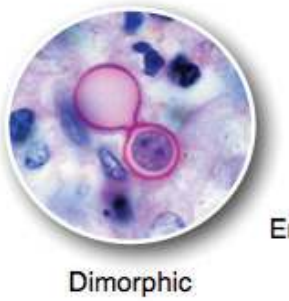

**Clinical**
• Most patients remain asymptomatic
• Disseminated infection more common in immunocompromised
• Primary infection: Flu-like
• Chronic infection: TB-like

**Treatment**
• Antifungal

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## B. Blastomycosis



Dimorphic






**Pneumonia (most common)**
- Alveolar infiltrates or mass lesion
- Miliary or reticulonodular also possible
- Small pleural effusions
- Chronic pneumonia



**Skin (Second most common)**
- Verrucous lesion
- Irregular borders
- Gray to violet in color
- May mimic squamous cell carcinoma

**Treatment**
- Amphotericin B (moderate to severe disease)
- Itraconazole (mild to moderate)

Notes

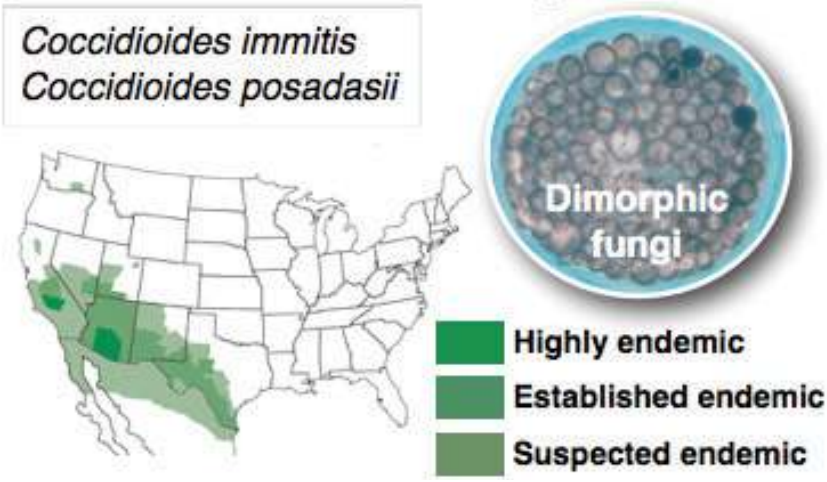


**Osteomyelitis (Third most common)**
- Soft tissue swelling
- Chronic draining sinus tract
- Well-circumscribed osteolytic lesion

**Diagnosis**
- Culture
- Histopathology
- Serologic testing
- PCR


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### C. Coccidioidomycosis




**Clinical**
- Acute pulmonary infection (most common)
- Chest pain, cough, dyspnea, hemoptysis, fever, fatigue
- Alveolar infiltrates
- Hilar and mediastinal lymphadenopathy
- Headache, pharyngitis, arthralgia, erythema nodosum
- Dissemination possible

**Treatment**
- Fluconazole, itraconazole (only those with severe disease)
- > 10% loss of body weight
- Night sweats persisting > 3 weeks
- Infiltrates involving > half of one lung or portions of both lungs
- Prominent or persistent hilar lymphadenopathy
- Anticoccidioidal complement fixing antibody concentration > 1:16
- Inability to work
- Symptoms persisting > 2 months

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## PNEUMONIAS: HIV-RELATED

### A. Pneumocystis Pneumonia

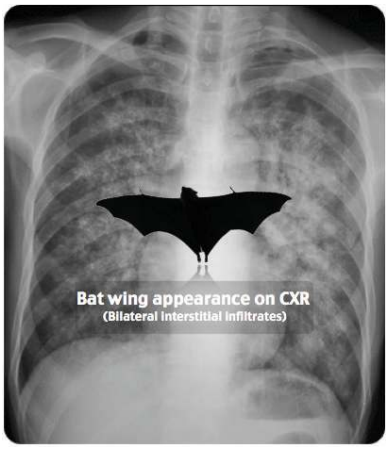
Pathophysiology
• **Most commonly** caused by ***Pneumocystis jirovecii***

Patient
• History of **HIV**

Presentation
• **Gradual onset** of **nonproductive cough**

Diagnostic Studies
• **CD4 < 200**, **increased LDH**
• Chest X-ray will reveal bilateral infiltrates (**bat wing pattern**)

Management
• **TMP-SMX**, **steroids for PaO2 < 70**




**Clinical**
• Caused by *Pneumocystis jirovecii* (formerly *carinii*)
• Immunocompromised host (HIV, transplant)
• Seen in HIV positive patients with CD4 < 200
• Symptoms progress over 2-3 weeks
• Fever, dry cough
• Oxygen desaturation common with ambulation
• LDH is often elevated

**Treatment**
• Steroids for $PaO_2$ < 70 or A-a gradient > 35
• Trimethoprim-sulfamethoxazole (TMP-SMX)

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PNEUMONIAS: VIRAL

Most commonly caused by **Influenza**

### A. Spontaneous Pneumothorax

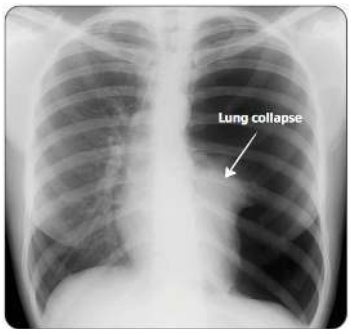
Patient
• Young, tall, thin man

Physical Exam
• **Unilateral decreased** breath sounds, **decreased** fremitus, **hyperresonance** to percussion

Diagnostic Studies
• Chest X-ray will show the **absence of lung markings** along lung periphery

Management
• < 20% in a healthy patient: **observation** with oxygen administration
• > 20%: **chest tube thoracostomy**

### B. Pneumothorax




**Spontaneous**
*Primary* – no clinically apparent lung disease (tall, thin males)
*Secondary* – patients with underlying lung disease (COPD, TB)

**Iatrogenic**
Result of diagnostic (e.g., throracentesis) or therapeutic (e.g., central venous line) procedure

**Traumatic**
Result of blunt or penetrating trauma

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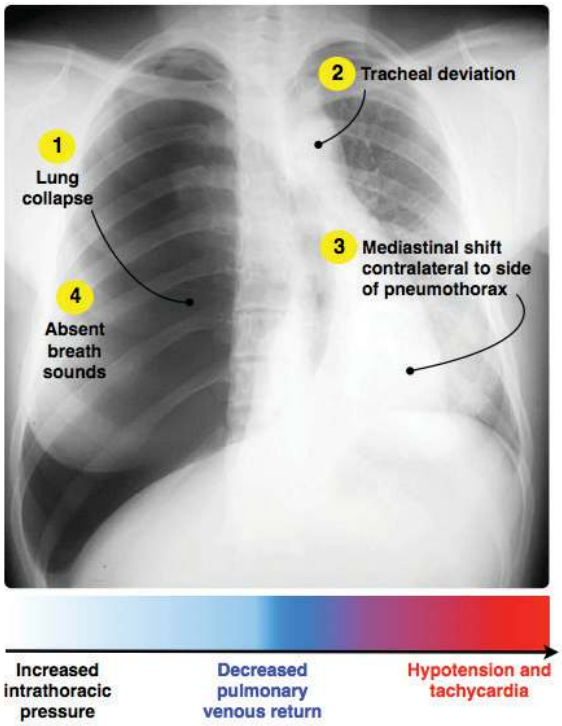
### C. Tension Pneumothorax

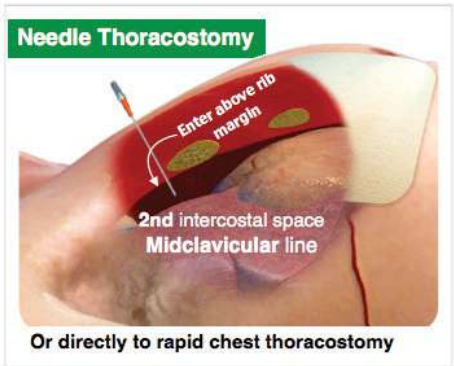
Patient
• History of chest **trauma**

Physical Exam
• **Unilateral diminished** or absent breath sounds, **tracheal deviation away** from the side of the injury, **hypotension**, jugular venous distension

Diagnostic Studies
• Diagnosis is made clinically

Management
• **Needle decompression** of the chest in the **second intercostal space** in the **midclavicular line** followed by chest tube insertion




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# XIV. Pulmonary Embolism

## A. Pulmonary Embolism

Pathophysiology
• **Most commonly** originates in the **lower extremities** and pelvis
• **Virchow triad**: venous stasis, endothelial trauma, and hypercoagulability

Patient
• History of long air travel, trauma to a lower extremity, malignancy

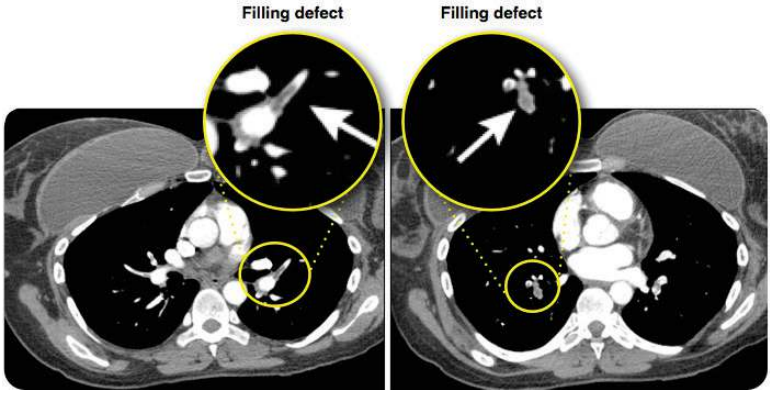
Presentation
• **Most common** presenting symptom is dyspnea

Physical Exam
• **Most common** sign is **tachypnea**
• ECG will show sinus tachycardia (**most common** abnormal finding), nonspecific ST segment and T wave changes, right heart strain, **S1Q3T3** (classic finding)

Diagnostic Studies
• Chest X-ray will show nonspecific abnormalities: **Hampton hump** (pleural-based wedge infarct), **Westermark sign** (vascular cutoff sign)
• **CT pulmonary angiography** is the test of choice in moderate to high probability cases

Management
• Anticoagulation (heparin, LMWH) and supportive care
• Thrombolytics in hemodynamically unstable patients

Comments
• In **low** clinical suspicion, a **negative D-dimer excludes pulmonary embolism**

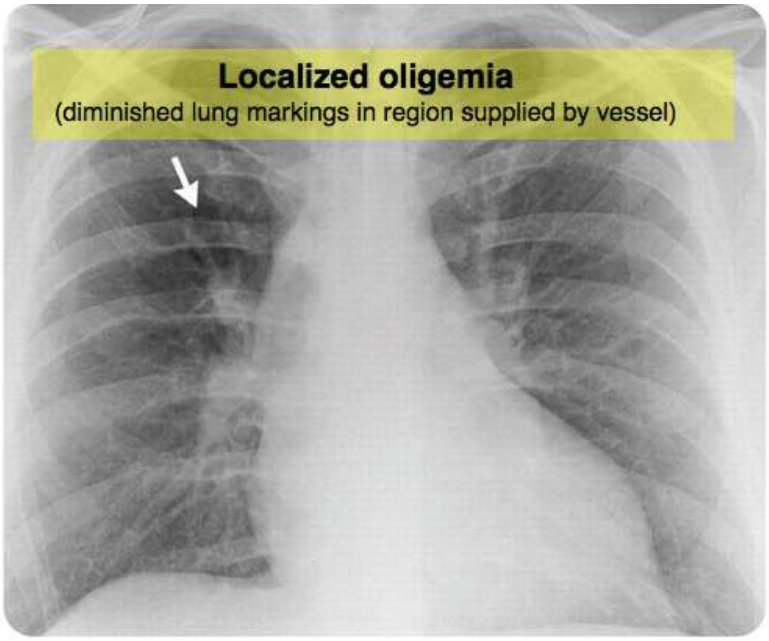



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**B. Westermark Sign**

**Associated with pulmonary embolism (rarely seen)**




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# XV. Respiratory Syncytial Virus

### A. Respiratory syncytial virus (RSV) infection

Pathophysiology
- Common cause of **bronchiolitis** in children

Management
- Supportive care
- Palivizumab vaccine for **high-risk** children

### B. Children at High Risk for RSV Lower Respiratory Tract Infection

| |
|---|
| Infants born before 29 weeks gestation |
| Infants < 32 weeks gestation with chronic lung disease of prematurity |
| Infants with hemodynamically significant congenital heart disease |
| Infants with pulmonary or neuromuscular disease that impairs cough |
| Children < 24 months old with chronic lung disease of prematurity who still require oxygen, corticosteroids, or diuretics |
| Children < 24 months old who are immunocompromised |

Notes


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# XVI. Shortness of Breath

### A. Dyspnea, or shortness of breath

| Physical Examination Signs of Dyspnea | | |
|---|---|---|
| **Sign** | **Clinical significance** | **Possible diagnosis** |
| **Absent or diminished breath sounds** | Decreased air movement | • COPD<br>• Severe asthma<br>• Pneumothorax<br>• Tension pneumothorax<br>• Hemothorax |
| **Accessory muscle use** | Muscle weakness, fatigue | • Respiratory failure<br>• Severe COPD<br>• Severe asthma |
| **Expiratory or mixed stridor** | Air flow obstruction below vocal cords | • Croup<br>• Foreign body<br>• Bacterial tracheitis |
| **Inspiratory stridor** | Air flow obstruction above vocal cords | • Foreign body<br>• Epiglottitis<br>• Angioedema |
| **Hyperventilation** | - | • Acidosis<br>• Sepsis<br>• Salicylate poisoning<br>• Anxiety |
| **JVD with clear lungs** | Right heart failure | • Cardiac tamponade<br>• Pulmonary embolism |
| **JVD with crackles** | Right and left heart failure | • Acute decompensated heart failure<br>• ARDS |
| **Heart murmur** | Valvular disease | • Valvular dysfunction |
| **Hepatojugular reflex** | Right heart failure | • Acute decompensated heart failure |
| **Pulsus paradoxus** | Poor right heart filling | • Right heart failure<br>• Pulmonary embolism<br>• Cardiogenic shock<br>• Pericardial tamponade<br>• Asthma exacerbation |
| **Crackles (rales)** | Interalveolar fluid | • Acute decompensated heart failure<br>• ARDS<br>• Pneumonia |
| **Wheezes** | Obstruction below trachea | • Asthma exacerbation<br>• Foreign body<br>• ADHF<br>• COPD |

Notes


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# XVII. Tuberculosis

## A. Pulmonary Tuberculosis (TB)

Patient
• History of **immunodeficiency**, immigrant, living in close quarters

Presentation
• Primary or latent TB: may be asymptomatic, otherwise fever is **most common** symptom
• Reactivation TB: fever, night sweats, weight loss, productive cough, hemoptysis

Physical Exam
• Fever, chest pain, fatigue cough, **erythema nodosum**

Diagnostic Studies
• Chest X-ray
  • Primary or latent: **Ghon focus**
  • Reactivation: **cavitary** lesions in **upper lobes**
• Establishing a diagnosis
  • Primary or latent: **PPD** (**gold standard**)
  • Reactivation: sputum smear for **acid-fast bacilli** (AFB), sputum or tissue culture for **AFB** (**gold standard**)

Management
• Primary or latent: nine months of **isoniazid**
• Reactivation: **r**ifampin, **i**soniazid, **p**yrazinamide, **e**thambutol (**RIPE**)

Comments
• Positive PPD criteria:
  • 15 mm: those without increased risk
  • 10 mm: high-risk, homeless, health care workers, IVDU, foreign-born
  • 5 mm: immunosuppressed, recent TB co tact, abnormal chest X-ray
• Supplement **pyridoxine** (vitamin B6) with isoniazid administration

Notes


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## B. Tuberculosis

**RIPE**: First line agents

**R**ifampin
**I**soniazid
**P**yrazinamide
**E**thambutol

### Multidrug Resistance (MDR-TB)

↳ Resistant to both **isoniazid** and **rifampin** (and possibly other agents)

**1 Initial phase**
5+ effective drugs including injectable for ≥ 6 months

- Pyrazinamide
- Ethambutol
- Fluoroquinolone (levofloxacin, moxifloxacin, gatifloxacin)
- Injectable agent (amikacin, capreomycin, kanamycin, streptomycin)
- Additional agent (ethionamide, cycloserine, linezolid, paraaminosalicylic acid)

**2 Continuation phase**
4+ effective drugs (in absence of injectable) for additional 12 months

- Pyrazinamide
- Ethambutol
- Fluoroquinolone (levofloxacin, moxifloxacin, gatifloxacin)
- Additional agent (ethionamide, cycloserine, linezolid, paraaminosalicylic acid)
- WHO recommends high-dose isoniazid

### Treatment Regimen for Latent TB Infection (LTBI)

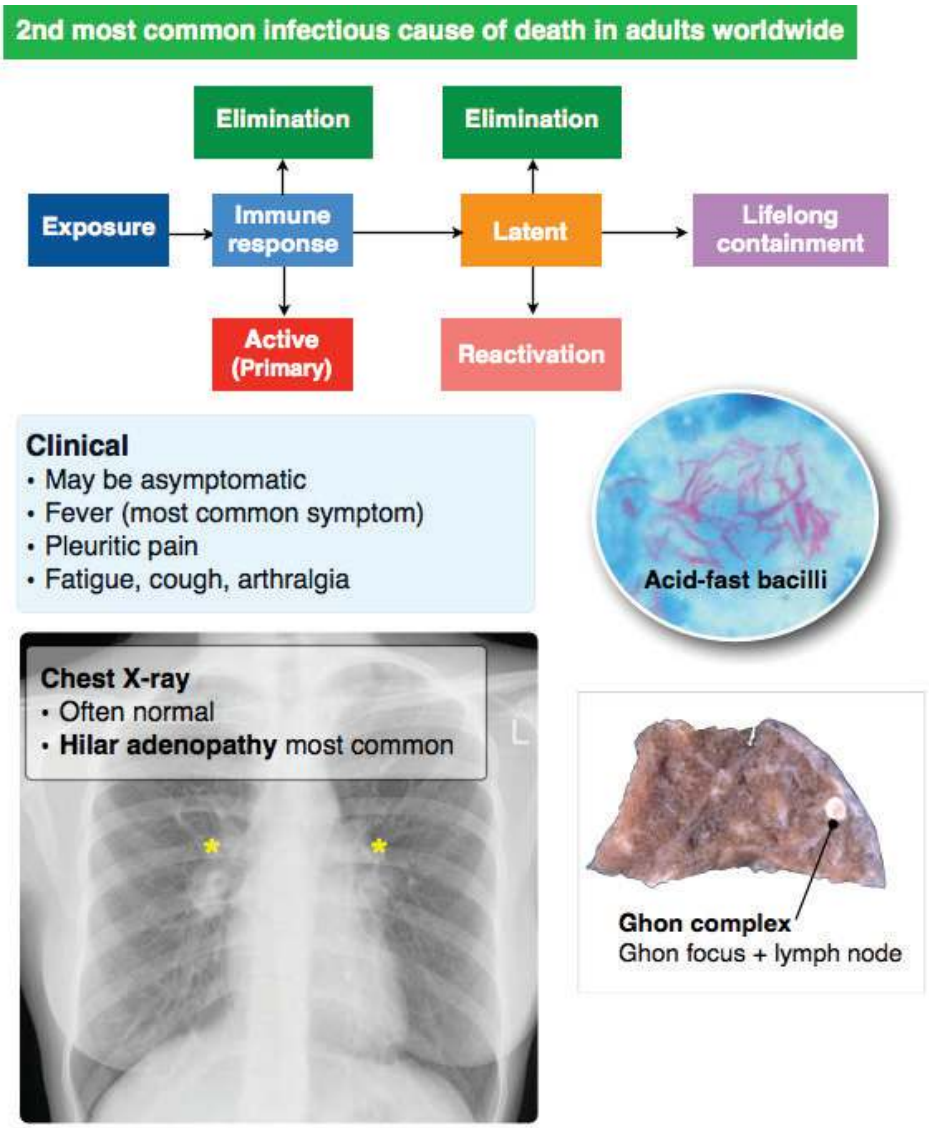
**Isoniazid (INH)** x 9 months (6 months may have same efficacy)
*Pyridoxine (Vit B6) supplementation recommended with INH regimen*

**Rifampin** x 4 months (alternative)

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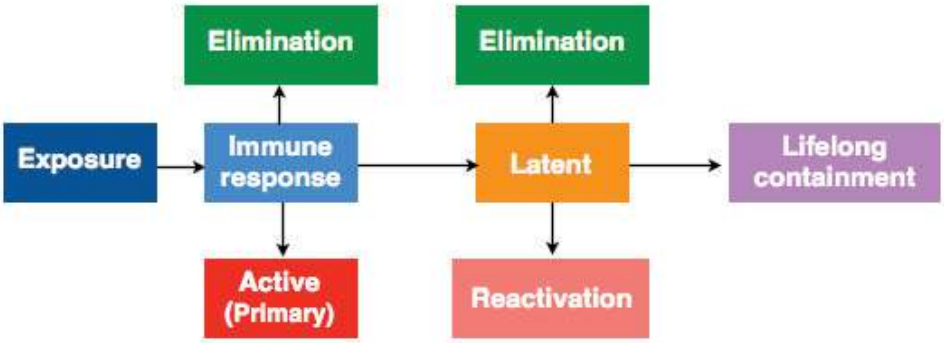
### C. Primary Tuberculosis




Notes

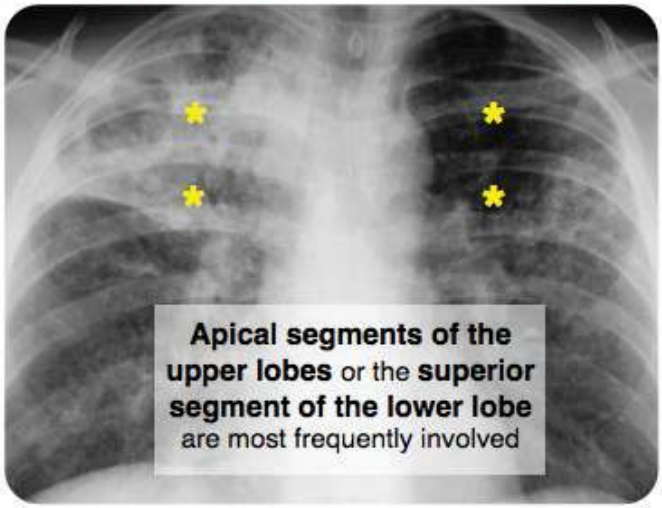

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### D. Reactivation Tuberculosis










**Clinical**
- Cough, weight loss, and fatigue
- Dyspnea
- Anorexia, wasting [consumption]

**Diagnosis**
- Isolation of *M. tuberculosis*
- Sputum acid-fast bacilli smear
- Nucleic acid amplification testing

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### E. Extrapulmonary Manifestations of Tuberculosis

| Location | Findings |
|---|---|
| Tuberculosis lymphadenitis | • Most commonly occurring form of extrapulmonary TB<br>• Cervical is most common |
| Pleural tuberculosis | • Pleural effusions<br>• Acute illness with cough, pleuritic chest pain, fever, or dyspnea |
| Skeletal tuberculosis | • Most often involves the spine (Pott's disease) |
| CNS tuberculosis | • Meningitis is most common presentation |
| GI tuberculosis | • Ulcerative (most common)<br>• Abdominal pain, diarrhea, weight loss, fever<br>• Ileocecal area and jejunoileum are most common sites involved |
| Tuberculosis peritonitis | • More common in HIV, cirrhosis, peritoneal dialysis<br>• Reactivation of latent TB |
| GU tuberculosis | • Dysuria, hematuria, flank pain |
| Miliary | • Any progressive, disseminated form of TB<br>• Can occur during primary dissemination or after years of untreated TB |
| Tuberculosis pericarditis | • Develops secondary to contiguous spread from mediastinal nodes, spine, or sternum or during miliary dissemination |
| TNF-A Inhibitor-Associated Tuberculosis | • Rare<br>• Occurs after treatment with TNF-alfa inhibitors (infliximab and etanercept) |

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CHAPTER 5

# Neurology

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CHAPTER 5
# Neurology


227 **ALTERED LEVEL OF CONSCIOUSNESS/ COMA**

228 **BELL PALSY**

230 **ENCEPHALITIS**

237 **HEMATOMAS**

240 **GUILLAIN-BARRÉ SYNDROME**

241 **HEAD TRAUMA/CONCUSSION/ CONTUSION**

244 **HEADACHE**
Migraine
Cluster
Tension

248 **INTRACEREBRAL HEMORRHAGE**

249 **LOSS OF CONSCIOUSNESS/CHANGE IN MENTAL STATUS**

251 **LOSS OF COORDINATION/ATAXIA**

254 **LOSS OF MEMORY**

257 **MENINGITIS**

263 **NUMBNESS/PARESTHESIA**

268 **SEIZURE**

273 **SPINAL CORD INJURY**

279 **STROKE**

283 **SUBARACHNOID HEMORRHAGE/ CEREBRAL ANEURYSM**

285 **SYNCOPE**

287 **TRANSIENT ISCHEMIC ATTACK**

288 **VERTIGO**

292 **WEAKNESS/PARALYSIS**



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# I. Altered Level of Consciousness/Coma




### A. Glasgow Coma Scale (GCS)

- All patients have ≥ 3
- Scored on:
  - Eye response
  - Verbal response
  - Motor response

| *Glasgow Coma Scale* | |
|---|---|
| **Best Eye Response (E)** | |
| 4 | Spontaneous - open with blinking at baseline |
| 3 | Opens to verbal command, speech, or shout |
| 2 | Open to pain, not applied to face |
| 1 | None |
| **Best Verbal Response (V)** | |
| 5 | Oriented |
| 4 | Confused conversation, but able to answer questions |
| 3 | Inappropriate responses, words discernible |
| 2 | Incomprehensible speech |
| 1 | None |
| **Best Motor Response (M)** | |
| 6 | Obeys commands for movement |
| 5 | Purposeful movement to painful stimulus (crosses midline) |
| 4 | Withdraws from pain |
| 3 | Abnormal (spastic) flexion, decorticate posture |
| 2 | Extensor (rigid) response, decerebrate posture |
| 1 | None |

Notes


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# II. Bell Palsy

**A. Bell Palsy**

Pathophysiology
• **Most commonly** caused by **HSV**

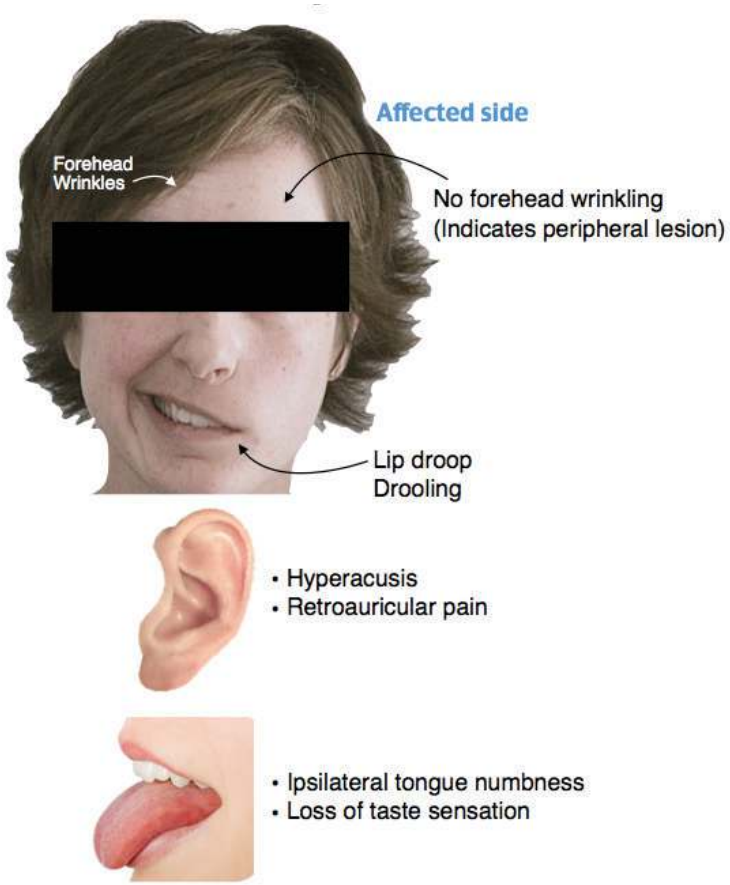
Patient
• History of a **viral prodrome**

Presentation
• **Unilateral facial nerve paralysis**, hyperacusis, and taste disturbance upon waking

Physical Exam
• **CN VII** nerve palsy that **involves the forehead** (as opposed to stroke that spares the forehead)

Management
• Prednisone, artificial tears, tape eyelid shut, acyclovir for severe presentations

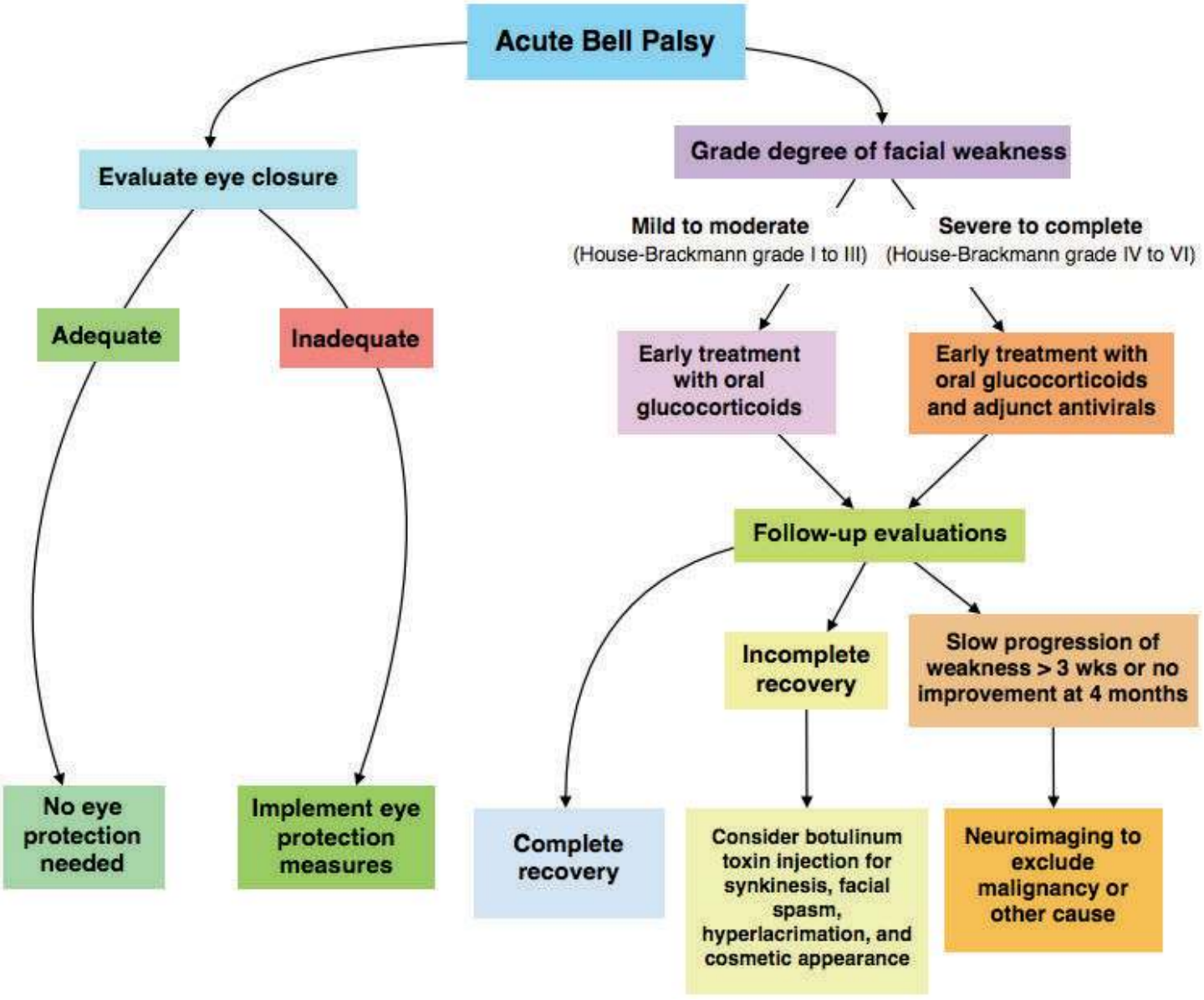
Comments
• **Bilateral palsy** linked to **Lyme disease**, **infectious mononucleosis**




Notes


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**B. Management of Acute Bell Palsy**




Notes


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# III. Encephalitis

### A. Hepatic Encephalopathy

Pathophysiology
• Caused by toxin accumulation due to liver dysfunction

Patient
• History of **liver failure** and recent infection, dehydration, medication change, GI bleed, hypoxia, or hypoglycemia

Presentation
• Complaining of **confusion**

Physical Exam
• **Scleral icterus** and **asterixis**

Diagnostic Studies
• **Elevated** serum ammonia

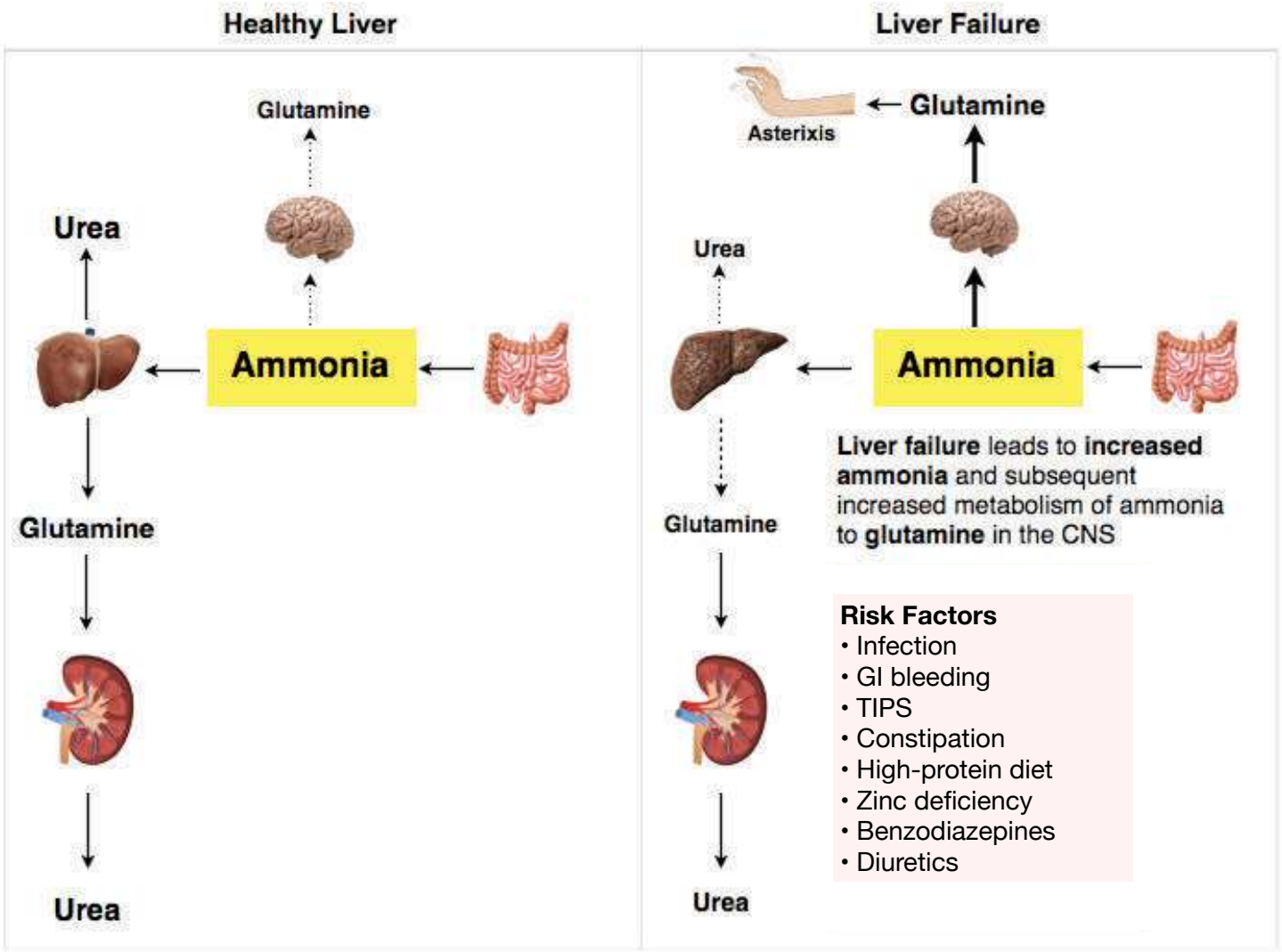
Management
• **Lactulose**, treatment of precipitating cause

Comments
• CSF glutamine correlates with severity

Notes


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**Hepatic Encephalopathy cont.**




Notes


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## B. Wernicke Encephalopathy

Pathophysiology
• Caused by **thiamine** (vitamin B1) **deficiency**

Patient
• **Chronic alcoholic**

Presentation
• **Ataxia** and **confusion**

Physical Exam
• **Nystagmus**, **gaze palsies**

Management
• Aggressive thiamine repletion

Comments
• Replace thiamine **before** glucose
• If untreated can lead to irreversible **Korsakoff syndrome**

### Wernicke Syndrome



1. **Encephalopathy**
   - Disorientation
   - Indifference
   - Inattentiveness
2. **Ocular motor dysfunction**
   - Nystagmus
   - Lateral rectus palsy
   - Conjugate gaze palsies
3. **Ataxia**
   - Gait
   - Stance

**Theoretical risk of worsening Wernicke syndrome**
Thiamine should be administered before glucose

**Management**
• Supportive
• Thiamine (IV)

Notes


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### C. Selected Pathogens Causing Encephalitis

| Virus | Transmission | Diagnosis | Treatment | Comments |
|---|---|---|---|---|
| **Arbovirus California encephalitis (LaCrosse)** | • Mosquito | • Serologic testing | • Supportive | • Mortality is low |
| **St. Louis encephalitis** | • Mosquito | • Serologic testing | • Supportive | • Mortality in up to 20% of patients |
| **West Nile** | • Mosquito | • PCR and IgM in CSF or serum<br>• Convalescent titers in serum | • Supportive | • Most infected patients present with febrile illness<br>• Advanced age is greatest risk factor for severe disease |
| **Enteroviruses** | • Fecal-oral | • CSF PCR | • Supportive | • Usually occurs in summer<br>• Rash, conjunctivitis, pleurodynia may occur |
| **Herpes simplex virus** | • Contact with mucosal surfaces | • CSF PCR | • Acyclovir | • HSV-1 usually causes encephalitis<br>• HSV-2 usually causes meningitis<br>• MRI may reveal abnormalities in temporal lobe with encephalitis |
| **Rabies virus** | • Bite from a rabid animal | • Viral antigen from a nape-of-neck biopsy | • Supportive<br>• Post-exposure prophylaxis with antiserum and active immunization | • Brainstem dysfunction causes diplopia, neuritis, excessive salivation, respiratory paralysis<br>• Consider diagnosis in spelunkers |

Notes

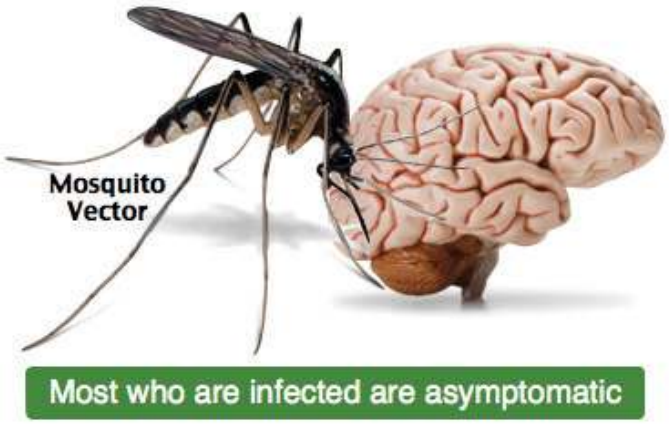

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**D. West Nile**

Pathophysiology
• Transmitted by **mosquitoes** in the summer and fall

Presentation
• **Flu-like** and URI symptoms, rash

Comments
• Complication: **meningoencephalitis**




Most who are infected are asymptomatic

**Clinical**
• Symptoms last 3-6 days
• Fever
• Flu-like illness
• Maculopapular rash

**Meningoencephalitis (<1%)**
• Asymmetric flaccid paralysis
• Bowel/bladder dysfunction
• No sensory abnormalities

**Diagnosis**
• CSF (elev lymps, protein, glucose)
• Serology
• Viral isolation

**Treatment**
• Supportive

Notes


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## E. Zika Virus



**Transmission**
- Bite of an infected mosquito
- Maternal-fetal transmission
- Sex (vaginal, anal, oral)
- Blood transfusion
- Organ transplantation
- Laboratory exposure



***Aedes* mosquitoes**

**Clinical**
- Low-grade fever
- Maculopapular pruritic rash
- Arthralgia (small joints of hands and feet)
- Conjunctivitis (nonpurulent)



***Microcephaly***

**Complications**
- Microcephaly
- Guillian-Barré syndrome

**Management**
- Supportive (no vaccine)
- Mosquito control

Notes


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## F. Herpes Encephalitis

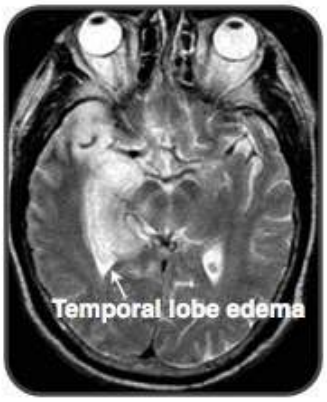
Presentation
• Headache, fever, **behavioral changes**

Diagnostic Studies
• CSF: **elevated** RBCs
• MRI will show **temporal lobe edema**

Management
• Acyclovir

Comments
• **Most common** cause of nonepidemic encephalitis in the US




**Lumbar puncture**
• Lymphocytic pleocytosis
• Increased red blood cells
• Elevated protein
• Normal glucose

**Clinical**
• Rapid onset of fever
• Headache
• Seizure
• Focal neurologic signs
• Impaired consciousness
• Personality changes

**Management**
• Intravenous acyclovir
• Empirically administered

Notes


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# IV. Hematomas

### A. Epidural Hematoma

Pathophysiology
• **Most common** artery ruptured is the **middle meningeal artery**

Patient
• History of a head injury with a loss of consciousness followed by a **lucid interval**

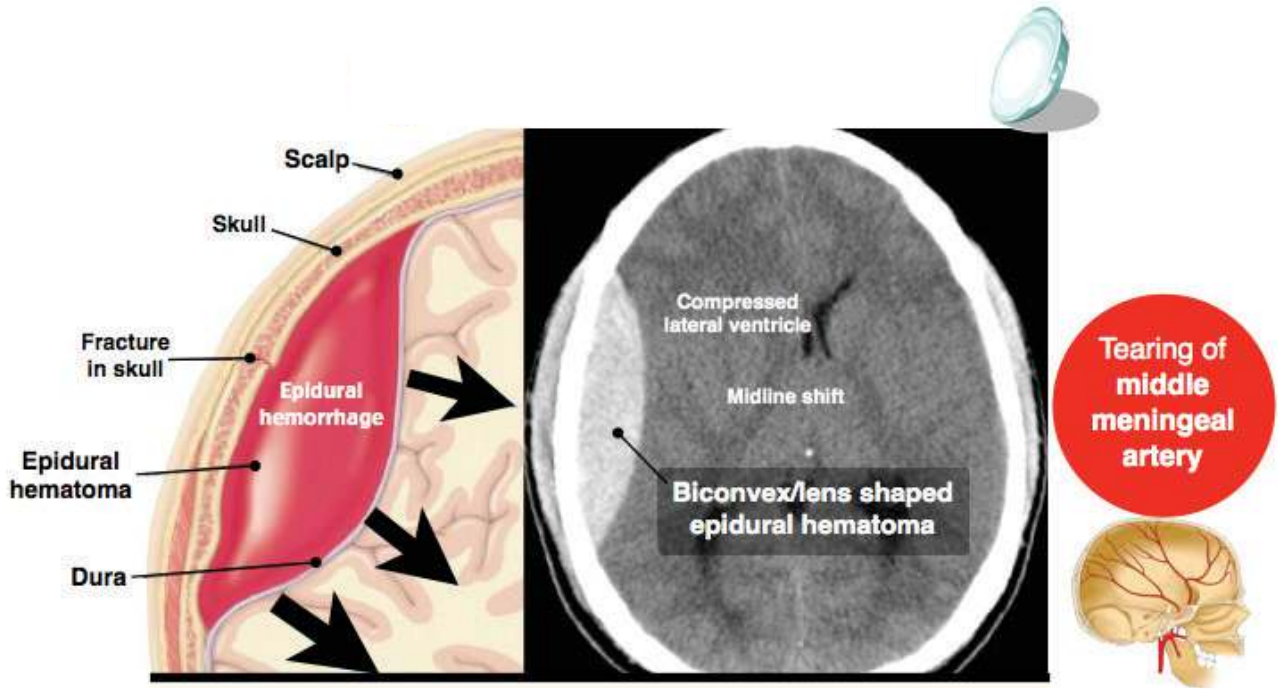
Diagnostic Studies
• CT will show a **biconvex opacity**

Management
• Emergent evacuation

**Etiology**
• Most common in adolescents and young adults
• Traffic accidents, falls, assaults

**Clinical**
• Blood between dura and skull
• Loss of consciousness followed by lucid interval then deterioration

**Management**
• Hematoma evacuation




Notes


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**B. Subdural Hematoma**

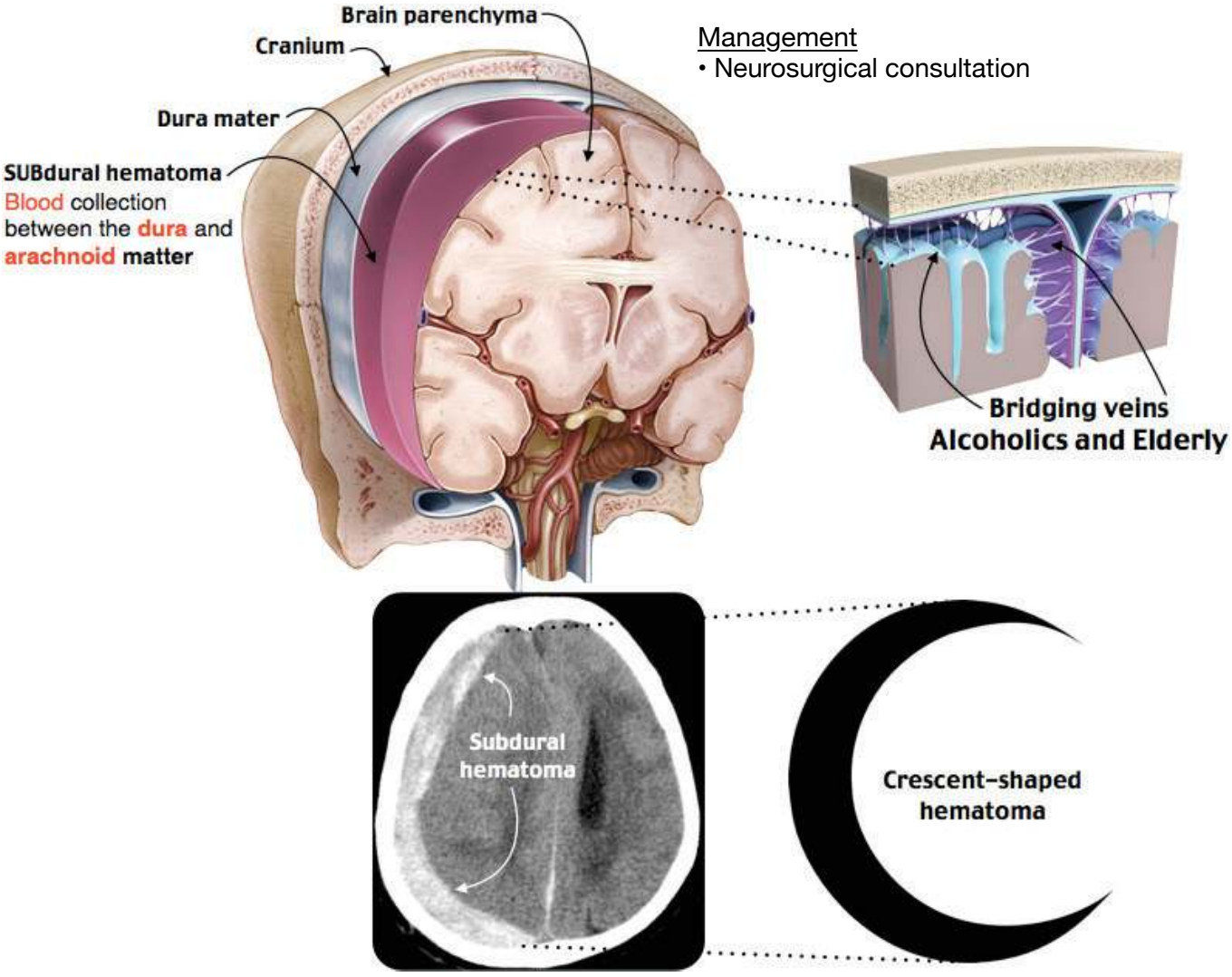
Pathophysiology
• Caused by a **rupture of the bridging veins**

Patient
• **Elderly** or **alcoholic** with a history of a **fall** or traumatic head injury

Presentation
• **Headache**, **mental status changes**, **seizures**, or **focal deficits**

Diagnostic Studies
• **Noncontrast CT** is diagnostic, revealing a **crescent-shaped hematoma**

Management
• Neurosurgical consultation




Notes

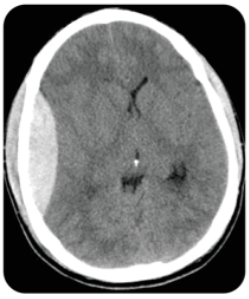

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## C. Cerebral Hemorrhages



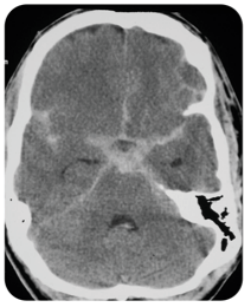


**Subdural hematoma**
- Crescent-shaped
- Blood collection between dura and arachnoid matter
- Tear in **bridging veins**
- Alcoholics and elderly are prone





**Epidural hematoma**
- Biconvex (lens) shaped
- Blood between dura and skull
- Tearing of **middle meningeal artery**
- Adolescents and young adults (trauma)





**Subarachnoid hemorrhage**
- Blood in circle of Willis, cisterns, and fissures
- Rupture of **berry aneurysm**
- Polycystic kidney disease (risk factor)





**Intracerebral hemorrhage**
- Blood in parenchyma and ventricles
- Hypertensive vasculopathy
- Territory of **penetrator arteries**

Notes


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# V. Guillain-Barré Syndrome

### A. Guillain-Barré Syndrome

Pathophysiology
• **Most commonly** caused by ***Campylobacter jejuni***

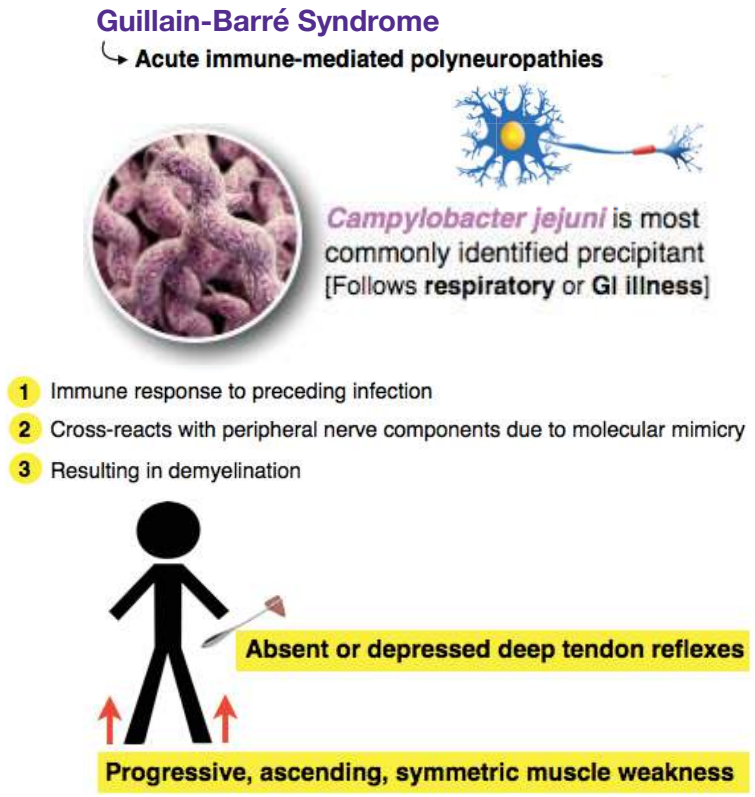
Patient
• History of recent minor respiratory or GI illness

Presentation
• **Symmetric**, progressive **ascending** muscle weakness

Physical Exam
• **Lack of deep tendon reflexes**

Diagnostic Studies
• LP will reveal **increased** **CSF protein** but a normal cell count

Management
• Supportive care, plasmapheresis, or **IVIG**




**Management**
• Supportive
• Measurement of vital capacity and negative inspiratory force (NIF)
• Plasmapheresis
• IVIG

**Diagnosis**
• Clinical
• Lumbar puncture: Albuminocytologic dissociation (Elevated protein with normal/mild pleocytosis)

Notes


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# VI. Head Trauma/Concussion/Contusion

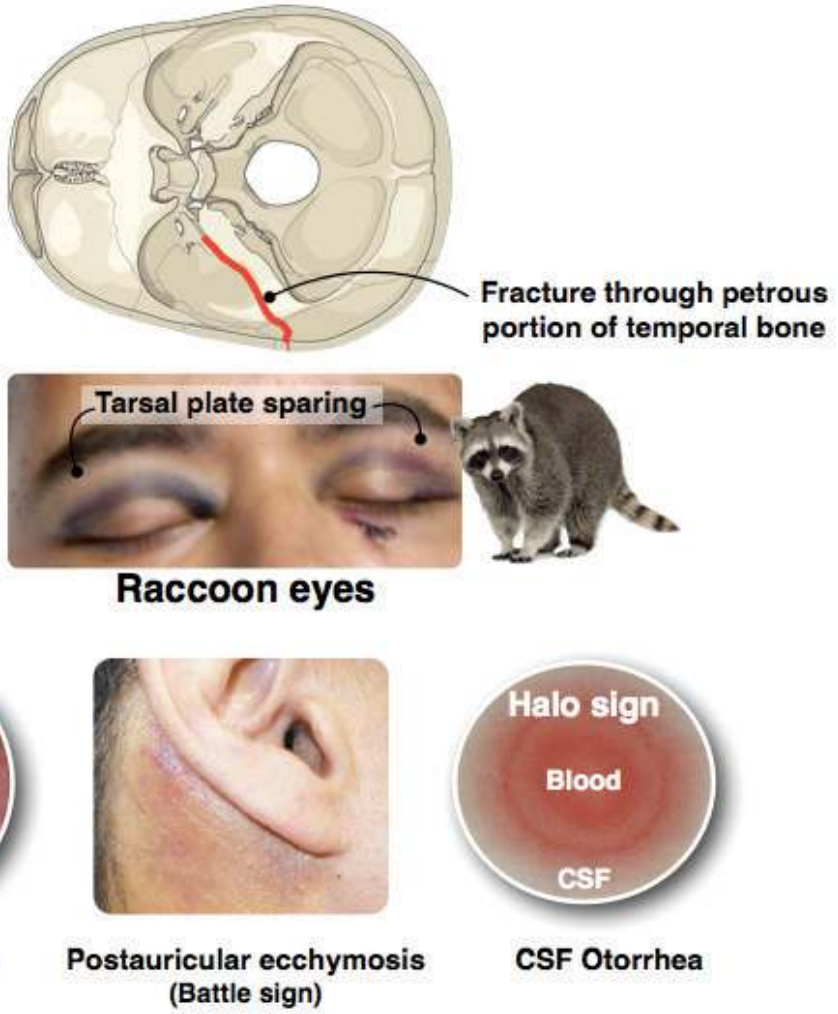
### A. Basilar Skull Fracture

Pathophysiology
• Petrous portion of **temporal bone** fracture **most common**

Presentation
• **Battle sign**, raccoon eyes, hemotympanum, CSF otorrhea/rhinorrhea

Diagnostic Studies
• CT

Comments
• Can result in CN VII, VIII entrapment




Notes


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**B. Concussion**

Presentation
• Brief loss of consciousness with amnesia

Physical Exam
• No focal neurologic deficits

Diagnostic Studies
• If indicated, CT scan will be negative for intracranial abnormality

**Graduated Return-to-Play Protocol**

| Stage | Aim | Activity | Goal of each step |
|---|---|---|---|
| 1 | Symptom-limited activity | Daily activities that do not provoke symptoms | Gradual reintroduction of work/school activities |
| 2 | Light aerobic exercise | Walking or stationary cycling at slow to medium pace. No resistance training | Increase heart rate |
| 3 | Sport-specific exercise | Running or skating drills. No head impact activities | Add movement |
| 4 | Noncontact training drills | Harder training drills, e.g., passing drills. May start progressive resistance training | Exercise, coordination, and increased thinking |
| 5 | Full contact practice | Following medical clearance, participate in normal training activities | Restore confidence and assess functional skills by coaching staff |
| 6 | Return to sport | Normal game play | |

Note: An initial period of 24 to 48 hours of both relative physical rest and cognitive rest is recommended before beginning the return to sport progression. There should be at least 24 hours (or longer) for each step of the progression. If any symptoms worsen during exercise, the athlete should go back to the previous step. Resistance training should be added only in the later stages (stage 3 or 4 at the earliest). If symptoms are persistent (e.g., more than 10 to 14 days in adults or more than 1 month in children), the athlete should be referred to a health care professional who is an expert in the management of concussion.

McCrory P, Meeuwisse W, Dvořák J, et al. Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016. Br J Sports Med 2017; 51:838.

Notes


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### C. Post-Concussive Syndrome



Pathophysiology
- Post TBI
- Multifactorial causes

Presentation
- **Nonspecific symptoms** (headache, difficulty concentrating, dizziness)

Diagnostic Studies
- Neuropsychological testing
- Imaging usually **not indicated** without red flag symptoms

Management
- Brain "rest"




- Difficulty thinking clearly
- Feeling slowed down
- Difficulty concentrating
- Difficulty remembering new information




- Headache
- Nausea/vomiting
- Dizziness
- Blurry vision
- Feeling tired
- Lack of energy
- Sensitivity to light or noise




- Irritability
- Depression
- Increased emotions
- Anxiety




- Increased or decreased
- Trouble falling asleep

Notes


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# VII. Headache

MIGRAINE • CLUSTER • TENSION

## A. Migraine Headache

Patient
• Woman

Presentation
• **Gradual onset** of a **unilateral**, throbbing, pulsating headache
  • **Without aura** (**most common**): nausea, vomiting, photophobia, phonophobia
  • **Aura**: scotoma, flashing lights, sounds

Management
• Abortive: triptans, dihydroergotamine, antiemetics, NSAIDs
• Prophylaxis: BBs, CCBs, TCAs

Comments
• Triptans, dihydroergotamine: **contraindicated** in HTN or cardiovascular disease
• Triggers include menstruation, pregnancy, certain foods, alcohol



**Triggers**
• Menstruation, pregnancy, contraceptives
• Food (chocolate, cheese, MSG, nitrites)
• Alcohol

**Clinical**
**[No Aura]**
• Slow onset
• Unilateral, pulsating
• Nausea, vomiting, photo/phono-phobia

**[With Aura]**
• Headache follows aura w/in 30 min
• Aura develops over minutes, reversible
• Visual - most common - scintillating scotomata
• Motor - hemiparesis, ophthalmoplegia, aphasia
• Sensory - hemiparesthesia, dysesthesia
• Brainstem - vertigo, ataxia

Notes


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### B. Migraine Headache Management

| Migraine Headache Management |
| --- |
| **Acute Exacerbation** |
| Triptans |
| Dihydroergotamine |
| NSAIDs |
| Acetaminophen |
| Antiemetics |
| **Prophylaxis** |
| Beta-blockers (propranolol) |
| Calcium channel blockers (verapamil) |
| Tricyclic antidepressants (amitriptyline) |
| Anticonvulsants (valproic acid, topiramate) |
| Botulinum toxin A |

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## C. Cluster Headache

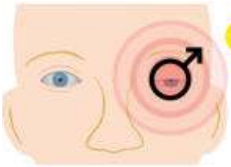
Patient
• Man

Presentation
• Sudden onset, brief, unilateral, and repetitive headaches localized behind one eye

Physical Exam
• Ipsilateral conjunctival injection, **lacrimation**, and **rhinorrhea**

Management
• **High-flow oxygen** in acute setting; **CCBs** as prophylaxis

**Always unilateral**
(But can change sides with new attack)



1 **Pain**
- Excruciating unilateral pain
- Periorbital and temporal

2 **Autonomic symptoms**
- Ptosis
- Miosis
- Lacrimation
- Conjunctival injection
- Rhinorrhea
- Nasal congestion

3 **Circadian periodicity**
- Short-lived (15 to 180 min) cluster attacks
- Attacks occur daily (in clusters) followed by remission

**Treatment**
- 100% oxygen
- Sumatriptan



Notes


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**D. Tension Headache**

Pathophysiology
• **Most commonly** caused by stress

Presentation
• **Bilateral**, nonpulsating, **bandlike pain** across forehead

Physical Exam
• Tenderness to neck musculature

Management
• **NSAIDs**

Comments
• **Most common** type of headache



• **Bilateral** headache
• Pain in **frontal** and **occipital** regions
• **Bandlike** distribution
• Exacerbated by **stress**, **fatigue**, **glare**, **noise**
• **Most common** type of headache

Notes


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# VIII. Intracerebral Hemorrhage

### A. Intracerebral Hemorrhage

Pathophysiology
• Caused by spontaneous bleeding from arterioles

Patient
• Older than 50 years of age with a history of **HTN** and **atherosclerotic risk factors**

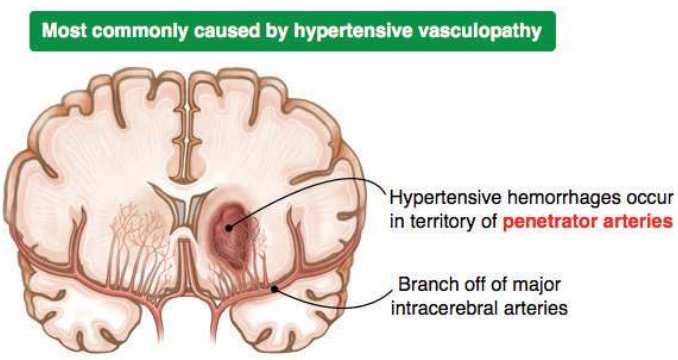
Presentation
• Neurological deficits (indistinguishable from ischemia)

Physical Exam
• Pontine lesion: pinpoint pupils, coma, **decerebrate posturing**
• Cerebellar lesion: vomiting, dizziness, **CN VI palsy**

Management
• Blood pressure control, ICP control, reverse all coagulopathies

**Spot Sign**
• Unifocal or multifocal contrast enhancement within an acute primary intracerebral hemorrhage visible on CTA source images and discontinuous from adjacent normal or abnormal blood vessels
• Should not be present on pre-contrast images
• Corresponds to a site of active, dynamic hemorrhage and is an independent predictor of ICH growth and poor outcome

Notes







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# IX. Loss of Consciousness/Change in Mental Status

### A. Loss of Consciousness

See above - Altered Level of Consciousness

### B. Change in Mental Status

**Altered Mental Status: AEIOU TIPS**

**A**: alcohol, acidosis
**E**: endocrine, epilepsy, electrolytes, encephalopathy
**I**: infection
**O**: opiates, overdose
**U**: uremia
**T**: trauma
**I**: insulin
**P**: poisoning, psychosis, pharmacology
**S**: stroke, seizure, syncope

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**C. Delirium**

Patient will present with an abrupt onset of a disturbance in baseline mental status that fluctuates

**D. Delirium VS. Dementia**

| Delirium vs. Dementia | | |
|---|---|---|
| **Characteristic** | **Delirium** | **Dementia** |
| **Onset** | Abrupt | Insidious |
| **Course** | Fluctuates | Slow decline |
| **Duration** | Hours to weeks | Months to years |
| **Attention** | Impaired | Intact early; impaired late |
| **Sleep-wake** | Disrupted | Usually normal |
| **Alertness** | Impaired | Normal |
| **Orientation** | Impaired | Intact early; impaired late |
| **Behavior** | Agitated, withdrawn or depressed | Intact early |
| **Speech** | Incoherent, rapid/slowed | Word-finding problems |
| **Thoughts** | Disorganized, delusions | Impoverished |
| **Perceptions** | Hallucinations/illusions | Intact early |

Notes


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# X. Loss of Coordination/Ataxia

## ATAXIA

### A. Motor Ataxia

• **Most common** cause: cerebellar lesion
• **Cerebellar gait**: wide-based, unsteady, irregular steps

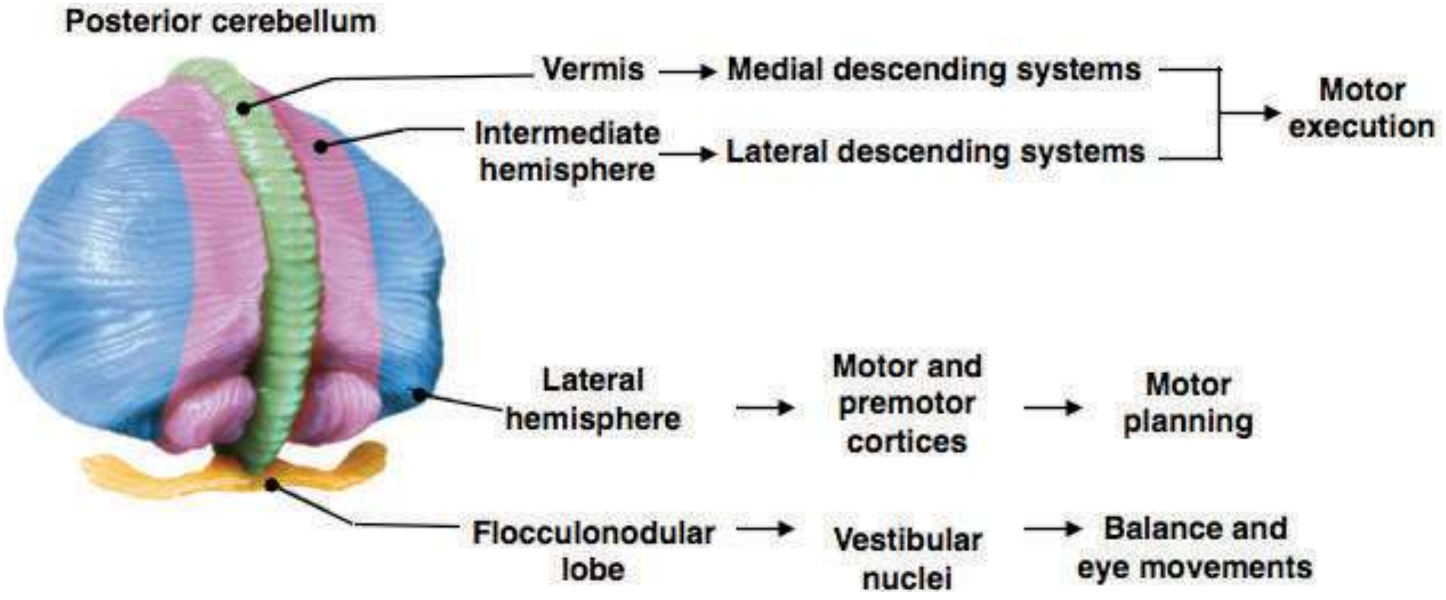
### B. Sensory Ataxia

• Decrease in visual input
• **Sensory ataxic gait**: abrupt leg movement, foot slapping impact
• **Romberg sign**

Notes


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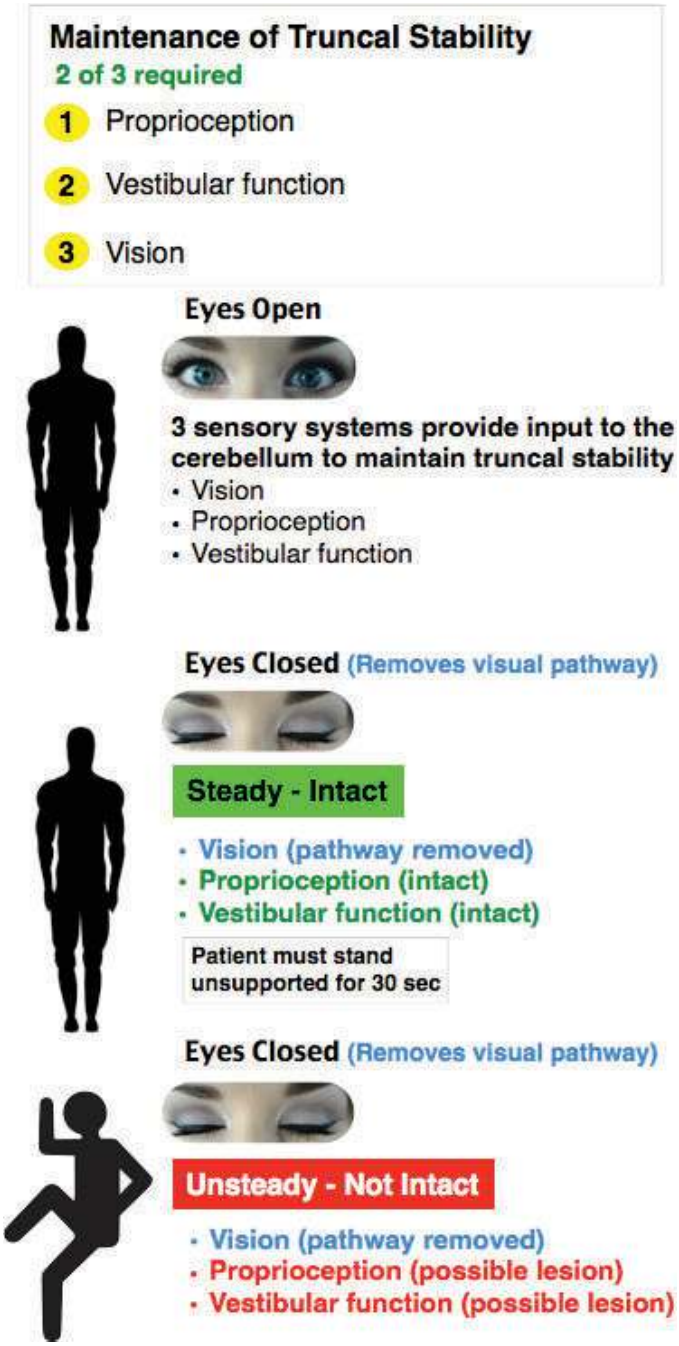
### C. Cerebellum




Notes


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## D. Romberg Test




**Positive Romberg Test (Sensory Ataxia)**
- Vitamin B12 deficiency
- Tabes dorsalis (dorsal column)
- Chronic inflammatory demyelinating polyradiculopathy
- Friedreich ataxia
- Ménière disease (vestibular)

Notes


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# XI. Loss of Memory

### A. Types of Dementia



**Alzheimer Disease**
• 2/3 of dementia cases
• Risk factors: advanced age, family history

**Vascular Dementia**
• 1/4 of cases
• Risk factors: HTN, dyslipidemia, DM, smoking, advanced age

**Lewy Body Dementia**
• Cognitive fluctuations, visual hallucinations, Parkinsonism

**Frontotemporal Dementia**
• Personality and social behavior changes, nonfluent speech

**Neurodegenerative Conditions**
• Huntington disease, metabolic abnormalities

Notes

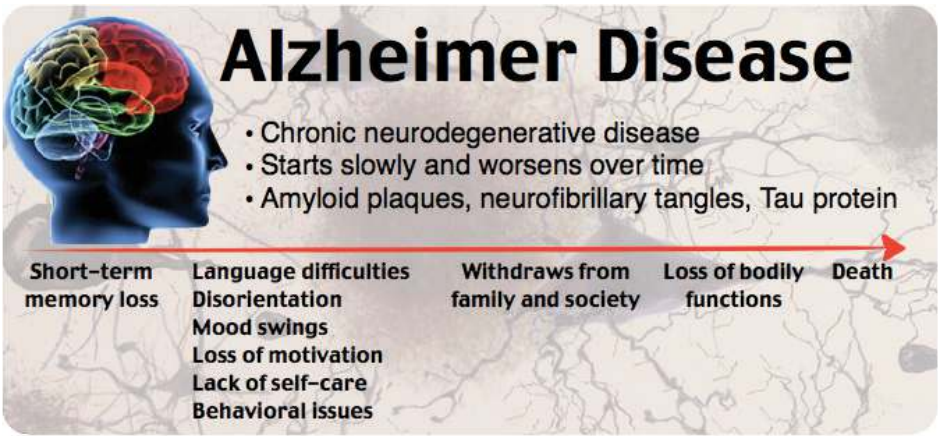

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**B. Alzheimer Disease**

Presentation
• **Short-term memory loss**, confusion about the location of familiar places, difficulty completing daily tasks that would normally take less time to do, poor judgment which can lead to bad decision making, changes in mood or personality

Diagnostic Studies
• Definitive diagnosis is confirmed by histopathologic examination of the brain showing **amyloid plaques** and **neurofibrillary tangles**
• In practice, diagnosis is made with clinical criteria
• Mini-mental state exam monitors progression

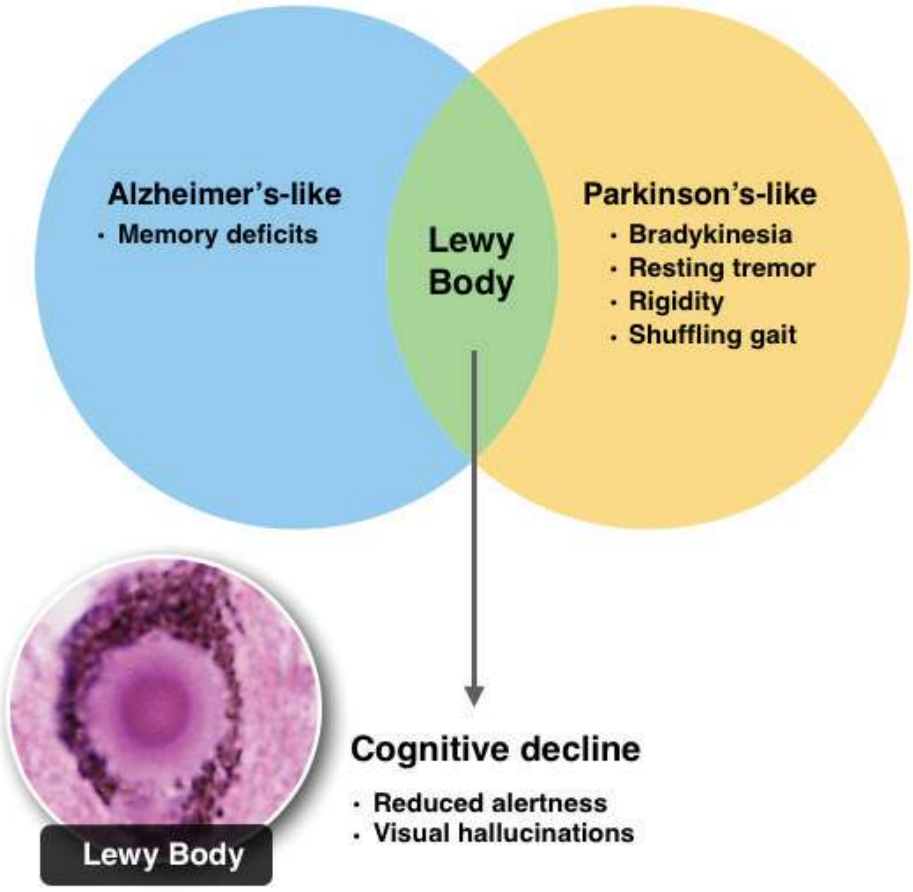
Management
• Cholinesterase inhibitors (donepezil), NMDA antagonists (memantine)
• No known cure; medications aim to slow progression




Notes


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**C. Lewy Body Dementia**




Notes


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# XII. Meningitis

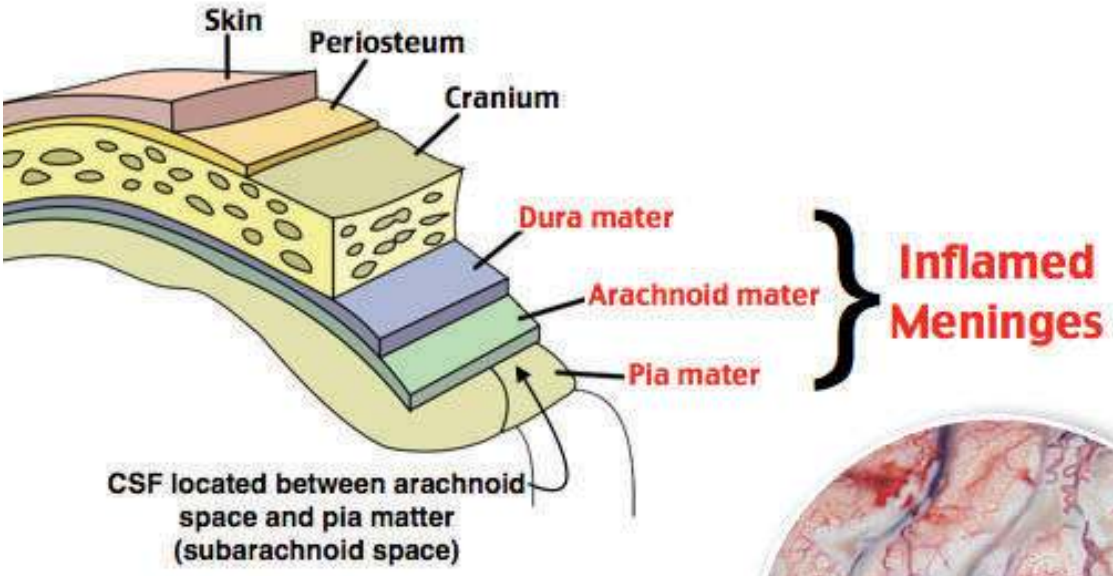
### A. Meningitis CSF Findings

| | Normal | Bacterial | Viral | Fungal | Tuberculosis |
|---|---|---|---|---|---|
| Opening pressure | 5 - 20 | Elevated | Normal or slightly elevated | Elevated | Variable |
| Leukocytes | < 5 | > 1,000 | 25 - 2,000 | 10 - 1,000 | 10 - 1,000 |
| Cell differential | 0 | Neutrophil predominance | Early: Neutrophils Late: Lymphocytes | Lymphocyte predominance | Lymphocyte predominance |
| Protein | 15 - 45 | High | High | High | Very high |
| Glucose | 45 - 85 | Low | Low/Normal | Low | Low |
| Cultures | --- | Positive | Negative | Positive (fungal) | Positive (AFB) |

Notes


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## B. Meningitis






**Etiology**
- Bacteria
- Viruses
- Fungus
- Drugs
- Autoimmune

**Clinical**
- Headache
- Neck stiffness
- Fever
- Confusion/AMS
- Photophobia
- Phonophobia

**Dx**
- Lumbar puncture

**Rx**
- Corticosteroids
- Antibiotics, antivirals, antifungals

Notes


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### C. Viral Meningitis

Pathophysiology
• **Most commonly** caused by **enteroviruses**

Diagnostic Studies
• CSF: normal glucose, mildly **elevated** protein (< 200 mg/dL), lymphocytic pleocytosis

Management
• Supportive care

Notes


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**D. Bacterial Meningitis (Adult)**

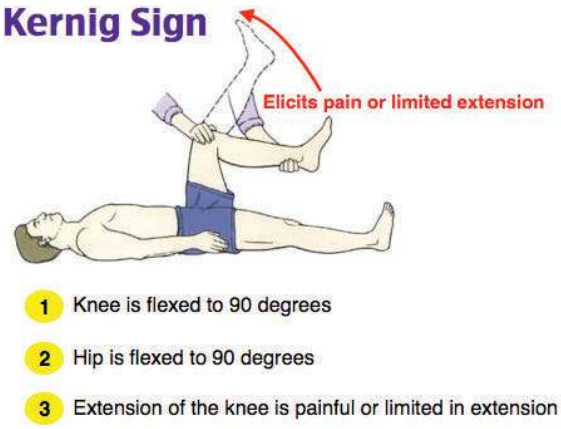
Pathophysiology
• **Most commonly** caused by ***Streptococcus pneumoniae*** (*Cryptococcus* in HIV patients)

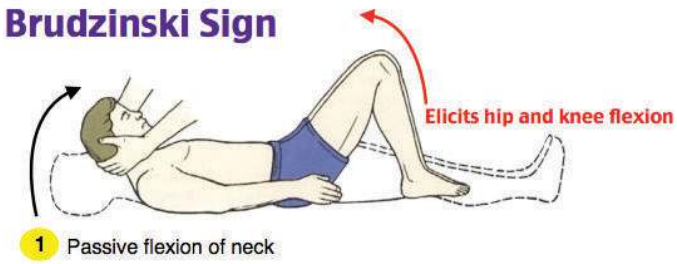
Presentation
• Headache, neck stiffness, photophobia, phonophobia, fever

Physical Exam
• Meningismus, **Brudzinski sign** (knees and hips flex when neck is flexed), **Kernig sign** (pain limits knee extension)

Management
• Initiate empiric antibiotics if LP or CT are delayed
  • 18–50 years of age: ceftriaxone + vancomycin
  • > 50 years of age: ceftriaxone + vancomycin + **ampicillin** (to cover *Listeria*)

Comments
• Mnemonic: **B**rudzinski - **B**end the **b**rain, **K**ernig - extend **k**nees




Notes


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### E. Bacterial Meningitis

| Bacterial Meningitis | | |
|---|---|---|
| **Age** | **Common organisms** | **Antibiotic therapy** |
| **Neonates (≤ 1 month)** | • *E. coli*<br>• Group B strep<br>• *Listeria monocytogenes*<br>• Gram-neg bacilli | Cefotaxime + Ampicillin OR<br>Gentamicin + Ampicillin |
| **Children (> 1 month–18 years)** | • *S. pneumoniae*<br>• *N. meningitidis*<br>• *H. influenzae* | Ceftriaxone + Vancomycin OR<br>Cefotaxime + Vancomycin |
| **Adults (18–50 years)** | • *S. pneumoniae*<br>• *N. meningitidis* | Ceftriaxone + Vancomycin |
| **Adults > 50 years and alcoholics** | • *S. pneumoniae*<br>• *N. meningitidis*<br>• *Listeria monocytogenes*<br>• Gram-neg bacilli | Ceftriaxone + Vancomycin + Ampicillin |
| **Adults with impaired cellular immunity** | • *S. pneumoniae*<br>• *N. meningitidis*<br>• *Listeria monocytogenes*<br>• Gram-neg bacilli<br>• *P. aeruginosa* | Vancomycin + Ampicillin + Cefepime OR Meropenem |

**General Principles of Antibiotic Therapy for Bacterial Meningitis**

1. The agent must be bactericidal against infecting organism
2. The agent must be able to penetrate blood-brain barrier

Notes


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## F. Meningococcemia

Pathophysiology
• **Most commonly** caused by ***Neisseria meningitidis***, an **aerobic**, **gram-negative diplococcus**

Patient
• Military recruit or student

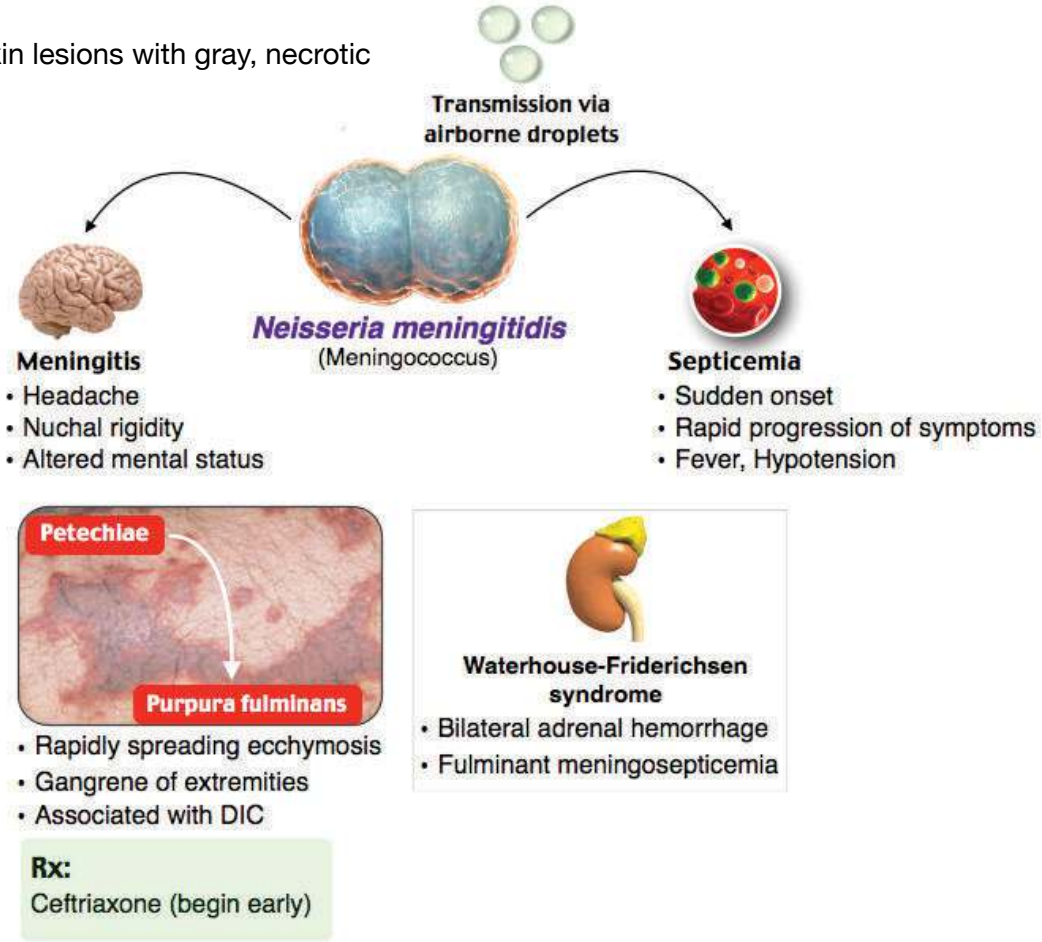
Presentation
• Fever, headache, arthralgias, rash

Physical Exam
• **Petechiae**, skin lesions with gray, necrotic centers

Diagnostic Studies
• Diagnosis is made clinically; can be confirmed by blood cultures and Gram stain or LP

Management
• **Ceftriaxone and vancomycin**

Comments
• **Waterhouse-Friderichsen syndrome**: bilateral adrenal hemorrhage with meningococcus




Notes


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# XIII. Numbness/Paresthesia

### A. Carpal Tunnel Syndrome

Pathophysiology
• Caused by **median nerve** compression

Patient
• History of **extensive** and **repetitive** wrist usage such as typing or assembly line work

Presentation
• Pain and numbness in the **first**, **second**, and **third digits especially at night**

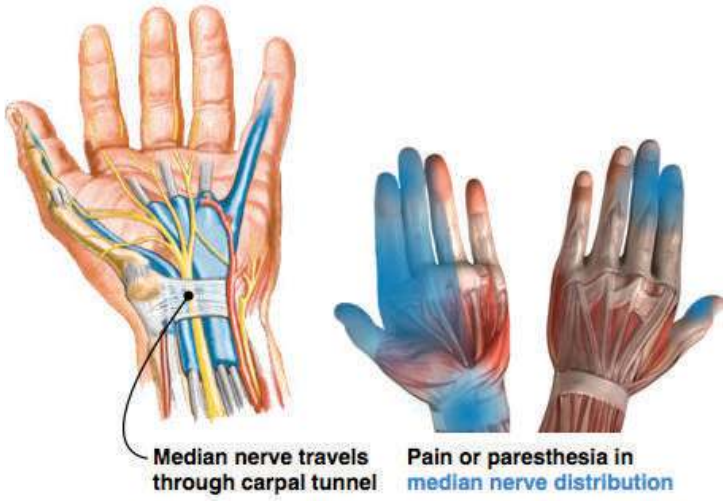
Physical Exam
• **Phalen sign**: reproduction of symptoms with wrist hyperflexion
• **Tinel sign**: reproduction of symptoms with percussion over the carpal tunnel

Management
• NSAIDs, **volar splint** in neutral position, glucocorticoid injections
• Surgical decompression if refractory to conservative management

**Treatment**
• Glucocorticoid injections
• Splinting
• Surgical decompression

**Carpal Tunnel Syndrome**

**Compression of median nerve**




| Risk Factors |
|---|
| Obesity |
| Female sex |
| Coexisting conditions (diabetes, pregnancy, rheumatoid arthritis, hypothyroidism, connective tissue diseases) |
| Genetic predisposition |
| Aromatase inhibitor |

Notes


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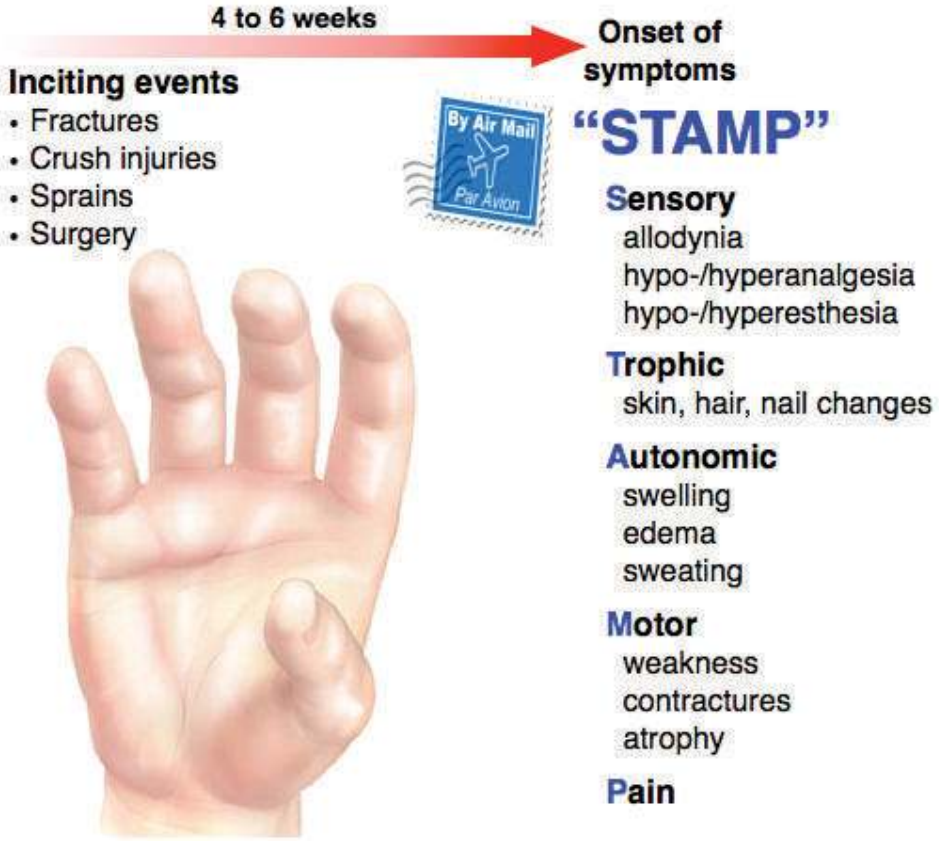
**B. Complex Regional Pain Syndrome**

Patient
• History of **previous extremity injury** or **fracture**

Presentation
• **Light touch causes extreme pain** and **allodynia** (pain felt from a nonpainful stimulus, such as clothes or bed sheets on the skin)

Management
• NSAIDs, gabapentin, sympathectomy

**Management**
• NSAIDs
• Anticonvulsant (gabapentin, pregabalin)
• TCA (amitriptyline)
• Bisphosphonate (clodronate)
• Topical lidocaine
• Pain management specialist




Notes

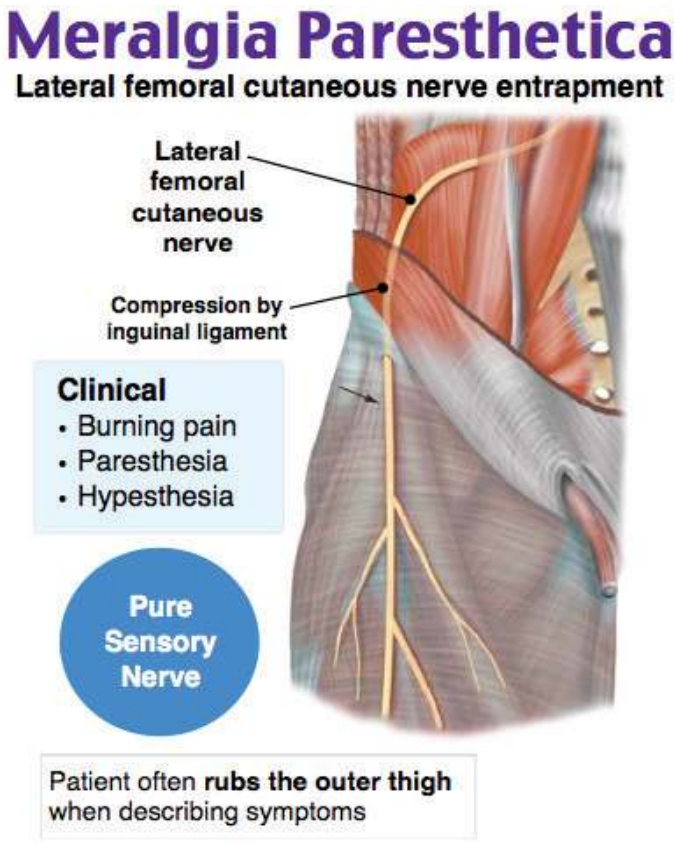

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**C. Meralgia Paresthetica**

Pathophysiology
• Entrapment of the **lateral femoral cutaneous** nerve
• Can be a result of an **inguinal ligament** injury

Presentation
• Dysesthesia and numbness of proximal anterolateral thigh

Management
• Loose clothing, weight loss




Notes


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### D. Cervical Radiculopathy




| Disk level | Root | Pain distribution | Weakness | Sensory loss | Reflex loss |
|---|---|---|---|---|---|
| C4–C5 | C5 | • Medial scapular border<br>• Lateral upper arm to elbow | • Deltoid<br>• Supraspinatus<br>• Infraspinatus | • Lateral upper arm | Supinator reflex |
| C5–C6 | C6 | • Lateral forearm, thumb, index finger | • Biceps<br>• Brachioradialis<br>• Wrist extensors | • Thumb and index finger | Biceps reflex |
| C6–C7 | C7 | • Medial scapula<br>• Posterior arm<br>• Dorsum of forearm, third finger | • Triceps<br>• Wrist flexors<br>• Finger extensors | • Posterior forearm<br>• Middle finger | Triceps reflex |
| C7–T1 | C8 | • Shoulder<br>• Ulnar side of forearm, fifth finger | • Thumb flexors<br>• Abductors<br>• Intrinsic hand muscles | • Fifth finger | -- |

Notes


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### E. Multiple Sclerosis

Pathophysiology
• Caused by a **demyelinating disorder**

Patient
• **Caucasian female**

Presentation
• Pain with eye movement, **monocular vision loss**, **sensory abnormalities**

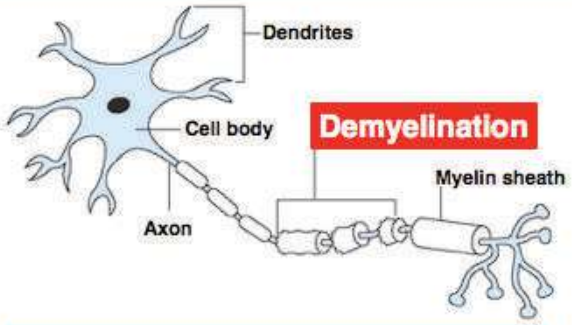
Physical Exam
• Spinal electric shock sensation with neck flexion (**Lhermitte phenomenon**)
• Bilateral internuclear ophthalmoplegia (eyes can't look at nose) is pathognomonic

Diagnostic Studies
• CSF will reveal **elevated IgG protein**, WBC pleocytosis
• **T2-weighted MRI** is diagnostic

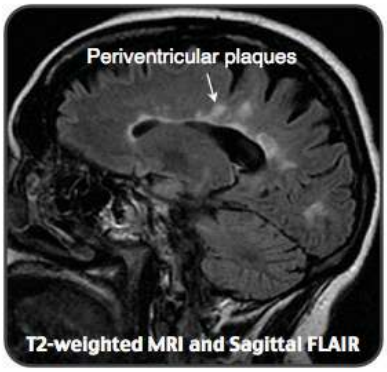
Management
• Symptomatic treatment, methylprednisolone

**Treatment**
• Glucocorticoids (acute attacks)
• Plasma exchange
• Disease-modifying therapy (DMT) (relapsing-remitting)
• Hematopoietic stem cell transplation (suggestive in small studies)

**Most common immune-mediated inflammatory demyelinating disease of the CNS**




| Features Suggestive of Multiple Sclerosis |
|---|
| Relapses and remissions |
| Onset between ages 15 and 50 years |
| Optic neuritis |
| Lhermitte sign |
| Internuclear ophthalmoplegia |
| Fatigue |
| Heat sensitivity (Uhthoff phenomenon) |




Notes


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# XIV. Seizure

### A. Common Features to Help Distinguish Epileptic and Psychogenic Nonepileptic Seizures

| Common Features to Help Distinguish Epileptic and Psychogenic Nonepileptic Seizures | | |
|---|---|---|
| **Sign** | **Epileptic** | **Psychogenic Nonepileptic Seizures** |
| **Duration** | • Usually brief, less than 1 to 2 minutes | • Usually longer than 2 minutes |
| **Eyes** | • Eyes usually open during event | • Eyes often closed<br>• Forced eye closure suggests PNES |
| **Motor activity** | • Stereotyped<br>• Synchronized<br>• Build, progress | • Variable<br>• Forward pelvic thrusting, rolling side to side, opisthotonus<br>• Wax and wane |
| **Vocalization** | • Uncommon, especially during convulsion | • May occur |
| **Prolonged ictal atonia** | • Very rare | • May occur |
| **Incontinence** | • Common in convulsive seizures | • Less common |
| **Autonomic signs** | • Cyanosis, tachycardia common with major convulsion | • Uncommon |
| **Postictal symptoms** | • Usually confused, drowsy<br>• Headache common | • May rapidly awaken and reorient<br>• Headache rare |

Notes


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### B. Absence Seizures (petit mal)



Patient
• **Child**, 5–10 years old

Presentation
• **Sudden** mental status alteration **without motor activity**, **blank stare**
• No aura or postictal state

Diagnostic Studies
• EEG would show **symmetric 3-Hz spike and wave activity**

Management
• **Ethosuximide**

**Clinical**
• Brief mental status change, staring spell, blinking
• No aura
• No postictal period
• May occur in clusters




**Treatment**
• Ethosuximide
• Valproic acid

| Absence seizure | Complex partial seizure |
|---|---|
| **Abrupt** onset | Starts as **simple partial seizure** |
| **Shorter** duration | **Longer** duration |
| Ends **abruptly** | Ends **gradually** |
| **No postictal** state | **Postictal** state |

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### C. Status Epilepticus

Pathophysiology
• **Most commonly** caused by a change in the medication regimen of someone with a seizure disorder

Physical Exam
• **Five or more minutes** of continuous seizure activity or more than one seizure without recovery from the postictal state between episodes

Management
1. **Benzodiazepines** (lorazepam, etc.)
2. **Phenytoin** or **fosphenytoin**
3. **Pentobarbital**

## Status Epilepticus

Continuous seizure activity > 5–10 min OR ≥ 2 seizures without full recovery

Always check finger stick blood glucose
Consider pyridoxine (B6) for INH toxicity

**1 First line**
- Lorazepam 2–4 mg IV
- Diazepam 5–10 mg IV
- Midazolam 2–4 mg IV (10 mg IM if no IV access)

**2 Second line**
- Phenytoin or fosphenytoin 20 mg/kg IV
  (Phenytoin max infusion 50 mg/min)
  (Fosphenytoin max infusion 150 mg/min)
- Valproic acid 20 mg/kg IV
- Phenobarbital 20 mg/kg IV at 50 mg/minute
- Levetiracetam 2,000–4,000 mg over 15 min

**3 Third line**
- Pentobarbital (5–15 mg/kg bolus then 0.5–10 mg/kg/hr infusion)
- Propofol (3–5 mg/kg bolus then 1–15 mg/kg/hr infusion)

Notes


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### D. Generalized Tonic-Clonic Seizure

**Tonic phase**
- Sudden muscle stiffening
- Impaired consciousness
- Falling to ground



**Clonic phase**
- Rhythmic jerking
- Usually involve the arms, neck, and face



**Postictal phase**
- Confusion
- Surpassed alertness
- May have focal neurologic deficits
- Consistent improvement in symptoms



Notes


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### E. Febrile Seizure

| Febrile Seizure | | | | | | |
|---|---|---|---|---|---|---|
| **Classification** | **Age** | **# of Seizures** | **Duration** | **Type of Seizure** | **Resolution** | **Return to baseline** |
| **Simple febrile** | 6 months to 5 years | 1 | < 15 min | No focal features | Medication not required | Yes |
| **Complex febrile *without* concerning features** | 6 months to 5 years | 2-3 febrile seizures in the preceding 24 hours | < 15 min | No focal features | Medication not required | Yes |
| **Complex febrile seizure *with* concerning features** | 6 months to 5 years | 4 or more febrile seizures in 24 hours | > 15 min duration or > 5 min in duration plus benzo | Focal features | Medication required | Failure to return to baseline in a reasonable time |

**Criteria for Febrile Seizure**

1. Convulsion associated with temperature > 38°C
2. Child older than 6 months and younger than 5 years of age
3. Absence of CNS infection or inflammation
4. Absence of acute systemic metabolic abnormality that may produce convulsions
5. No history of previous afebrile seizures

Notes


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# XV. Spinal Cord Injury

### A. Back Strain/Sprain

Pathophysiology
• Caused by an injury to the **paravertebral spinal muscles**

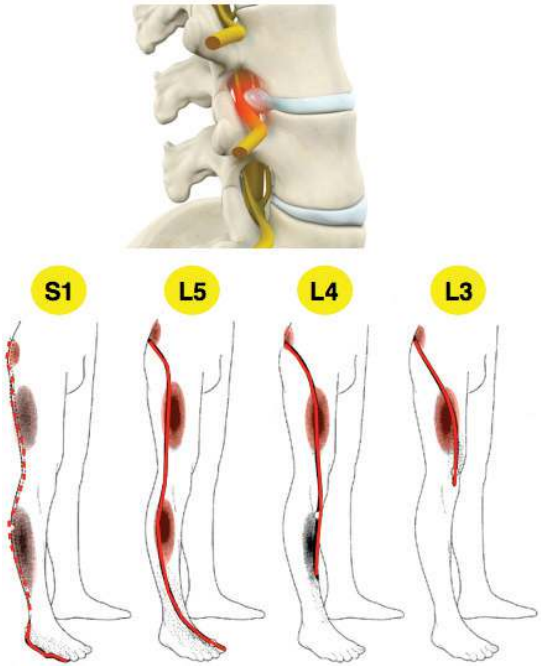
Patient
• History of repeated lifting and twisting

Physical Exam
• Diffuse tenderness in the lower back or SI region with decreased ROM, especially flexion

Diagnostic Studies
• X-ray indicated only if "red flags" are present: pain at rest, pain at night, weight loss, acute bony tenderness, or significant trauma

Management
• Conservative management with continued activity as tolerated, NSAIDs, physical therapy (avoid opioids)

### B. Lumbar Radiculopathy




Notes


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### C. Pharmacologic Therapy for Acute Low Back Pain

| | |
|---|---|
| **Initial Monotherapy** *(2 to 4 weeks)* | • NSAIDS<br>• Acetaminophen (if NSAID contraindication) |
| **Second-line Combination Therapy** | Combination with muscle relaxants<br>• Cyclobenzaprine<br>• Diazepam<br>• Methocarbamol<br>• Carisoprodol |
| **Refractory or Severe Pain** | • Opioids<br>• Tramadol |
| **Other** | • Antidepressants<br>• Glucocorticoids<br>• Antiepileptics |
| **Adjunctive** | • Exercise<br>• Physical therapy<br>• Spinal manipulation<br>• Acupuncture |

Notes


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**D. Red Flags of Back Pain**



1. Night pain and weight loss **(Tumor)**
2. Fevers, chills, and sweats **(Bone or disk infection)**
3. Acute bony tenderness **(Fracture)**
4. Morning stiffness > 30 min in young adult **(Seronegative spondyloarthropathy)**
5. Urinary/bowel retention, saddle anesthesia **(Cauda equina)**
6. Recent spinal instrumentation **(Spinal epidural abscess or hematoma)**
7. Immunodeficiency **(Bone or disk infection)**
8. Extremes of age
9. Coagulopathy **(Spinal epidural hematoma)**

Notes

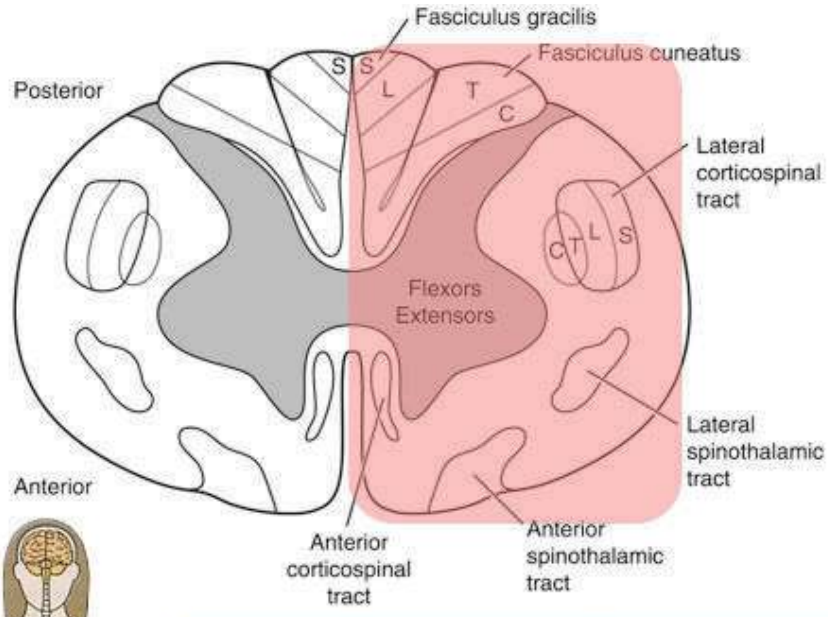

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### E. Brown-Séquard Syndrome

Pathophysiology
- Caused by **spinal cord hemisection**

Patient
- History of penetrating trauma

Physical Exam
- **Ipsilateral** loss of **motor**, **position**, and **vibratory** sensation
- **Contralateral** loss of **pain** and **temperature** sensation



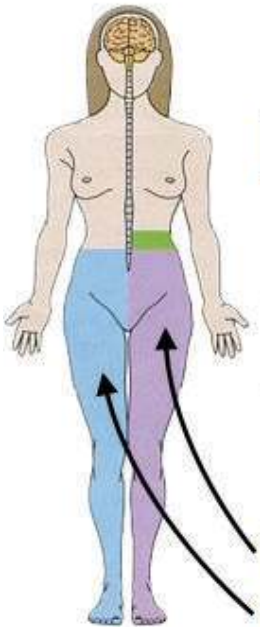

**Causes**

Trauma (gunshot wound, stabbing)

Spinal cord tumor

Ischemia

Infection



| Syndrome | Mechanism | Clinical | Prognosis |
|---|---|---|---|
| Anterior cord | Flexion or vascular | Complete loss of motor, pain, & temperature below injury, but retains proprioception and vibratory sensation | Poor |
| Central cord | Forced hyperextension | Sensory and motor deficit<br>Upper > Lower Extremities | Average |
| **Brown-Séquard** | **Penetrating trauma** | **Ipsilateral loss of motor, vibratory sensation, and proprioception with contralateral loss of pain and temperature sensation** | **Good** |

**Ipsilateral loss of motor, vibratory, and proprioception**

**Contralateral loss of pain and temperature sensation**

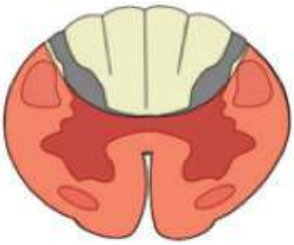
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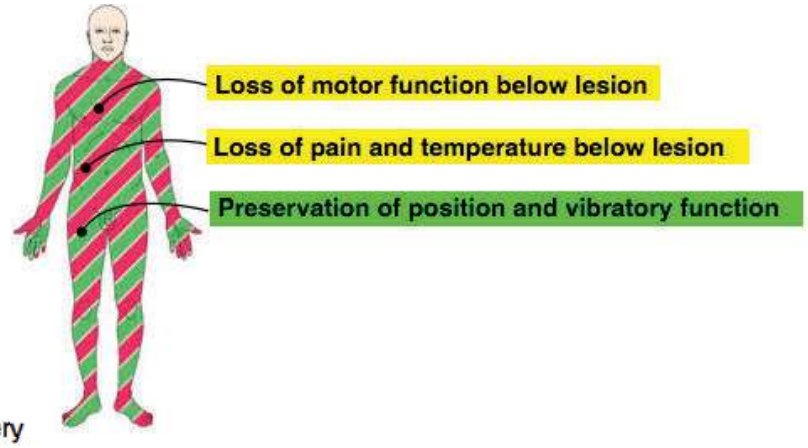

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## F. Anterior Cord Syndrome

Pathophysiology
- Caused by **flexion injury**

Physical Exam
- Loss of **motor**, **pain**, and **temperature** sensation below injury
- **Proprioception** and **vibratory** sensation intact






**Mechanism**

*Direct injury*
- Crush or compression from hematoma

*Indirect injury*
- Ischemia from compression of anterior spinal artery

| Syndrome | Mechanism | Clinical | Prognosis |
|---|---|---|---|
| **Anterior cord** | **Flexion or vascular** | **Complete loss of motor, pain, & temperature below injury, but retains proprioception and vibratory sensation** | **Poor** |
| Central cord | Forced hyperextension | Sensory and motor deficit<br>Upper > Lower Extremities | Average |
| Brown-Séquard | Penetrating trauma | Ipsilateral loss of motor, vibratory sensation, and proprioception with contralateral loss of pain and temperature sensation | Good |

Notes


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## G. Central Cord Syndrome

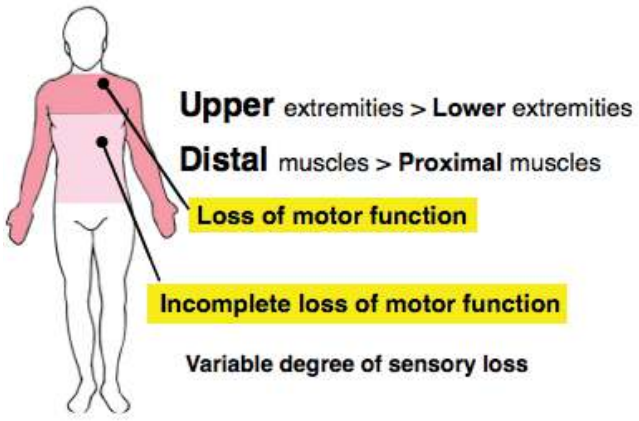
Patient
- History of **hyperextension** injury

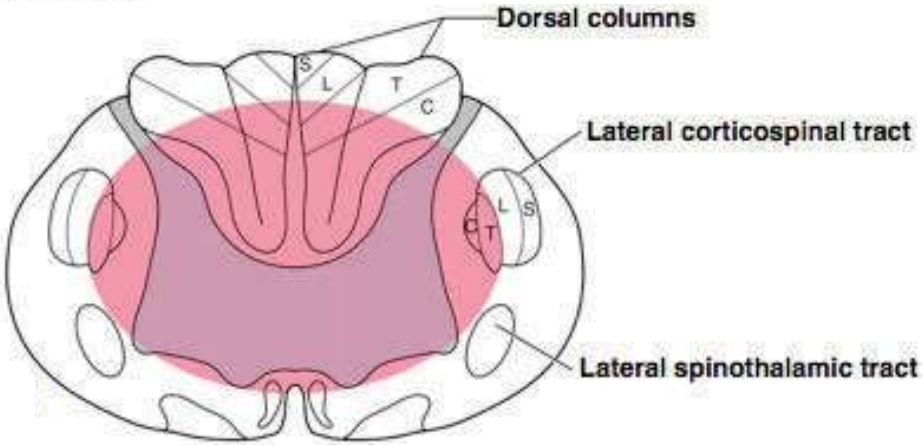
Presentation
- **Bilateral** motor/sensory deficits
- Affecting **upper extremities** more significantly than lower



Most commonly caused by **extension** injury

Comments
- **Most common** incomplete spinal cord syndrome




Notes


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# XVI. Stroke

### A. Stroke Distribution

• **Anterior** Cerebral (Frontal and Parietal): hemiparesis affecting **leg** more than arm

• **Middle** Cerebral (Frontal, Temporal and Parietal): hemiparesis affecting **arm and face** more than leg, **aphasia** in dominant hemisphere

• **Posterior** Cerebral (Occipital): **homonymous hemianopsia**

• **Carotid** Circulation (Hemispheric): emiplegia, hemianesthesia, neglect, aphasia, visual field defects; less often headaches, seizures, amnesia, confusion

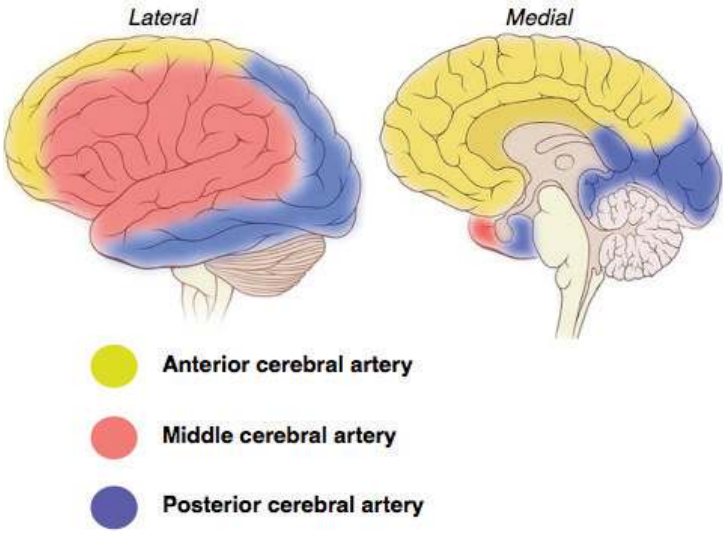
• **Vertebrobasilar** (Brainstem or Cerebellar): diplopia, vertigo, ataxia, facial paresis, Horner syndrome, dysphagia, dysarthria, impaired level of consciousness

• **Cerebellar**: headache, nausea, vomiting, and ataxia

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### B. Anterior Cerebral Artery

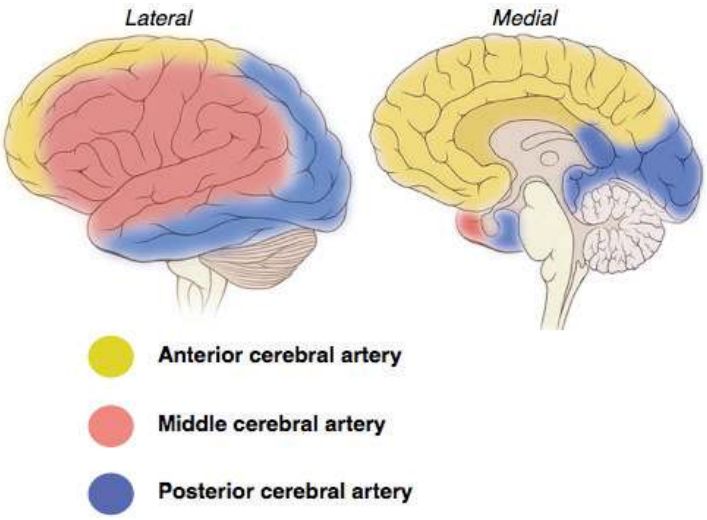



**Anterior cerebral artery**
- Paralysis of contralateral foot and leg
- Sensory loss over toes, foot and leg
- Impairment of gait and stance
- Abulia (slowness and prolonged delays to perform acts)
- Flat affect, lack of spontaneity, slowness, distractibility
- Cognitive impairment
- Urinary incontinence

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### C. Middle Cerebral Artery

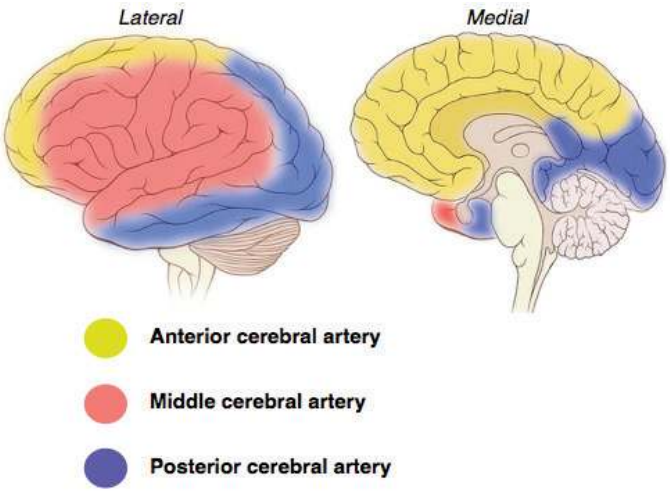



**Middle cerebral artery**
- Paralysis of contralateral face, arm, and leg
- Sensory impairment over contralateral face, arm, and leg
- Homonymous hemi- or quadrantonopia
- Paralysis of gaze to opposite side
- Aphasia, dysarthria

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### D. Posterior Cerebral Artery




**Posterior cerebral artery**

***Peripheral (cortical)***
- Homonymous hemianopia
- Memory deficits
- Perseverations
- Visual deficits

***Central (penetrating)***
- Thalamus: contralateral sensory loss, spontaneous pain
- Cerebral peduncle: CN III palsy with contralateral hemiplegia
- Brain stem: CN palsies, nystagmus, pupillary abnormalities

Notes


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# XVII. Subarachnoid Hemorrhage/Cerebral Aneurysm

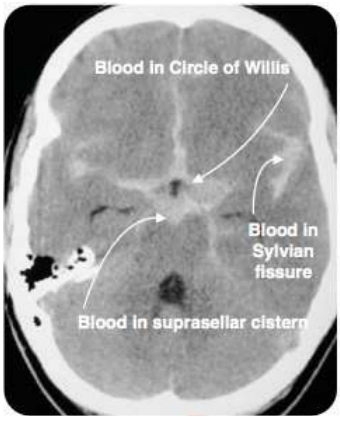
## A. Subarachnoid Hemorrhage

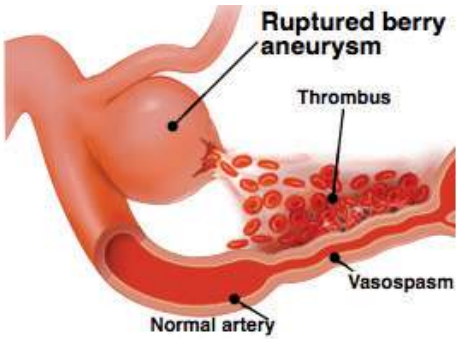
Pathophysiology
• **Most commonly** caused by a **ruptured aneurysm**

Presentation
• Abrupt onset of **"worst headache of their life,"** or **"thunderclap"** headache

Diagnostic Studies
• Noncontrast CT scan is diagnostic
If CT negative and suspicion high, lumbar puncture

Management
• Supportive care and nimodipine (decreases vasospasm)




**Management of vasospasm**
• Nimodipine 60 mg every 4 hours
• Ideally given within 4 days of aneurysmal rupture
• Must be given orally (or NG tube)

Notes


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| Time Following Subarachnoid Hemorrhage | CT Scan Sensitivity |
|---|---|
| 0 - 12 hrs | 98% - 100% |
| 24 hrs | 93% |
| 6 days | 57% |

Notes


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# XVIII. Syncope

## A. Syncope

Pathophysiology
• Acute, **transient** loss of consciousness, frequently related to inadequate blood supply to the brain
• Commonly caused by **cardiac dysfunction** (e.g., aortic stenosis, dysrhythmias, decreased stroke volume), **vasovagal response**, orthostatic hypotension, and cerebrovascular ischemia

Presentation
• There may be a prodrome of lightheadedness, nausea, and weakness preceding loss of consciousness (i.e., fainting)
• Few generalized spasms may be observed
• Patient quickly regains consciousness
• Hypotension, dysrhythmias, and neurologic deficits may be detected following the episode

Diagnostic Studies
• **Labs**:
  • glucose levels should be measured
  • orthostatics or tilt testing (i.e., patient response is measured with rapid posture changes) with or without BB infusion can help diagnose cardiovascular cause
  • echocardiography or stress testing may be useful
• **ECG**: useful for diagnosing **cardiac** causes; ambulatory monitoring may be used to monitor extended periods of time
• **EEG**: useful for ruling out alternative diagnosis
• **MRI**, **MRA**, or **angiography** may detect **vascular** abnormalities

Notes


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**B. Causes of Syncope**

| Syncope | | |
|---|---|---|
| **Reflex (neurally mediated)** | **Vasovagal**<br>• Mediated by orthostatic or emotional stress | **Situational**<br>• Cough, sneeze, micturition, post-exercise, post-prandial |
| | **Carotid sinus** | **Unknown** |
| **Orthostatic** | **Primary autonomic**<br>• Pure autonomic, Parkinson's, Lewy body dementia | **Secondary autonomic**<br>• Diabetes, amyloidosis, uremia, spinal cord injuries |
| | **Drug induced**<br>• Alcohol, vasodilators, diuretics, phenothiazines, antidepressants | **Volume depletion**<br>• Hemorrhage diarrhea, vomiting |
| **Cardiac** | **Bradydysrhythmia**<br>• AV block, tachy-brady syndrome | **Tachdysrhythmia**<br>• Supraventricular tachycardia, ventricular tachycardia |
| | **Structural heart disease**<br>• Drug-induced, valvular disease, ischemia, cardiomyopathy, tamponade | **Other**<br>• Pulmonary embolism, aortic dissection, pulmonary hypertension |

Notes


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# XIX. Transient Ischemic Attack

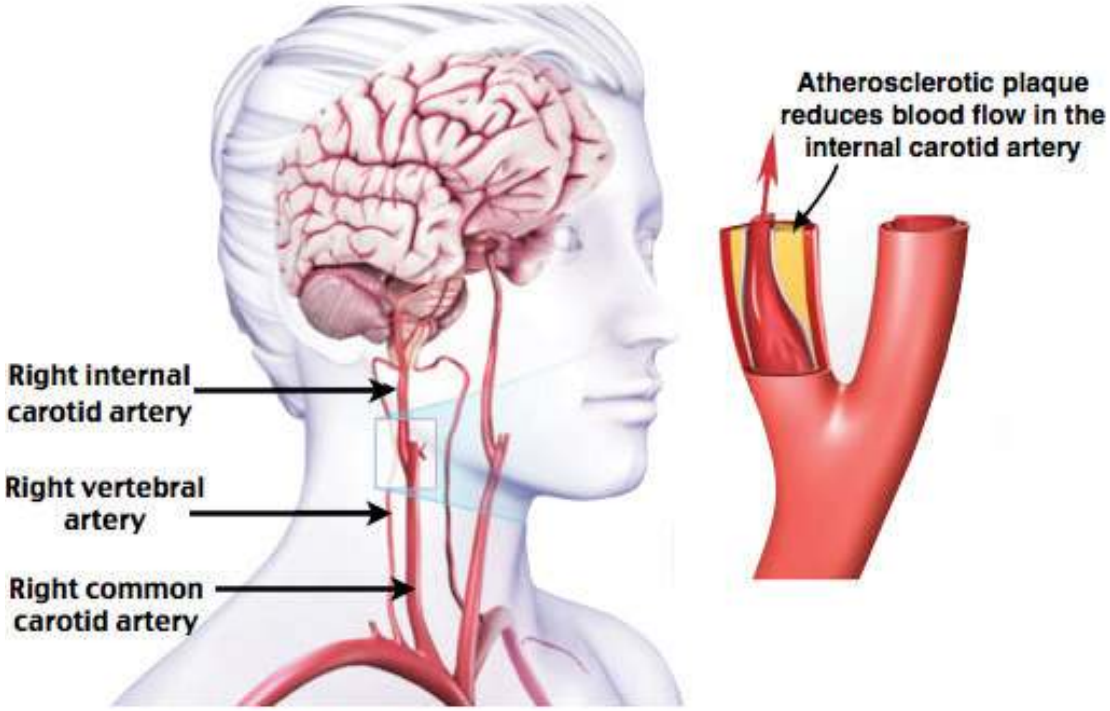
### A. Transient Ischemic Attack

Pathophysiology
• Transient episode of neurological dysfunction **without acute infarction**

Comments
• 10% of TIA patients will have a stroke within **90 days**
• **ABCD2 score**: predicts the likelihood of subsequent stroke within two days

Management
• **Aspirin + dipyridamole** or clopidogrel monotherapy

**Transient Ischemic Attack (TIA)**
A **transient** episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal **ischemia**, without acute infarction




Notes


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# XX. Vertigo

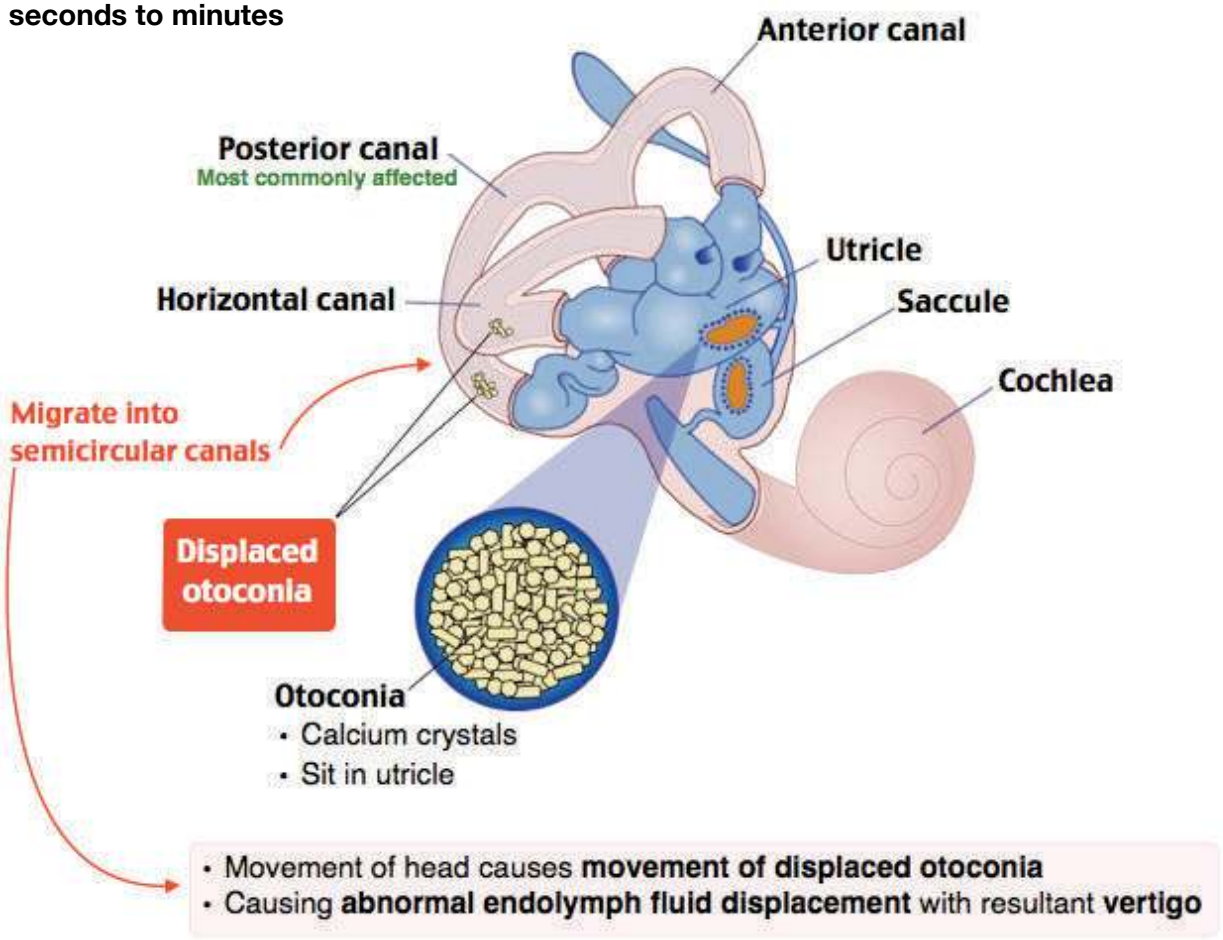
### A. Benign Paroxysmal Positional Vertigo

Pathophysiology
- **Most commonly** caused by **displaced otoliths** in the labyrinth system

Presentation
- **Sudden-onset room-spinning** sensation in connection with **positional changes of the head**, lasting **seconds to minutes**

Physical Exam
- **Positive Dix-Hallpike maneuver**

Management
- **Epley maneuver**




Notes


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**B. Dix-Hallpike Maneuver**

Tests for **canalithiasis** of the **posterior semicircular canal**, which is the **most common cause of benign paroxysmal positional vertigo (BPPV)**



1. With the patient sitting up, turn the head 45 degrees to one side
2. Lie the patient down with head overhanging the edge of the bed and look for nystagmus
3. Repeat on the contralateral side

**Positive** if the maneuver **provokes paroxysmal vertigo and nystagmus**

Notes


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**C. Epley Maneuver**

Used to treat **benign paroxysmal positional vertigo** of the posterior or anterior canals

1
- Begin in upright sitting posture
- Legs extended
- Head rotated 45 degrees towards the side of positive Dix-Hallpike test



2



- Patient is quickly and passively forced down backwards into supine position
- Head held approx. in a 30-degree neck extension

3



- Observes patient's eyes for primary stage nystagmus
- Patient remains in this position for 1 - 2 minutes

4



- Patient's head rotated 90 degrees to opposite direction
- Patient remains in this position for 1 - 2 minutes

5



- Patient rolls onto shoulder, rotating head 90-degrees
- Patient now looking downward at 45-degree angle
- Observe for secondary stage nystagmus
- Remains in position for 1 - 2 minutes

6



- Patient slowly brought upright, maintaining 45-degree rotation of head
- Patient holds seating position for 30 seconds

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**D. Central vs. Peripheral Vertigo Characteristics**

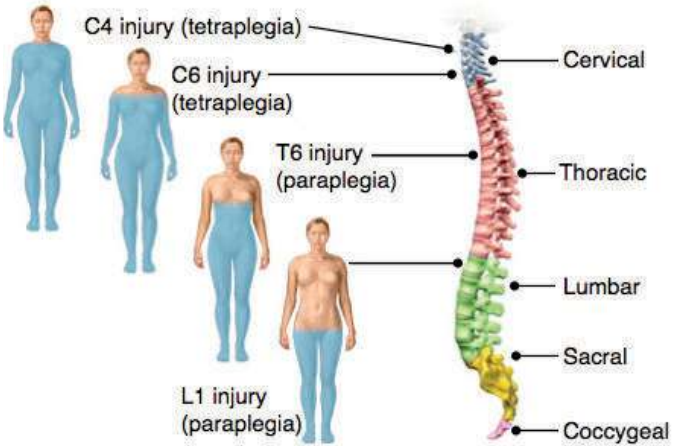
| Characteristic | Peripheral | Central |
|---|---|---|
| **Onset** | Sudden | Gradual or Sudden |
| **Duration** | Seconds to minutes (intermittent) | Variable (can be subacute and progressive or brief and intermittent) |
| **Intensity** | Severe | Mild |
| **Effect of head position** | Worsened by position | Minimal change |
| **Direction of nystagmus** | Unidirectional (never vertical) | Horizontal, vertical, rotary, and bidirectional |
| **Any neurologic findings** | No | Often |
| **Any auditory findings** | Occasionally | No |
| **Causes** | • BPPV<br>• Acute otitis media<br>• Labyrinthitis<br>• Mèniére disease<br>• Vestibular neuronitis<br>• Trauma | • Meningitis<br>• Encephalitis<br>• Vertebral basilar insufficiency<br>• Cerebellar hemorrhage<br>• Temporal lobe epilepsy<br>• Tumors |

Notes


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# XXI. Weakness/Paralysis

### A. Traumatic Spinal Cord Injury




**Clinical**
- Complete cord injury (ASIA grade A)
- Rostral one of spared sensory levels (e.g., C5 and higher dermatomes spared in a C5-6 fracture dislocation)
- Reduced sensation in the next caudal level
- No sensation in levels below, including none in the sacral segments S4-S5
- Reduced muscle power in the level immediately below the injury
- Complete paralysis in more caudal myotomes
- Reflexes absent (acute stage)
- No response to plantar stimulation
- Muscle tone is flaccid
- Priapism (men)
- Bulbocavernous reflex usually absent
- Urinary retention and bladder distention occur

Notes


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## B. Tick Paralysis

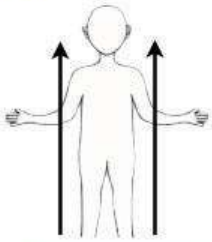


**Tick Vectors**
- *Dermacentor andersoni* (Rocky Mountain wood tick)
- *Dermacentor variabilis* (American dog tick)



**Pathogenesis**
- Female tick begins to feed
- Symptoms begin 4 to 7 days later
- Tick salivary toxin has pathologic effects

1. Slowing of motor nerve conduction velocity
2. Lowering of the height of the nerve and muscle action potential
3. Impaired propagation of afferent nerve fiber signals



**Ascending Paralysis**

**Ataxia**

**Management**
- Remove tick
- Symptoms resolve within hours

Notes


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CHAPTER 6

# ENOT/ Ophthalmology

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CHAPTER 6

# ENOT/Ophthalmology


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334 **RETINAL DETACHMENT**

335 **RETINAL VEIN OCCLUSION**

337 **TRAUMA/HEMATOMA (EXTERNAL EAR)**

338 **TYMPANIC MEMBRANE PERFORATION**

339 **VERTIGO**

340 **VISION LOSS**



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# I. Acute Laryngitis

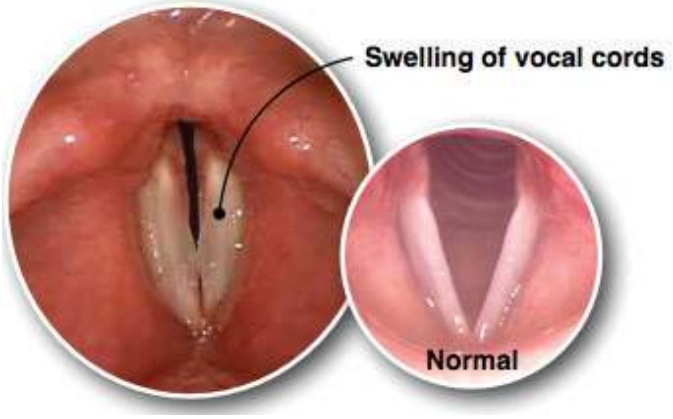
**A. Laryngitis**

Pathophysiology
• Viral **more common** than bacterial
• Can be related to acute vocal cord strain

Presentation
• Hoarseness

Management
• Self-limiting
• Humidified air, hydration, vocal rest

**Acute Laryngitis**
**Self-limited lasting** inflammatory condition **< 3 weeks**




**Etiology**
• Upper respiratory infection
  Viral (most common)
  Bacterial: *M. catarrhalis, H. influenzae, S. pneumoniae*
• Acute vocal cord strain

**Management**
• Self-limited
• Hydration, humidification, voice rest

Notes


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# II. Acute Otitis Media

### A. Acute Otitis Media

Pathophysiology
• **Viral** infections **more common** than bacterial
• If bacterial, *S. pneumoniae*, *H. influenzae*, *M. catarrhalis* are **most common** pathogens

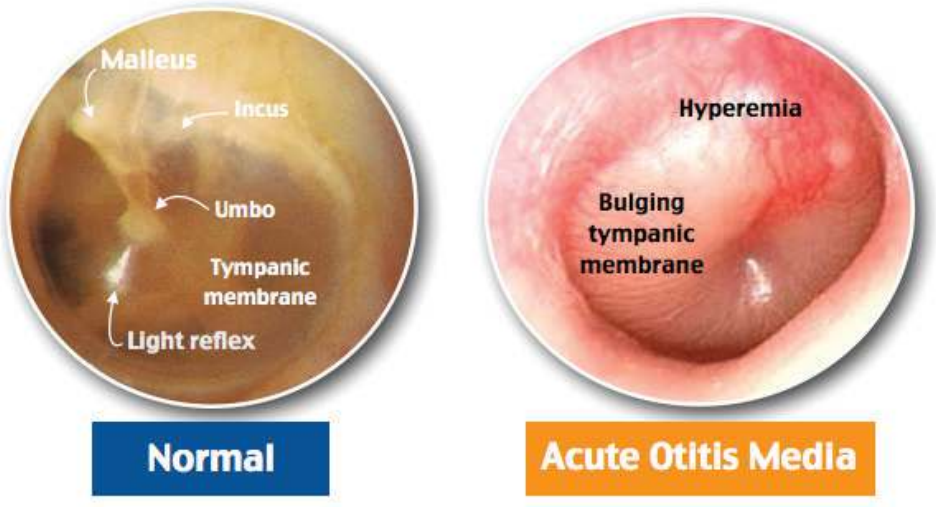
Patient
• Infant or young child

Presentation
• Ear pain, fever, URI symptoms

Physical Exam
• TM erythema and **decreased mobility of TM** on insufflation

Management
• **Amoxicillin**

**Most common bacterial causes**
• *Streptococcus pneumoniae*
• *Haemophilus influenzae* (non-typeable)
• *Moraxella catarrhalis*




Notes


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# III. Acute Pharyngitis (Viral, Bacterial)

## A. Pharyngitis

Pathophysiology
- **Viral** infection **more common** than bacterial
- If bacterial, **group A *Streptococcus*** is **most common** pathogen

Presentation
- Sore throat, **no cough**

Physical Exam
- Fever, cervical lymphadenopathy, tonsillar exudate

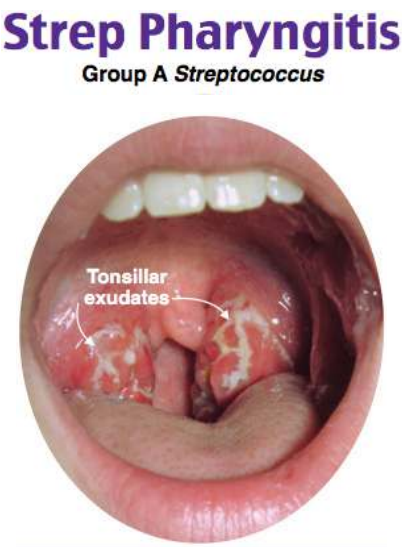
Diagnostic Studies
- Rapid strep test or culture if indicated by **CENTOR criteria**

Management
- Viral: supportive care
- Bacterial: **penicillin** (erythromycin if penicillin allergy), steroids

## B. Strep Pharyngitis

**Management**
- Supportive
- Penicillins
- Cephalosporins
- Clindamycin
- Macrolides

**Clinical**
- Temp > 38°C
- Tender anterior cervical lymphadenopathy
- Absence of cough
- Pharyngotonsillar exudates




Notes


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**C. Modified Centor Criteria**

| | |
|---|---|
| Absence of cough | +1 |
| Swollen and tender cervical lymphadenopathy | +1 |
| Fever by history | +1 |
| Tonsillar exudates or swelling | +1 |
| Age < 15 years | +1 |
| Age ≥ 45 years | -1 |

| Recommendation | Probability of strep pharyngitis | Centor Score |
|---|---|---|
| No further testing or antibiotics | 1 to 2.5% | -1 to 0 |
| | 5 to 10% | 1 |
| Optional rapid strep testing or culture or both | 11 to 17% | 2 |
| Consider rapid strep testing or culture or both | 28 to 35% | 3 |
| | 51 to 53% | > 4 |

*IDSA and ASIM no longer recommend empiric treatment for strep based on symptomatology alone*

Notes


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**D. Clinical Features of Group A *Streptococcus* Pharyngitis and Viral Pharyngitis**

| Group A *Streptococcus* pharyngitis | Viral pharyngitis |
|---|---|
| Sudden onset of sore throat | Cough (often with fever and malaise) |
| Fever | Nasal congestion |
| Tonsillopharyngeal and/or uvular edema | Coryza |
| Patchy tonsillar pharyngeal exudates | Conjunctivitis |
| Anterior cervical adenitis (tender lymph nodes) | Hoarseness |
| Scarlatiniform skin rash (Scarlet fever) | Oral ulcers |
| History of group A *Streptococcus* exposure | Viral exanthema |

Notes


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# IV. Acute Sinusitis

### A. Sinusitis

Pathophysiology
• **Most commonly viral** in etiology
• If symptoms persist longer than **10–14 days**, pathogen is more likely to be bacterial

Presentation
• Pain over sinuses and rhinorrhea

Physical Exam
• Purulent rhinorrhea and **sinus tenderness**

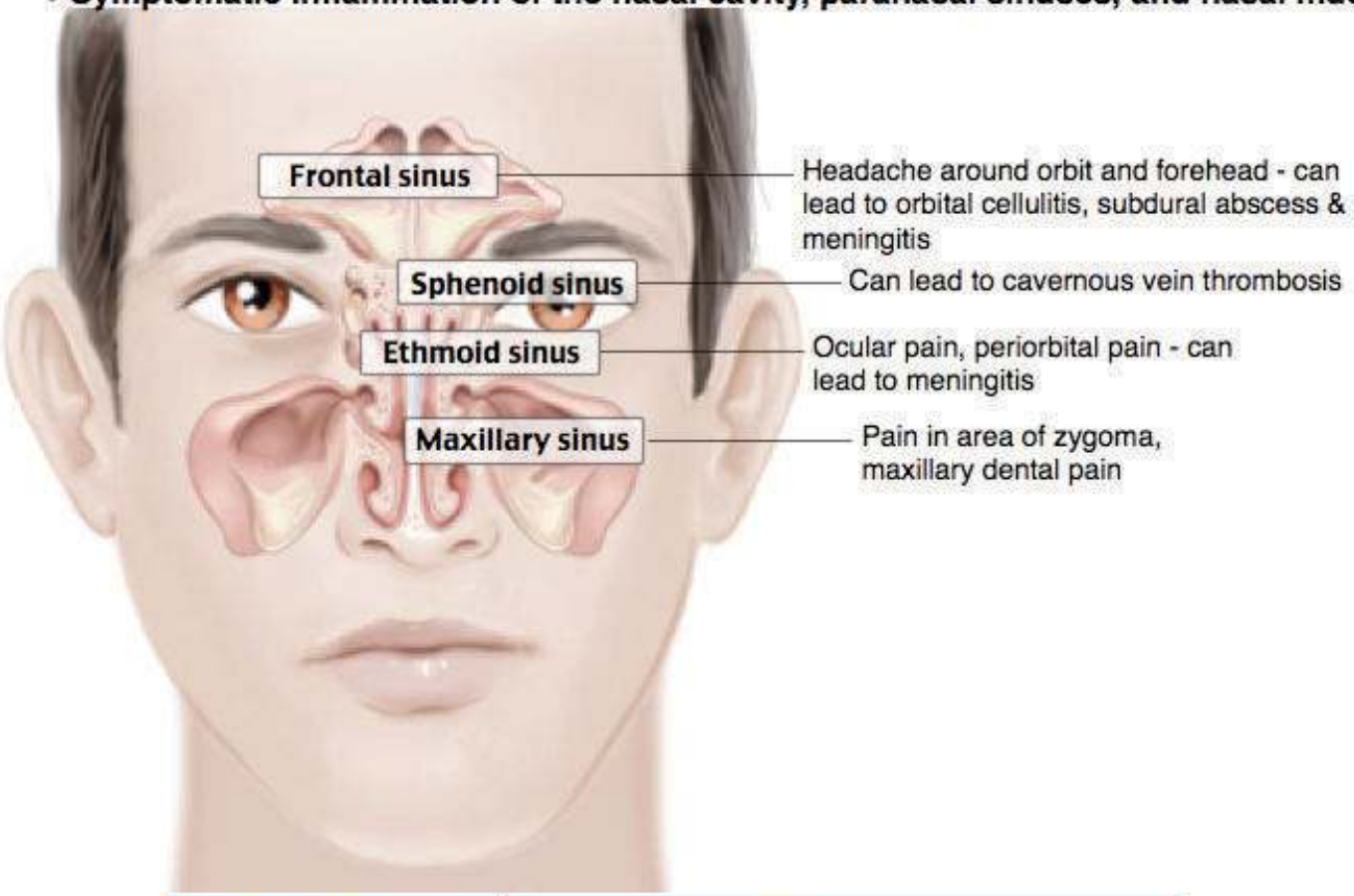
Management
• Supportive care
• If bacterial sinusitis is suspected, **amoxicillin-clavulanate**

Notes


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**B. Rhinosinusitis**

**Symptomatic inflammation of the nasal cavity, paranasal sinuses, and nasal mucosa**




| Classification | Duration |
|---|---|
| Acute | Symptoms < 4 weeks |
| Subacute | Symptoms for 4 to 12 weeks |
| Chronic | Symptoms > 12 weeks |
| Recurrent acute | ≥ 4 episodes per year with interim sx resolution |

Notes


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# V. Allergic Rhinitis

### A. Allergic Rhinitis

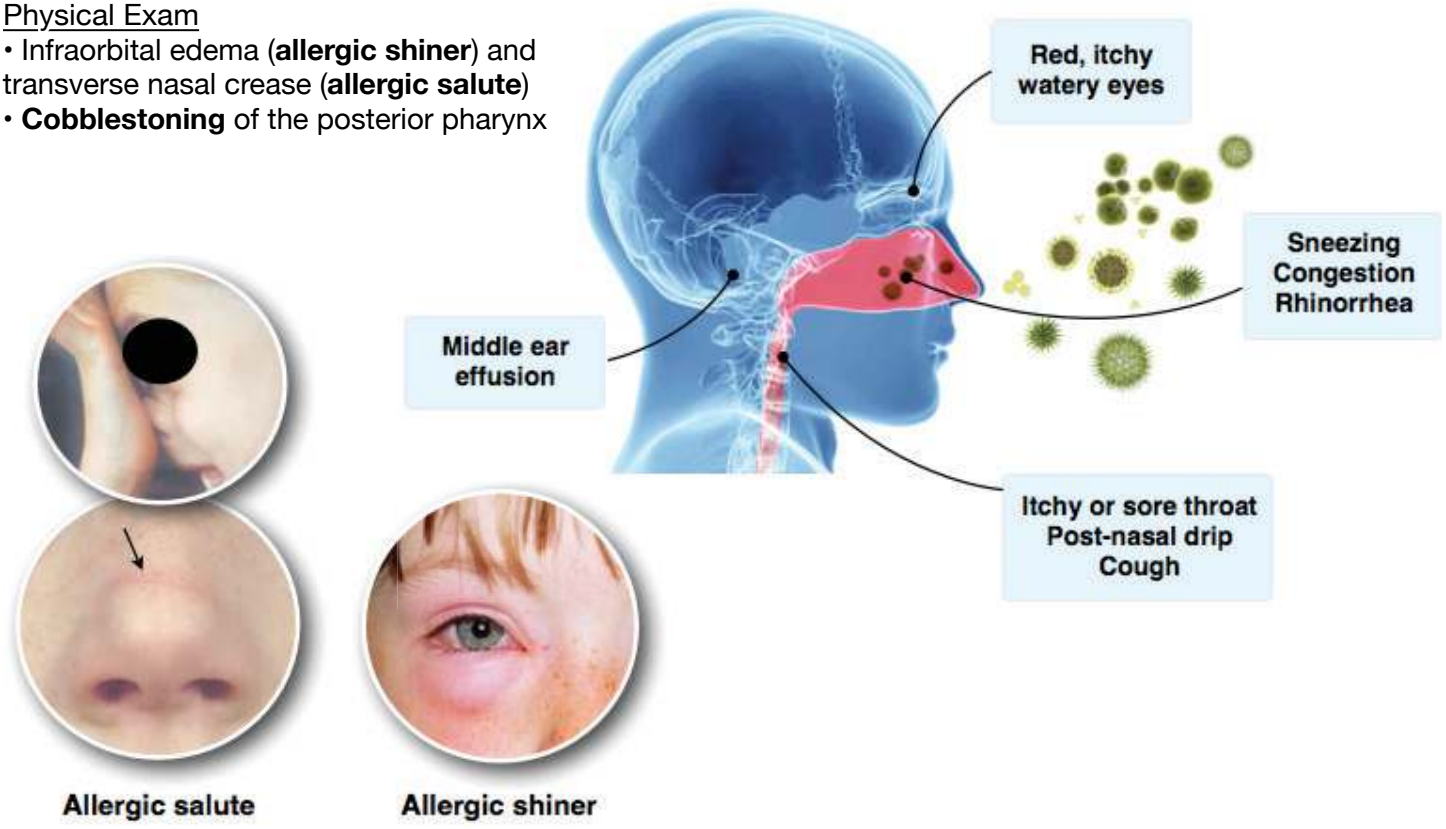
Patient
• History of asthma and atopic dermatitis

Presentation
• Sneezing, rhinorrhea, nasal congestion

Physical Exam
• Infraorbital edema (**allergic shiner**) and transverse nasal crease (**allergic salute**)
• **Cobblestoning** of the posterior pharynx

Diagnostic Studies
• **Elevated** serum **IgE**

Management
• Glucocorticoid nasal spray




Notes

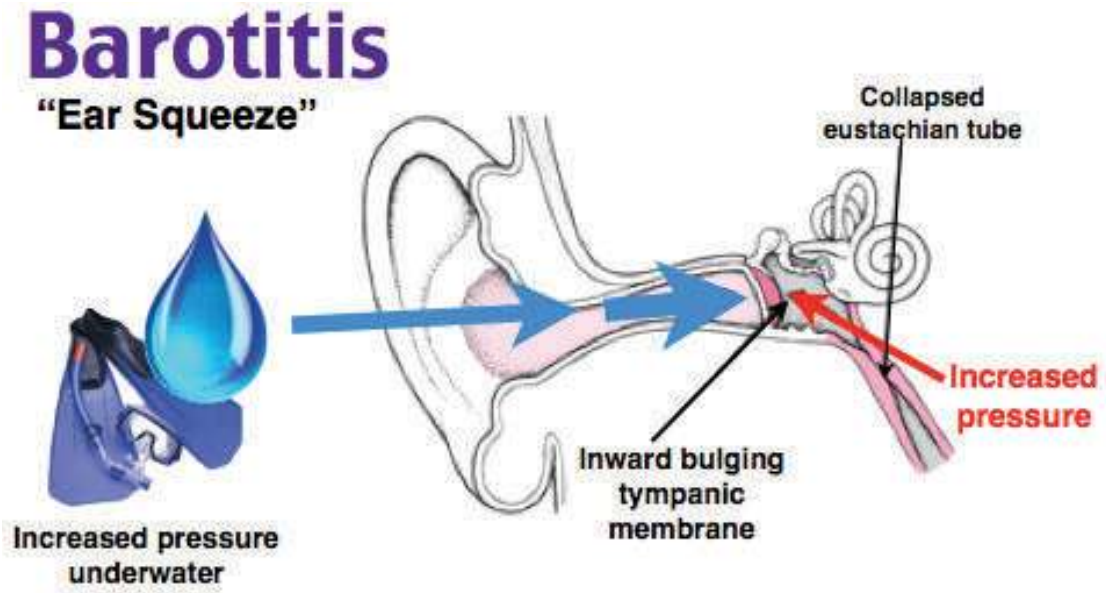

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# VI. Barotrauma

### A. Barotitis

Pathophysiology
• Ear pain and hearing loss that **persists past the inciting event**, associated with pressure changes
• Common injury in divers or while flying, sudden onset of pain that may resolve with a **"pop"**

**Clinical**
• Ear pain
• Hearing loss
• Tympanic membrane rupture
• Nystagmus
• Seventh nerve palsy




Notes


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# VII. Blepharitis

### A. Blepharitis

Pathophysiology
• **Most commonly** caused by **dysfunctional meibomian gland**

Presentation
• Eyelid changes and eyelash flaking

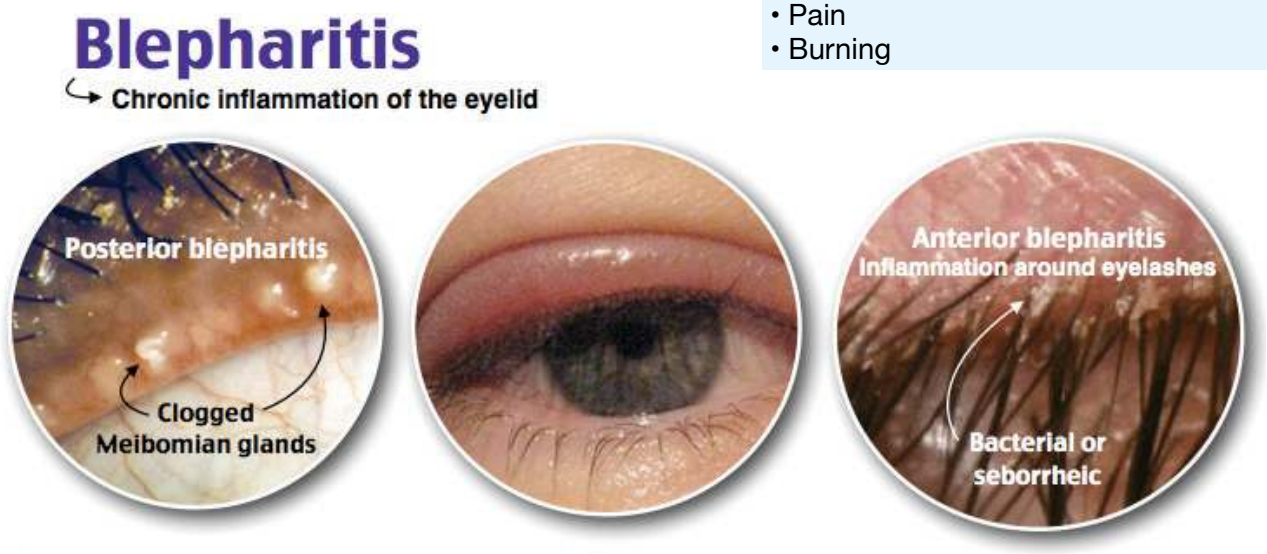
Physical Exam
• Crusting, **scaling**, **red-rimming** of eyelid

Diagnostic Studies
• Slit-lamp exam

Management
• **Warm compresses**, irrigation, lid massage
• Topical antibiotics if no improvement

Comments
• Associated with seborrhea and rosacea

**Clinical**
• Watering
• Foreign body sensation
• Crusting of lashes
• Red eyes
• Photophobia
• Pain
• Burning




Notes


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# VIII. Blow-Out Fracture

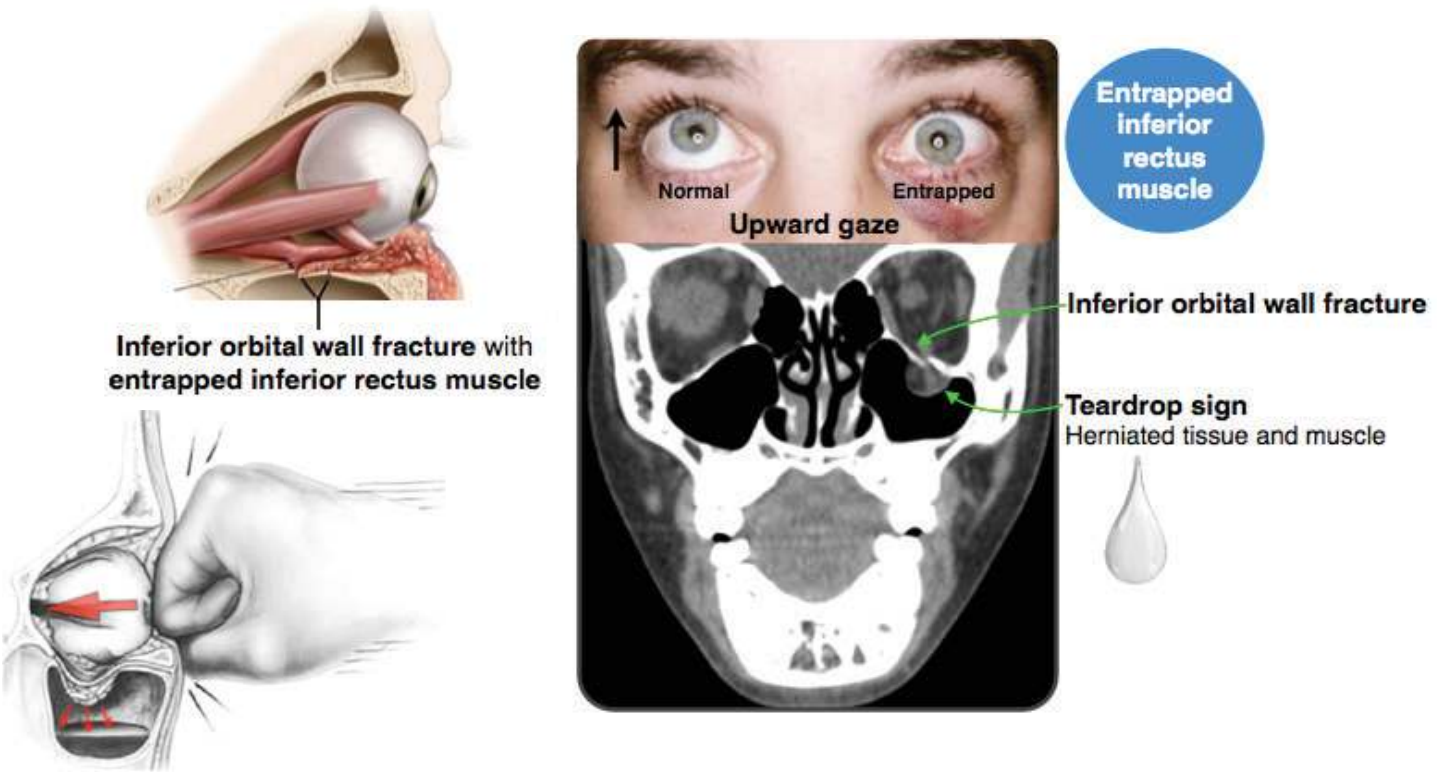
### A. Orbital Blowout Fracture

Patient
• History of direct trauma to the orbit

Presentation
• Limited upward gaze, infraorbital anesthesia

Physical Exam
• Inferior rectus entrapment, enophthalmos

Diagnostic Studies
• CT will show **teardrop sign**




Notes


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# IX. Conjunctivitis

## A. Bacterial Conjunctivitis

Pathophysiology
• **Most commonly** caused by *S. aureus*, *S. pneumoniae*, *H. influenzae*
• **Contact lens wearers: *Pseudomonas aeruginosa***

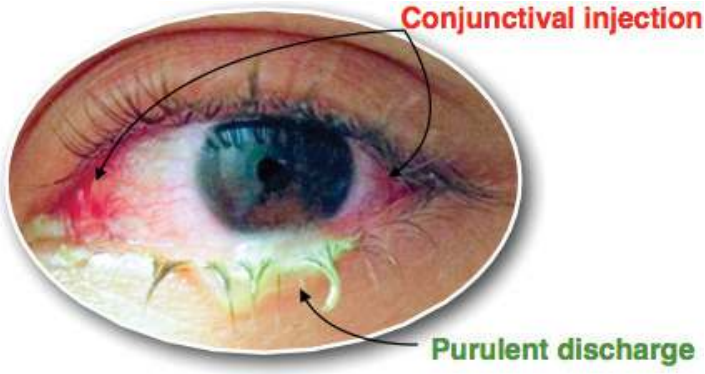
Presentation
• Red or pink eye with discharge, usually **worse in the morning**

Physical Exam
• **Purulent, yellow** discharge and crusting

**Microbiology**
• Staphylococcus aureus
• Streptococcus pneumoniae
• Haemophilus influenzae
• Moraxella catarrhalis

**Treatment**
• Erythromycin ophthalmic ointment
• Trimethoprim-polymyxin B
• Sulfacetamide
• Bacitracin-polymyxin B

Management
• Ophthalmic **antibiotic** drops or ointment
• Antipseudomonal (e.g., moxifloxacin) for contact lens wearers




**Special case**



Prone to ***Pseudomonas aeruginosa*** infection
**Treat** with **fluoroquinolone**

Notes

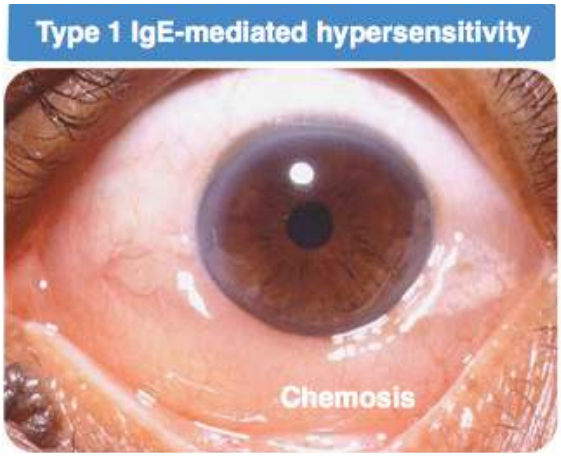

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### B. Allergic Conjunctivitis

Presentation
• Bilateral red eyes, **itching**, tearing

Physical Exam
• **Cobblestone mucosa** on inner and upper eyelid

Management
• Topical **H1 blockers** (e.g., olopatadine)




**Clinical**
• **Pruritis** (prominent symptom)
• **Redness** (universal)
• Conjunctival edema (chemosis)
• Usually bilateral
• Eye pain is NOT characteristic

**Management**
• Basic eye care (cool compresses, artificial tears)
• Topical antihistamine with mast cell-stabilizers
• NSAIDs

Notes


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### C. Viral Conjunctivitis

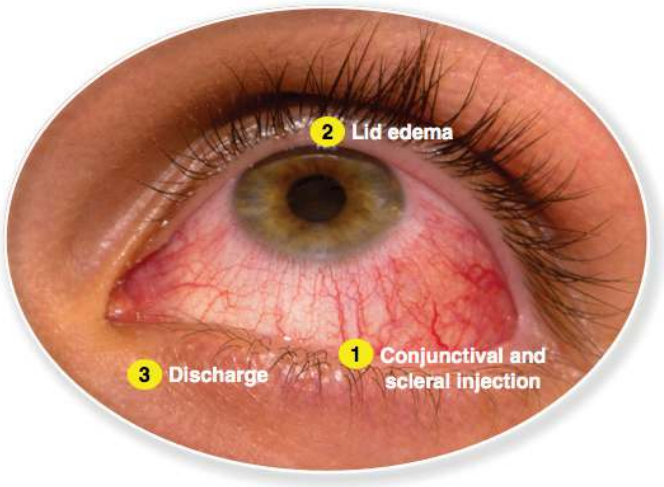
Pathophysiology
• **Most commonly** caused by **adenovirus**

Presentation
• Bilateral red or pink eye

Physical Exam
• Preauricular lymphadenopathy, **copious watery** discharge, **scant mucoid** discharge

Management
• Cool compresses, artificial tears, topical antihistamine/mast cell stabilizer (e.g. olopatadine) for itching and redness

### D. Acute Conjunctivitis




Notes


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# X. Corneal Abrasion/Ulcer

## A. Corneal Abrasion

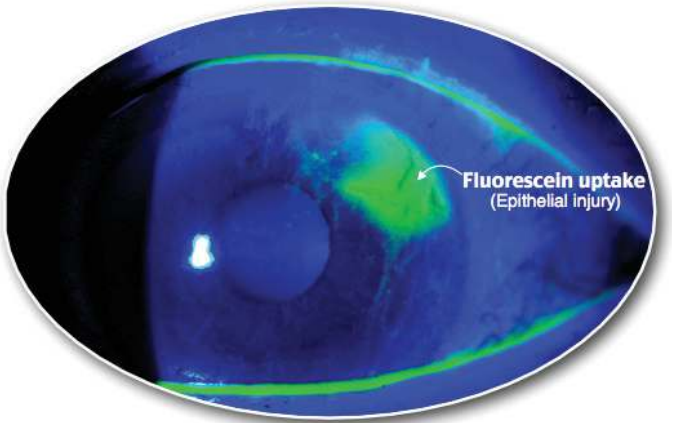
Patient
• Recent trauma to eye

Presentation
• Pain, tearing, photophobia, **foreign body sensation**

Diagnostic Studies
• Slit-lamp exam with **positive fluorescein dye uptake**

Management
• Remove foreign body if present
• Antipseudomonal coverage for **contact lens** wearers

Comments
• A **"zig-zag"** pattern may indicate retained foreign body requiring removal
• **Do not** prescribe **topical anesthetics** as long-term use, may result in **corneal ulcer**




**Clinical**
• Pain, photophobia, tearing
• Foreign body sensation
• Conjunctival injection
• Topical antibiotics
• Antipseudomonal for contact lens wearers

Notes


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**B. Corneal Ulcer**

Pathophysiology
• **Most commonly** caused by *Staphylococcus*, ***Pseudomonas* (contact lens wearers)**, *Streptococcus pneumoniae*

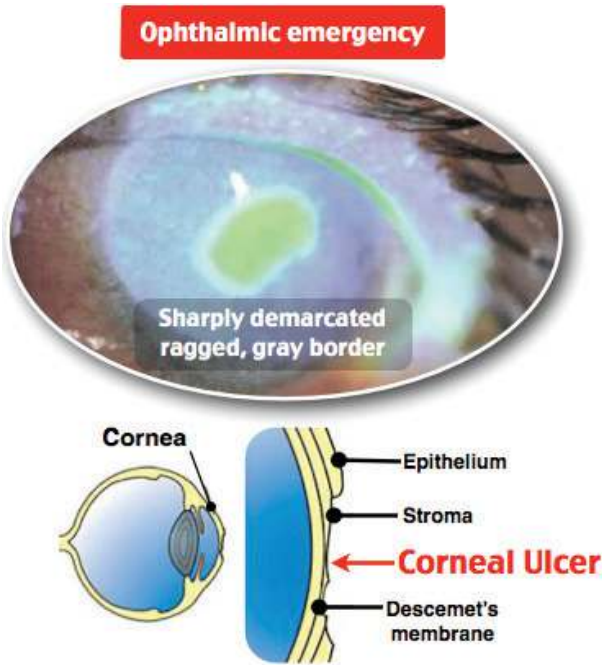
Patient
• History of trauma or extended contact lens use

Presentation
• Pain and **photophobia**

Physical Exam
• Oval ulcer with **ragged edges**, severe conjunctival inflammation

Diagnostic Studies
• **Fluorescein** dye test

Management
• **Emergent** ophthalmology consult




**Clinical**
• Pain
• Foreign body sensation
• Photophobia
• Vision changes
• Eye discharge

Notes


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# XI. Dacryoadenitis

### A. Dacryocystitis

Pathophysiology
• Infectious obstruction of nasolacrimal **duct** (inferomedial region)

Management
• Oral or IV antibiotics depending on severity

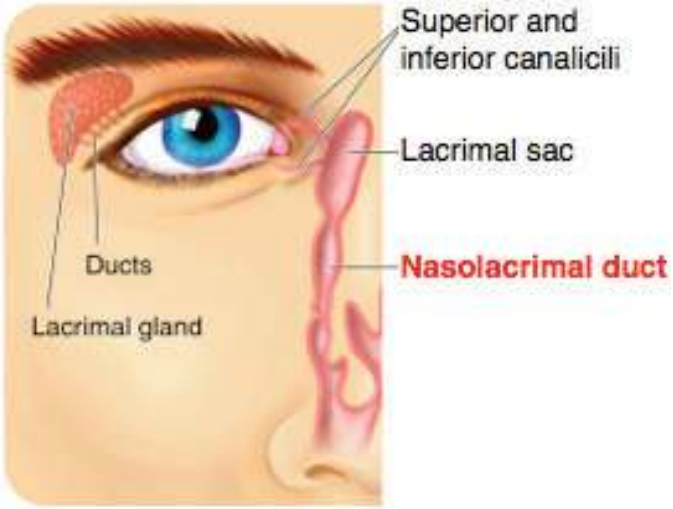
Comments
• Complications include **orbital cellulitis**

**Clinical**
• Unilateral, painful
• Erythema, swelling, warmth
• Tenderness of lacrimal sac
• Possibly purulent discharge
• Epiphora (overflow of tears)

**Complications**
• Preseptal or orbital cellulitis
• Meningitis

**Treatment**
• Mild: Clindamycin
• Severe: Vancomycin, 3rd gen cephalosporin

**Dacryocystitis**
**Infection of the nasolacrimal system**







Notes


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**B. Dacryoadenitis**

• Inflammation of the nasolacrimal **gland** (supratemporal)




Notes


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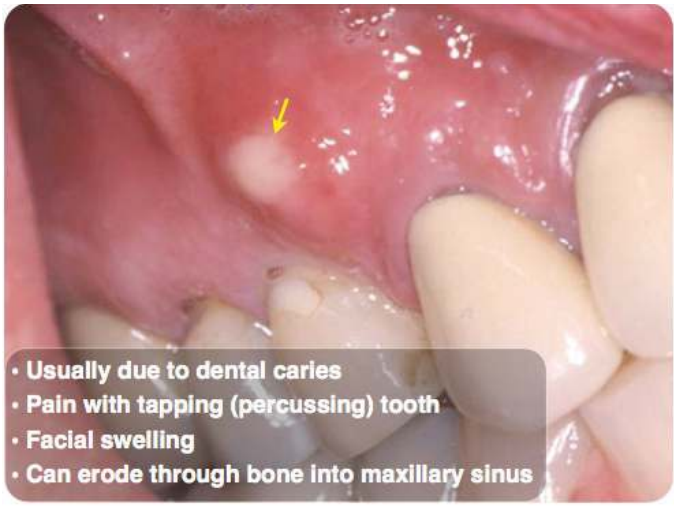
# XII. Dental Abscess

### A. Dental Abscess

Pathophysiology
• Poor dental health is a risk factor for dental abscess or facial cellulitis

Management
• Mild to moderate: penicillin or amoxicillin-clavulanate
• Severe: ampicillin-sulbactam IV

### B. Periapical Abscess




Notes


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# XIII. Epiglottitis

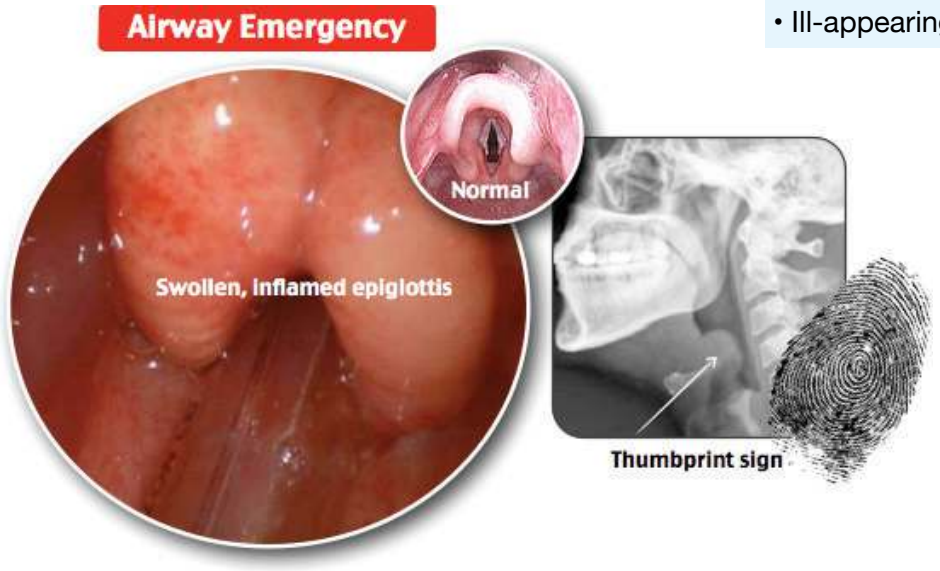
## A. Epiglottitis

Pathophysiology
• **Most commonly** caused by *Strep.* and *Staph.* species
• ***H. influenzae*** (prior to the Hib vaccine)

Patient
• **Unvaccinated** child

Presentation
• Rapid onset of **fever** and **dysphagia**
• **Leaning forward**, **drooling**, **inspiratory stridor**

Diagnostic Studies
• Lateral neck X-ray will show **"thumbprint" sign**




Management
• IV antibiotics and **airway management**

**Etiologies**
• *Haemophilus influenzae* type B (pre HiB vaccine)
• *Streptococcus spp.*
• *Staph. aureus*
• *Moraxella catarrhalis*

**Clinical (Rapid onset)**
• Fever
• Sore throat
• Drooling
• Muffled voice
• Anxious
• Ill-appearing

Notes

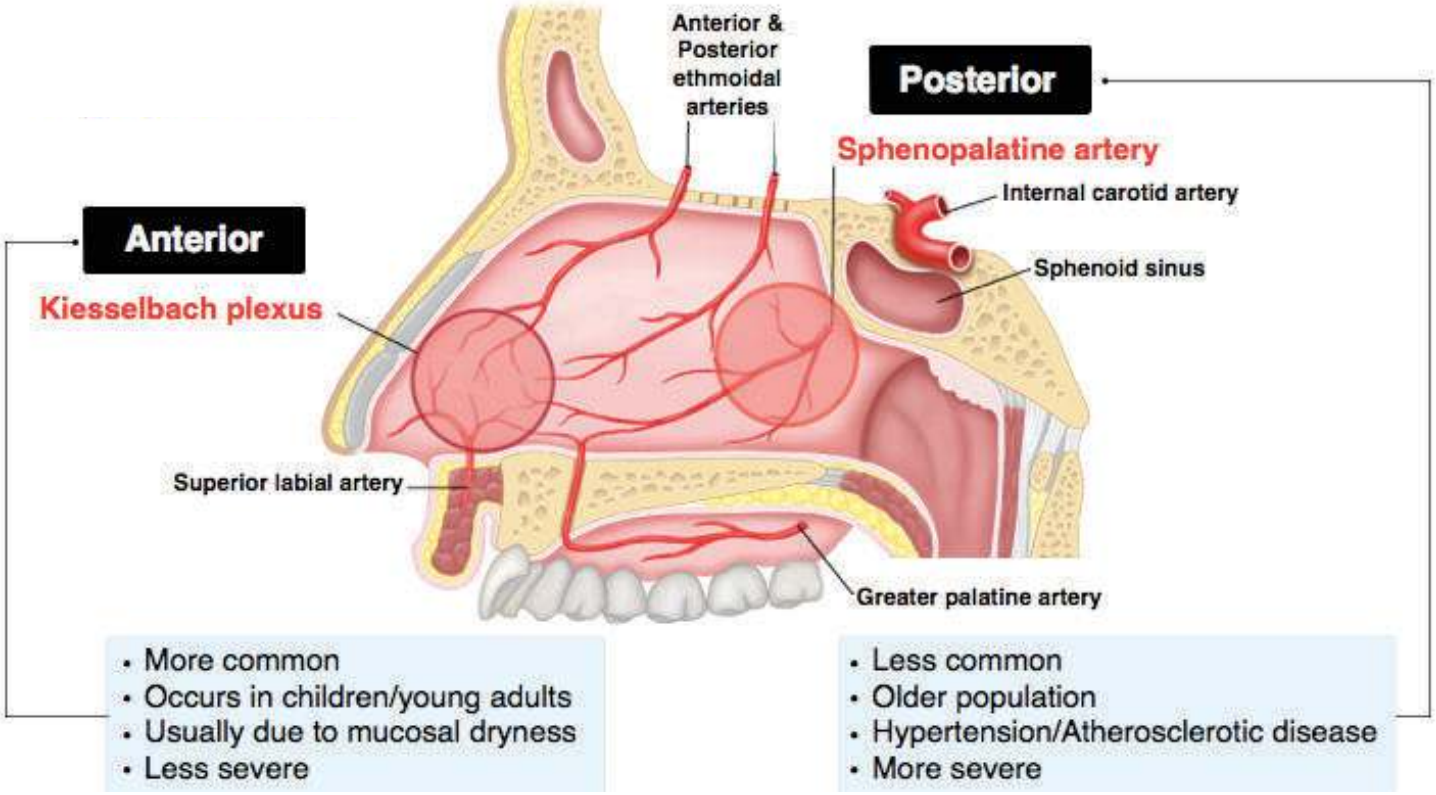

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# XIV. Epistaxis

### A. Epistaxis

Pathophysiology
• **Most common** site of bleeding is in the anterior nare at **Kiesselbach plexus**

Management
• Anterior bleed: direct pressure, **cautery** (if vessel visualized)
• Posterior bleed: **packing** (Foley, gauze pack, intranasal balloon device)

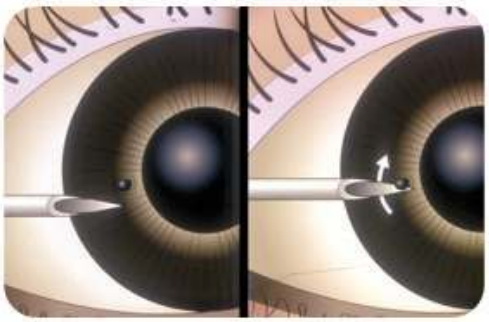



Notes


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# XV. Foreign Body

## A. Corneal Foreign Body Removal



1. Under slit-lamp guidance
2. 25- or 27-gauge needle to lift foreign body
3. Fine forceps, irrigation, wet cotton-tipped applicator to remove from surface

**Other considerations**
- Rust ring removal by properly trained clinician
- Treat as corneal abrasion and administer topical antibiotics
- Always be suspicious for open globe injury




Notes


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**B. Nasal Foreign Body**

Patient
• Child

Presentation
• **Unilateral**, mucopurulent, and **foul-smelling** nasal drainage




Notes


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# XVI. Glaucoma

### A. Acute Angle-Closure Glaucoma

Patient
• At a **movie theater** or entering a dark room

Presentation
• Halos and **acute**, **unilateral painful vision loss**
• **Vomiting**

Physical Exam
• **Cloudy cornea** and **fixed**, **mid-dilated pupil**

Diagnostic Studies
• **Increased** IOP (> 21 mm Hg)

Management
• **Topical BBs**, carbonic anhydrase inhibitors, steroids, miotics

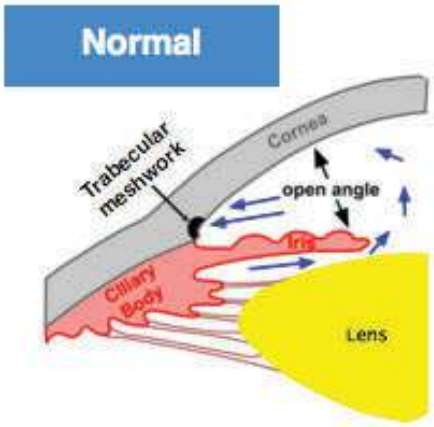
**Management**
*Empiric*
• Timolol (topical beta-blocker)
• Apraclonidine (topical alpha-agonist)
• Pilocarpine (topical miotic)
*Systemic*
• Acetazolamide (carbonic anhydrase inhibitor)
• Mannitol (osmotic diuretic)

**Clinical**
• Decreased vision
• Halos around lights
• Headache
• Severe eye pain
• Nausea and vomiting

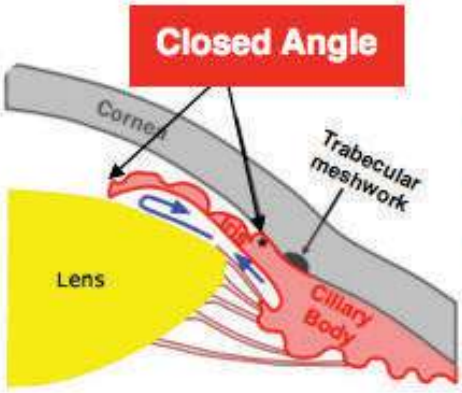
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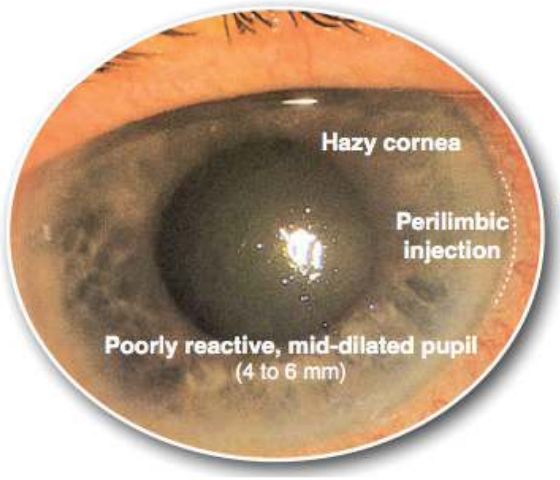
**Acute Angle-Closure Glaucoma cont.**




- Ciliary body produces aqueous humor
- Aqueous humor flows through pupil
- Aqueous humor reaches anterior chamber angle
- Aqueous humor exits the eye




- Sudden blockage of angle
- Intraocular pressure rises rapidly
- Acute eye changes occur




**Intraocular pressure (IOP)**

Normal 8 to 21 mm Hg

Elevated > 30 mm Hg

Notes


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# XVII. Hyphema

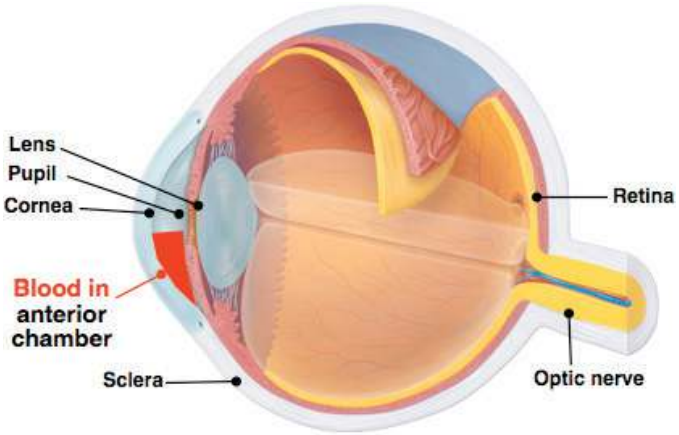
## A. Hyphema

Patient
• History of blunt or penetrating trauma

Presentation
• Blurry vision

Physical Exam
• Unequal pupils, injected conjunctiva and sclera, and **blood** in the **anterior chamber**

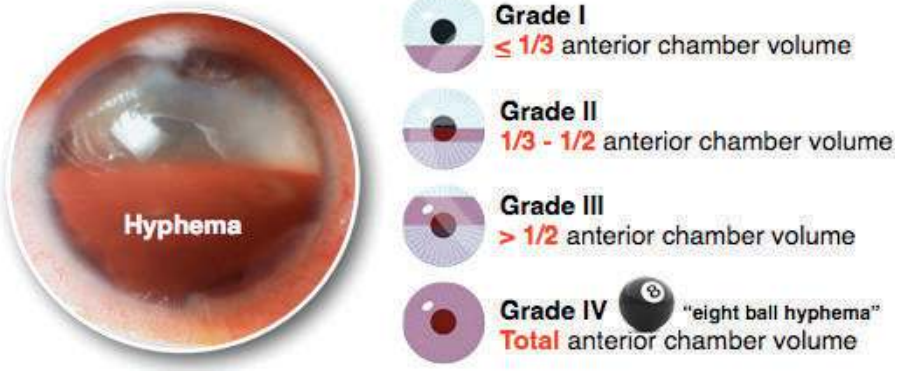
Management
• Eye protection and rest with the **head of bed at 30 degrees at all times**
• **Stat ophthalmology consult**




Notes

**Clinical**
• Decreased vision
• Photophobia
• Pain
• Absence of red reflex

**Management**
• Elevated head of bed 30-45 degrees
• Avoid antiplatelets and anticoagulants
• Avoid carbonic anhydrase inhibitors in sickle cell patients
• Ophthalmology consultation





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# XVIII. Labyrinthitis

**A. Labyrinthitis**

Patient
• History of **recent URI**

Presentation
• Sudden and **persistent** onset of **vertigo** and **hearing loss** lasting **several days** up to a **week**

**Clinical**
• Benign, self-limited
• Possibly due to viral or postviral inflammation of vestibular CN VIII
• Nystagmus (unilateral, horizontal, suppressed with fixation)
• Positive head thrust

**Abnormality of the inner ear vestibular system**

**Vestibular neuritis + unilateral hearing loss = Labyrinthitis**

1. Vertigo (severe)
2. Gait instability
3. Nausea, vomiting
4. Unilateral hearing loss



Notes


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# XIX. Macular Degeneration

### A. Macular Degeneration

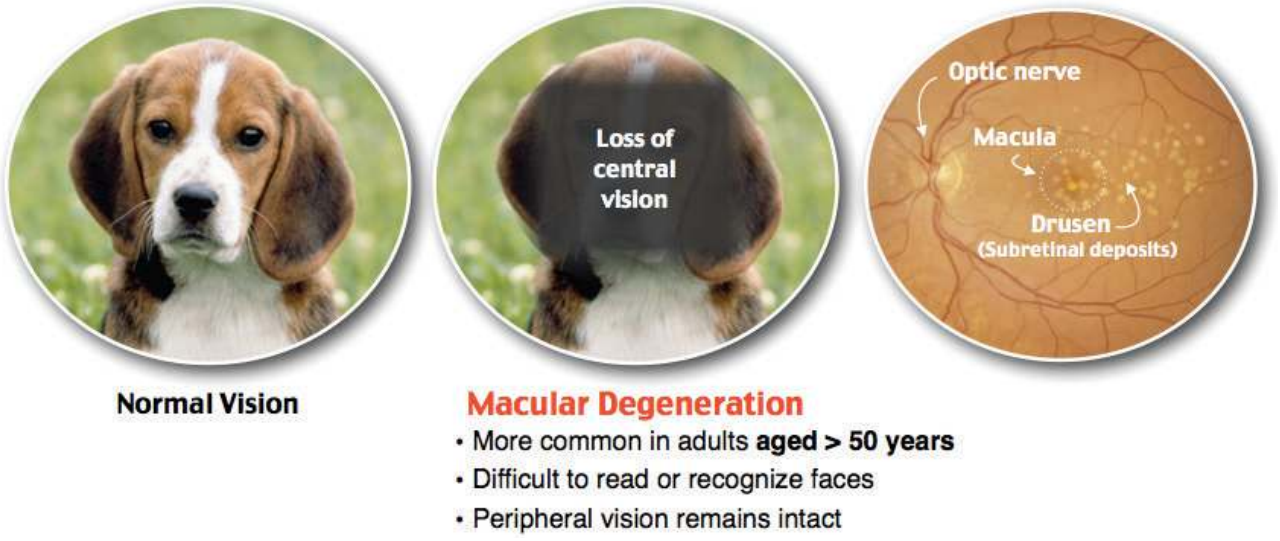
Patient
• Older

Presentation
• Bilateral, gradual, **central field** vision loss

Physical Exam
• Dry macular degeneration (**most common**): atrophic changes and yellow retinal deposits (**Drusen spots**)
• Wet macular degeneration: vascular changes

Diagnostic Studies
• **Amsler grid**

Comments
• **Most common** cause of **blindness** in the **elderly**




Notes


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**B. Age-Related Macular Degeneration**

| Dry (Nonexudative) | Wet (Exudative) |
|---|---|
| Drusen | Neovascularization |
| More common | Less common |
| Gradual loss of vision | Sudden loss of vision |
| Macular thinning | Bleeding, leakage of fluid |
| Not total blindness | Not total blindness but often more severe central visual loss |

Notes


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# XX. Mastoiditis

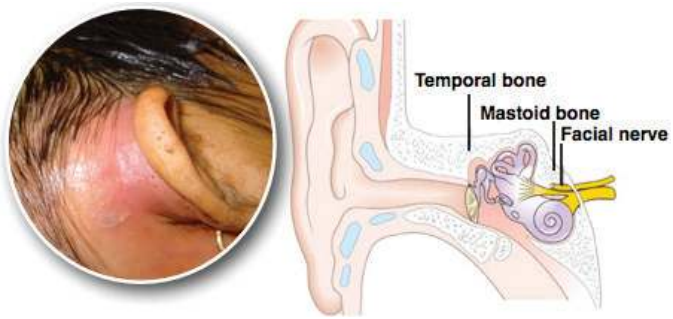
## A. Mastoiditis

Pathophysiology
• **Most commonly** a complication from preceding **acute otitis media**

Presentation
• Fever, otalgia, and erythema **posterior** to the ear

Physical Exam
• **Forward displacement** of the external ear

Management
• **IV antibiotics** and hospital admission




**Clinical**
• Lethargy and malaise
• Abnormal tympanic mem brane
• Postauricular erythema and tenderness
• Fever
• Narrowing of external auditory canal
• Ostalgia
• Otorrhea

**Management**
• Antibiotics
• Surgical drainage

**Complications**
• Meningitis
• Epidural or subdural abscess
• Facial nerve palsy
• Hearing loss
• Labyrinthitis
• Osteomyelitis
• Venous sinus thrombosis

Notes


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# XXI. Optic Neuritis

### A. Optic Neuritis

Pathophysiology
• **Most commonly** caused by **multiple sclerosis**

Presentation
• Acute **monocular** vision loss, pain **worse** **with eye movements**
• **Loss** of color vision (**red**), transient worsening of vision with increased body temperature (**Uhthoff phenomenon**)

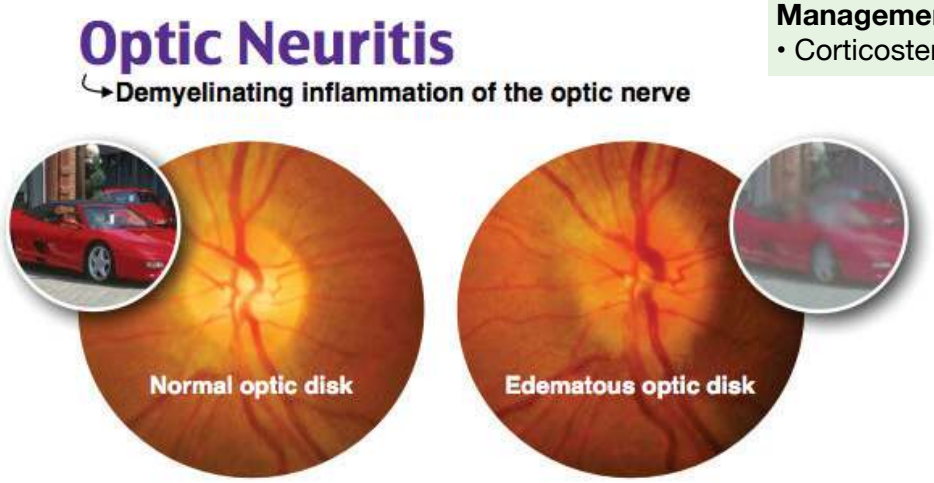
Diagnostic Studies
• Diagnosis is made **clinically**
• **MRI** will confirm demyelination

Management
• IV **methylprednisolone**

**Clinical**
• Sudden loss of monocular partial or complete vision
• Pain with movement of affected eye
• Afferent pupillary defect
• **Loss** of color (**red**) vision
• **Uhthoff's phenomenon**
• Transient worsening of vision with increased body temperature

**Etiologies**
• **Multiple sclerosis** (most common)
• Infection (Lyme, Herpes, Syphillis)
• Autoimmune (Lupus, Neurosarcoidosis)
• Methanol poisoning
• B12 deficiency
• Diabetes

**Management**
• Corticosteroids (intravenous)




Notes


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# XXII. Orbital Cellulitis

### A. Orbital Cellulitis

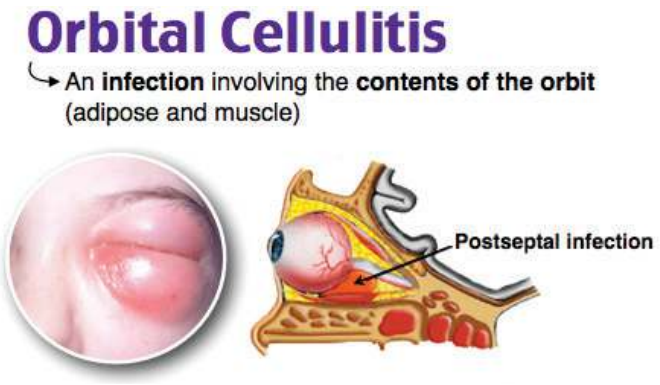
Presentation
• Swelling around the eye

Physical Exam
• **Painful** and **limited extraocular movement**, proptosis

Diagnostic Studies
• Diagnosis is made **clinically**; CT to confirm

Management
• **Urgent ophthalmology evaluation**
• Broad-spectrum antibiotics

Comments
• Compare to **pre-orbital cellulitis** which affects the lid and is less worrisome; extraocular movements will be intact




**Etiology**
• Children > Adults
• Bacterial rhinosinusitis (most common)
• Orbital trauma with fracture/foreign body
• Dacryocystitis
• Teeth, middle ear, face infection
• Infected mucocele

**Management**
• Broad-spectrum antibiotics (e.g., vancomycin + piperacillin-tazobactam)
• Surgery

Notes


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**Orbital Cellulitis cont.**

| Clinical feature | Preseptal cellulitis | Orbital cellulitis |
|---|---|---|
| Eyelid swelling (w/w/o erythema) | Yes | Yes |
| Eye pain/ tenderness | May be present | Yes, deep eye pain |
| Pain with eye movements | No | Yes |
| Proptosis | No | Usually, may be subtle |
| Ophthalmoplegia (+/- diplopia) | No | Yes |
| Vision impairment | No | May be present (Afferent pupillary defect) |
| Chemosis | Rarely present | May be present |
| Leukocytosis | May be present | May be present |

Notes


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# XXIII. Otitis Externa

### A. Otitis Externa

Pathophysiology
• **Most commonly** caused by ***Pseudomonas aeruginosa***

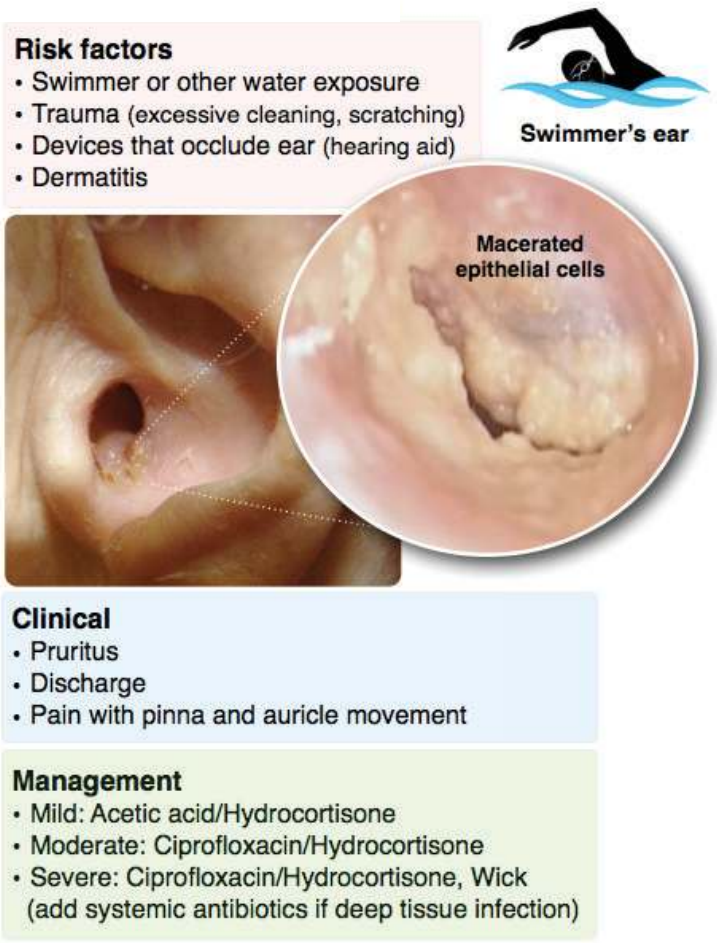
Patient
• History of **swimming** or exposure to moisture

Presentation
• Malodorous discharge and **pruritus**

Physical Exam
• Tenderness with manipulation of **tragus** or **pinna**

Management
• Topical **antimicrobials** with or without steroids
• If no resolution, consider treating for presumed **fungal** etiology

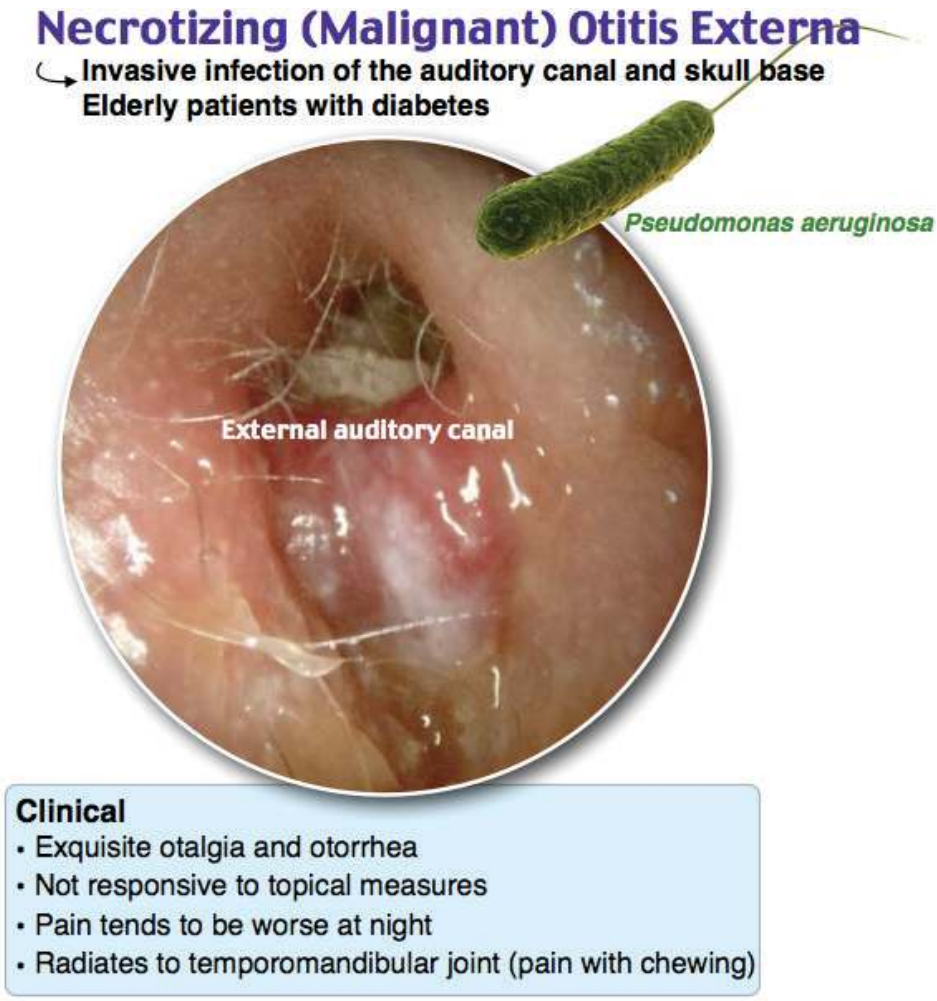
Comments
• **Necrotizing (malignant) otitis externa** is a complication seen in diabetic or immunocompromised patients




Notes


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**B. Necrotizing (Malignant) Otitis Externa**




**Clinical**
- Exquisite otalgia and otorrhea
- Not responsive to topical measures
- Pain tends to be worse at night
- Radiates to temporomandibular joint (pain with chewing)

Notes


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# XXIV. Papilledema

## A. Papilledema

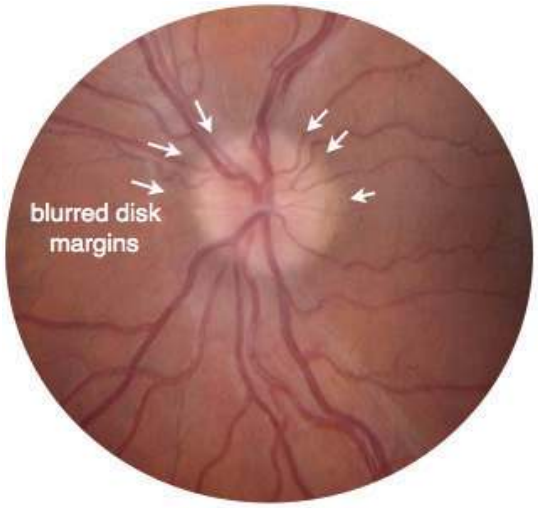
Pathophysiology
• **Most commonly** secondary to increased intracranial pressure

Presentation
• Headaches, blurred vision, and vomiting

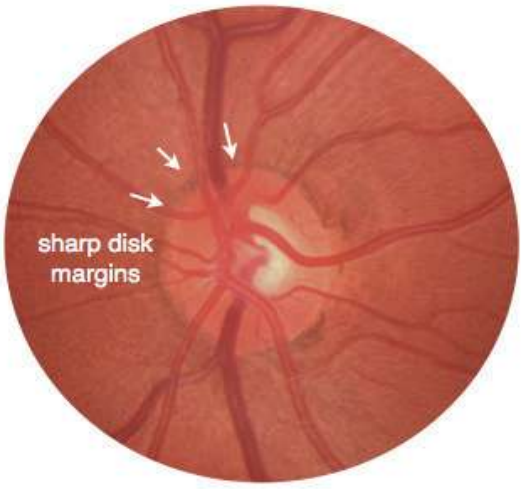
Physical Exam
• **Optic disc swelling**

Management
• Treat underlying disorder

Comments
• Focus on finding **intracranial** pathology



Papilledema



Normal Optic Disk

Notes


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# XXV. Peritonsillar Abscess

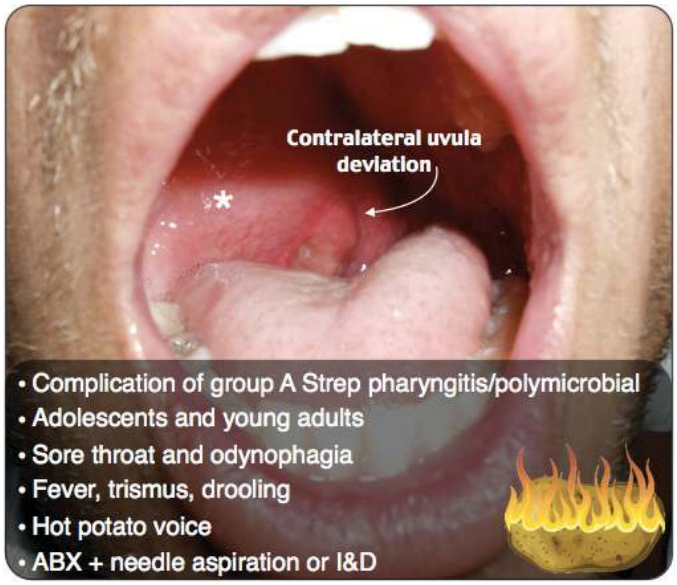
**A. Peritonsillar Abscess**

Patient
• Adolescent with a history of pharyngitis

Presentation
• Severe sore throat, drooling, and fever

Physical Exam
• Trismus, **"hot potato"** voice, and **uvular displacement** away from abscess

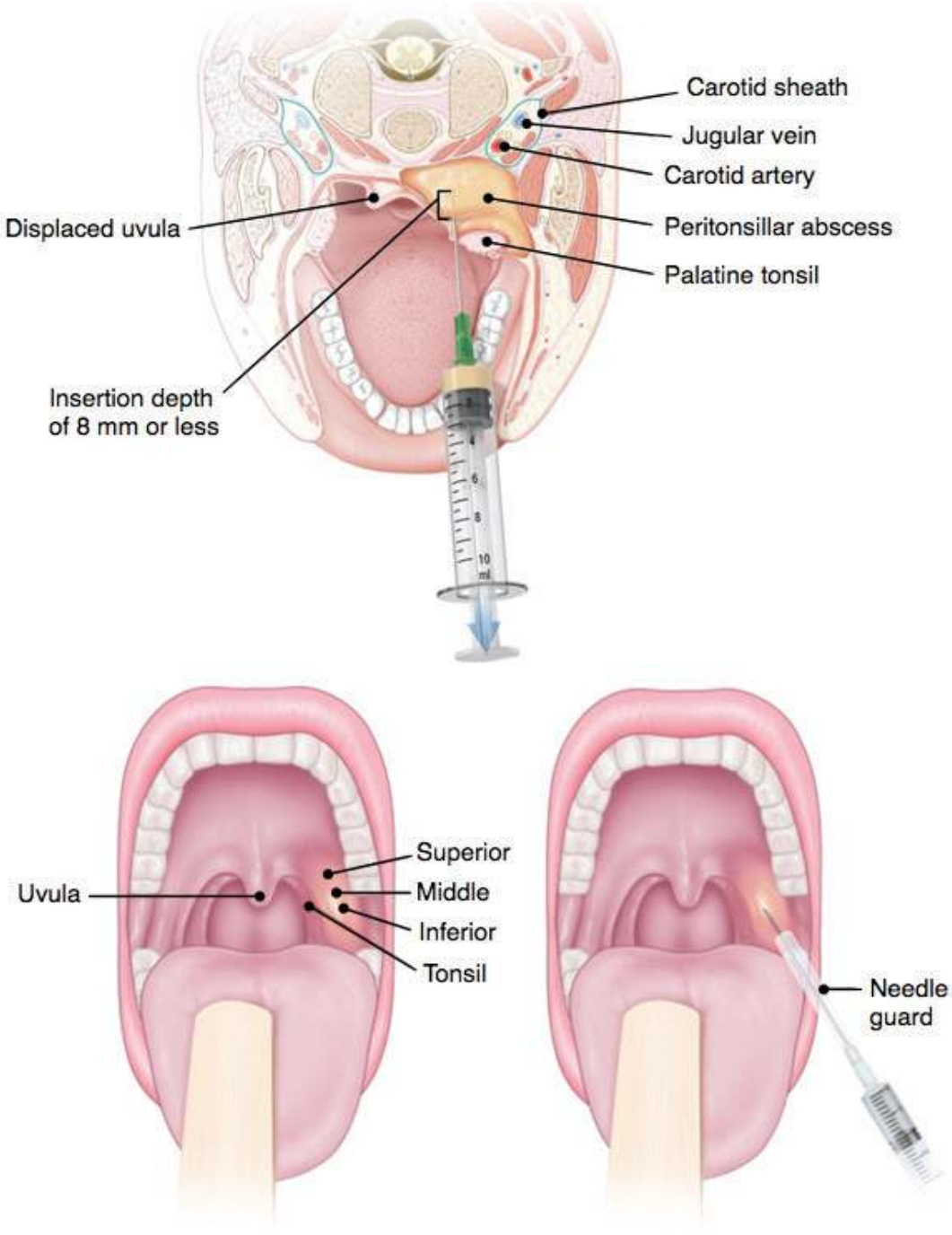
Management
• I&D or needle aspiration followed by antibiotics




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**B. Peritonsillar Abscess Needle Aspiration**




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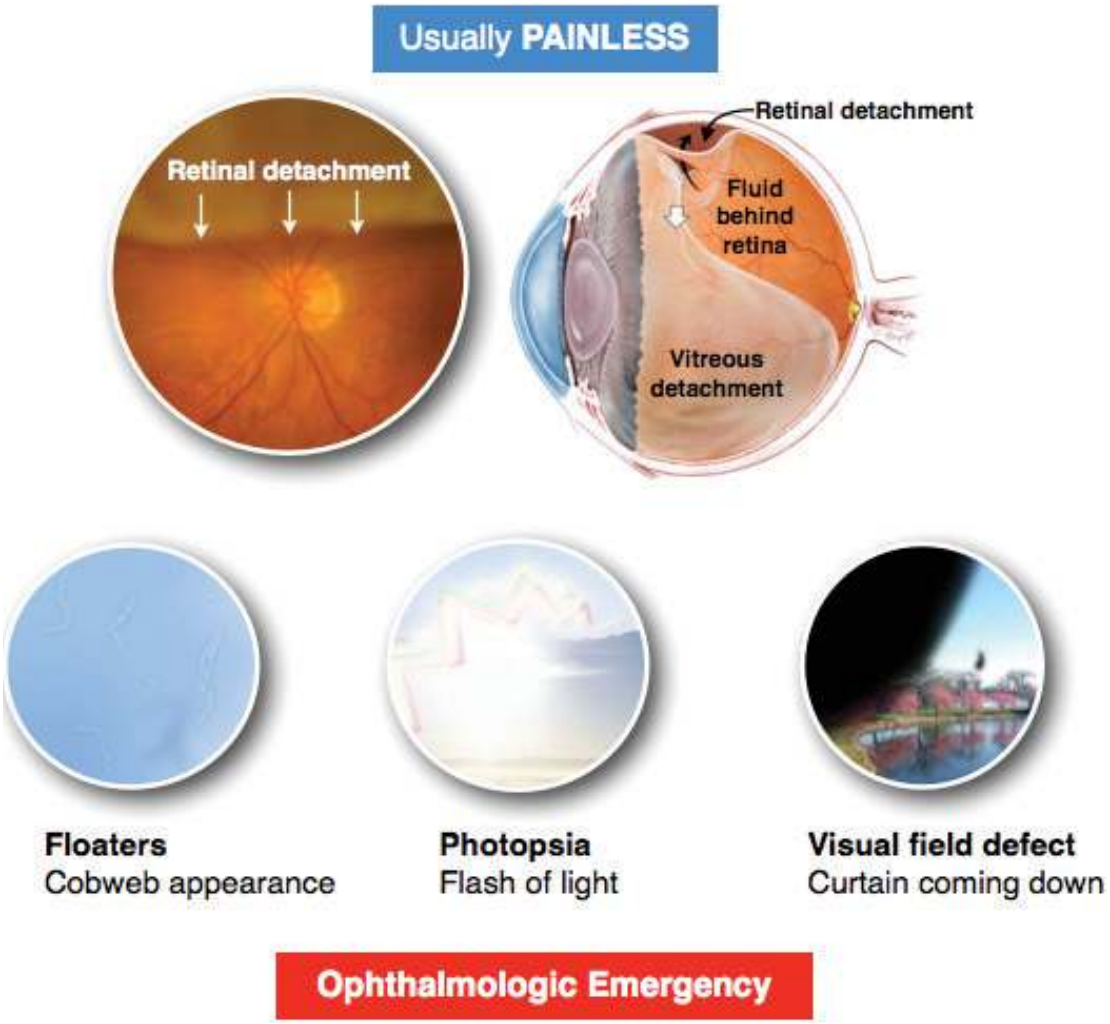
# XXVI. Retinal Detachment

### A. Retinal Detachment

Presentation
- **Painless** loss of vision, floaters, flashing lights, **curtain-lowering** sensation

Physical Exam
- Retina appears **hazy gray** with white folds

Management
- **Emergent** ophthalmology consult




Notes


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# XXVII. Retinal Vein Occlusion

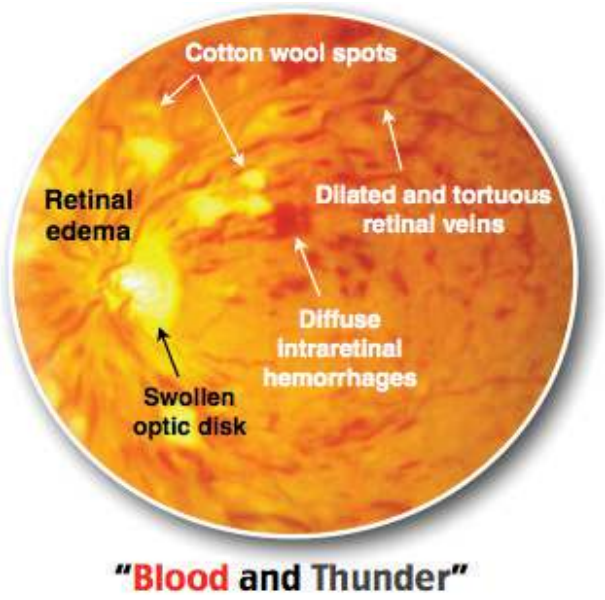
**A. Retinal Vein Occlusion**

Patient
• History of HTN or DM

Presentation
• Varying degree of **sudden**, **painless**, **monocular** vision loss

Physical Exam
• Fundoscopy with **"blood and thunder"** appearance
• **Tortuous** and **dilated** retinal veins

Management
• Ophthalmology consult




Notes


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**Retinal Vein Occlusion cont.**

**1 Non-ischemic**
- Mild
- Absence of afferent pupillary defect
- Good retinal perfusion
- Can progress to ischemic type

**2 Ischemic**
- Venous stasis
- Increased vascular back-pressure
- Results in decreased arterial flow to retina
- Visual acuity < 20/100
- Afferent pupillary defect

**Clinical**
- **Sudden, painless, monocular loss of vision**
- "Blood" and thunder" fundoscopic exam

**Management**
- Ophthalmology consult
- Anti-vascular endothelial growth factor
- Dexamethasone implant
- Triamcinolone (intravitreal)



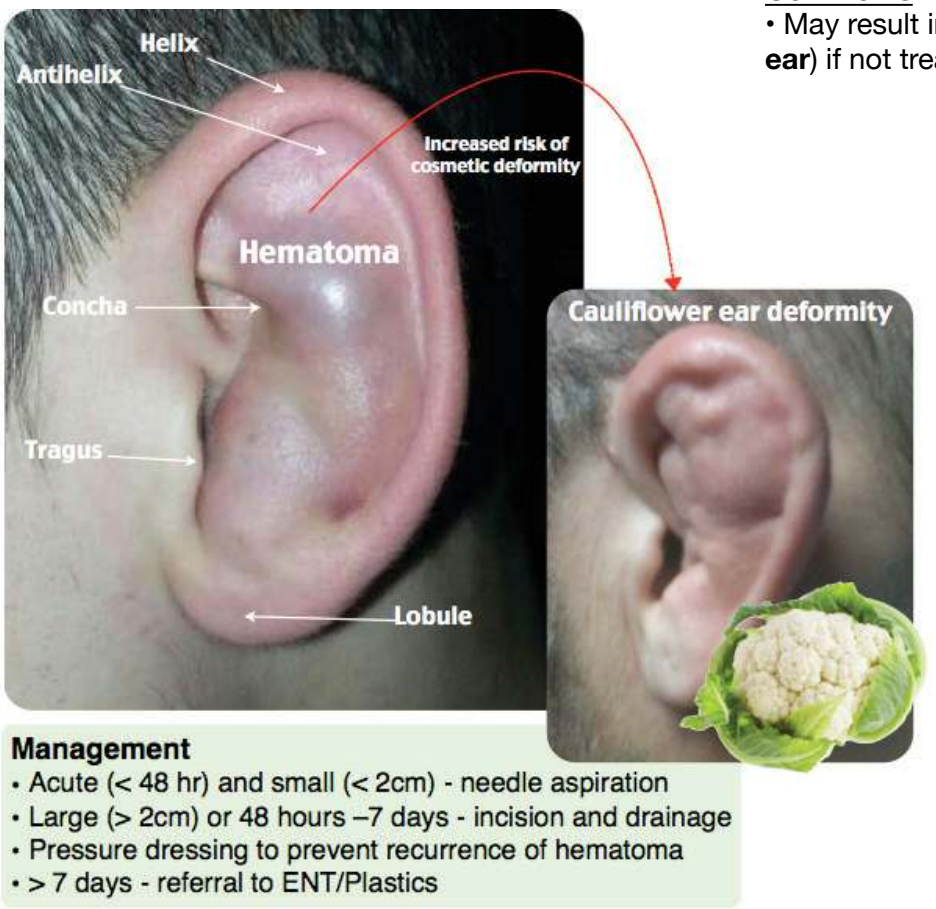

Notes


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# XXVIII. Trauma/Hematoma (External Ear)

### A. Auricular Hematoma

Pathophysiology
• **Blunt trauma** to ear with **shearing forces** to the anterior auricle leads to separation of the anterior auricle perichondrium from the underlying cartilage




**Management**
• Acute (< 48 hr) and small (< 2cm) - needle aspiration
• Large (> 2cm) or 48 hours –7 days - incision and drainage
• Pressure dressing to prevent recurrence of hematoma
• > 7 days - referral to ENT/Plastics

Management
• **Evacuate blood**, pressure dressing, antibiotics (cephalexin)

Comments
• May result in thickening of cartilage (**cauliflower ear**) if not treated promptly

Notes

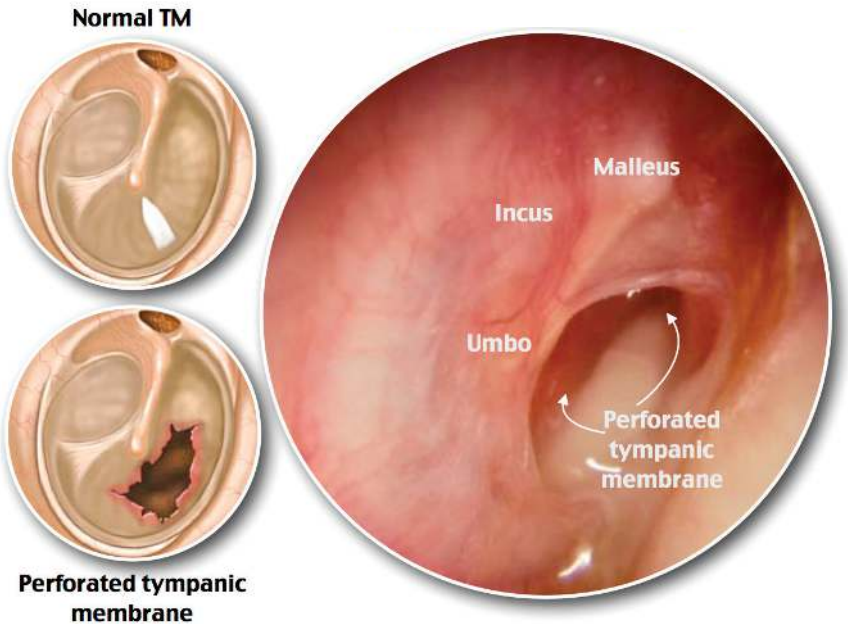

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# XXIX. Tympanic Membrane Perforation

### A. Tympanic Membrane Perforation

Presentation
• Pain, otorrhea, and hearing reduction or loss

Management
• Most heal spontaneously, keep clean and dry, treat with antibiotics if contaminated; the only class of antibiotics that are non-ototoxic are **ofloxacin drops**
• Surgery if persists past two months




Notes


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# XXX. Vertigo

**A. Vertigo**

• See Neurology chapter - Vertigo

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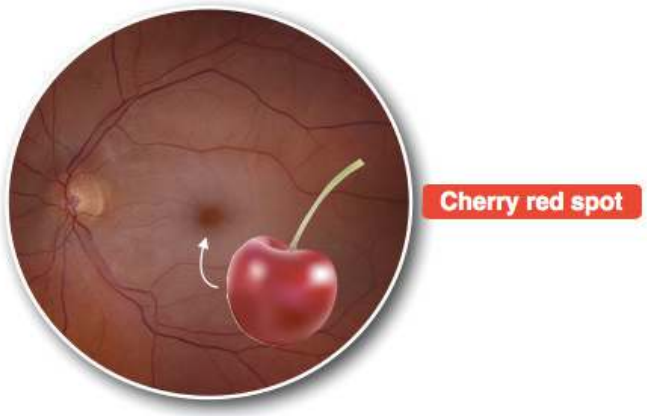
# XXXI. Vision Loss

### A. Central Retinal Artery Occlusion

Presentation
• **Sudden**, **painless**, **monocular** vision loss

Physical Exam
• Fundoscopy with **"boxcar"** appearance or **"cherry red spot"**

Management
• Globe massage, **lower** IOP (e.g., mannitol IV), ophthalmology consult




**Clinical**
• **Sudden**, **painless**, **complete loss of vision**
• Pale retina
• Cherry red spot on fovea

**Management**
• Immediate ophthalmology consult
• Orbital massage to attempt clot dislodgement
• Reduction of IOP (acetazolamide, mannitol (IV))
• Vasodilators (pentoxifylline, nitroglycerin, isosorbide)




Notes


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**B. Typical Features of Common Causes of Transient Visual Loss**

| | |
|---|---|
| **Etiology** | Monocular ischemia, carotid disease, other embolic source |
| | Giant Cell arteritis |
| | Papilledema |
| | Idiopathic retinal vasospasm |
| | Migraine |
| | Vertebrobasilar ischemia |
| | Seizure: Ictal |
| | Postictal |
| **Typical duration** | 1 to 10 minutes |
| | Variable |
| | Seconds |
| | 5 to 60 minutes |
| | 10 to 30 minutes |
| | 1 to 10 minutes |
| | 3 to 5 minutes |
| | 20 minutes, longer |
| **Pattern of visual loss** | Monocular, rapid onset, altitudinal onset |
| | Usually monocular |
| | Monocular graying or blurring |
| | Monocular positive or negative symptoms |
| | Usually binocular, positive symptoms with spread |
| | Homonymous hemianopia |
| | Binocular, lateralized, positive phenomenon common |
| | Binocular visual field loss |


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**Typical Features of Common Causes of Transient Visual Loss cont.**

| | |
|---|---|
| **Associated symptoms and signs** | Hollenhorst plaque, hemispheric symptoms |
| | Headache, neck pain, jaw claudication |
| | Headache, diplopia |
| | Transient retinal arterial narrowing, headache |
| | Usually followed by migraine headache |
| | Isolated or accompanied by other brainstem deficits |
| | Altered consciousness, motor symptoms |
| | Preceding ictus |
| **Mechanism** | Retinal embolism (usually) |
| | Ischemia of optic nerve |
| | Elevated intracranial pressure |
| | Vasospasm |
| | Spreading cortical depression, possible retinal vasospasm |
| | Embolic |
| | Epileptic discharge |
| | Cortical inhibition |

Notes


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CHAPTER 7

# Urology/Renal

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CHAPTER 7

# Urology/Renal


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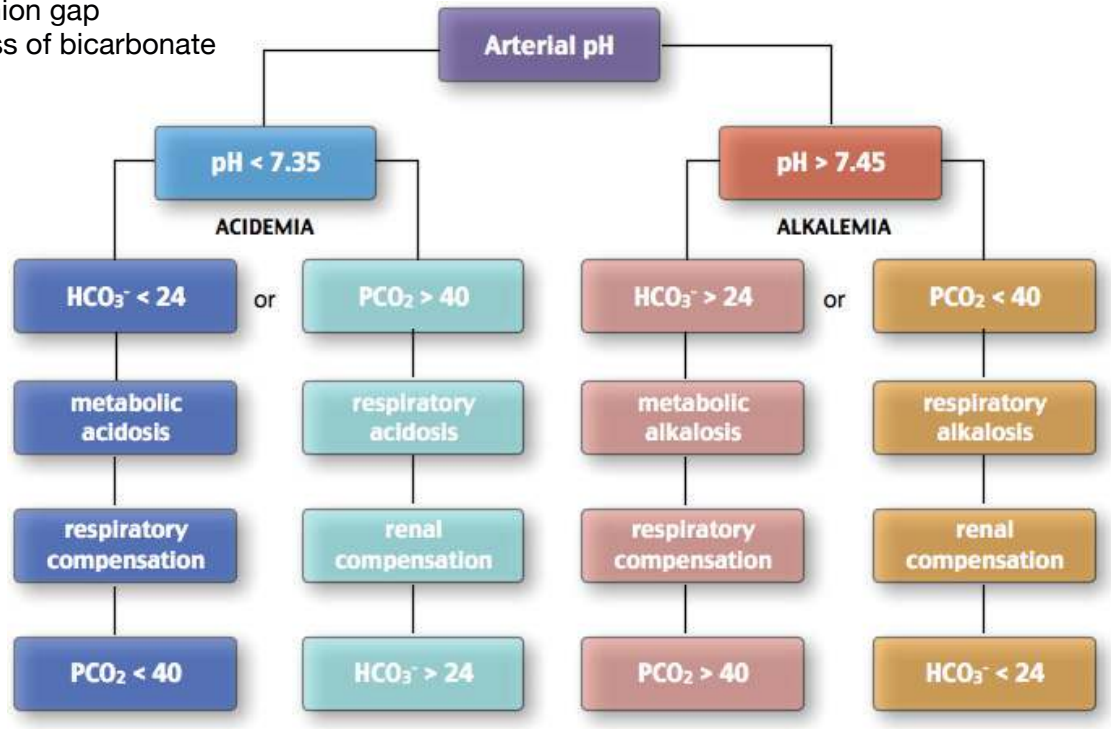
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# I. Acid/Base Disorders

**A. Acute Respiratory and Metabolic Acidosis and Alkalosis**
(Normal ABG values pH:7.35–7.45; PaCO2: 35–45; HCO3: 22–26)

Pathophysiology
- **Respiratory Acidosis**
  - **Lungs fail to excrete CO2** (Breathing too slow (holding onto CO2)
- **Respiratory Alkalosis**
  - **Excessive elimination of CO2** (Breathing too fast (blowing of CO2)
- **Metabolic Acidosis**
- How to calculate ion gap: **Na – (Cl + HCO3-) = 8-16 mEq/L**
  - Increased ion gap metabolic acidosis
    - **A CAT MUDPILE**
  - Low anion gap
    - Loss of bicarbonate

Comments
- **Winter Formula** is used to evaluate respiratory compensation when metabolic acidosis is present in a patient. PCO2 = (HCO3 x 1.5) + 8 ± 2
- **Delta ratio** is used to assess elevated anion gap metabolic acidosis and to evaluate whether a mixed acid-base disorder is present




Notes


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**B. Respiratory Acidosis** pH: **< 7.35; PaCO2: > 45**

**Acute (Acute respiratory failure)**
- $PaCO_2$ is elevated (> 45 mm Hg)
- Accompanying acidemia (pH < 7.35)

**Chronic (COPD, Obesity hypoventilation syndrome)**
- $PaCO_2$ is elevated (> 45 mm Hg)
- Normal or near-normal pH (pH ≈ 7.4) [Renal compensation]
- Elevated serum bicarbonate (> 30 mEq/L)

**Physiologic compensation**

1. **Cellular buffering** occurs over minutes to hours (1 mEq/L for each 10 mm Hg increase in $PaCO_2$)
2. **Renal compensation** occurs over 3 - 5 days [Excretion of carbonic acid, bicarbonate reabsorption] (3 - 5 mEq/L for each 10 mm Hg increase in $PaCO_2$)

**Change in pH**

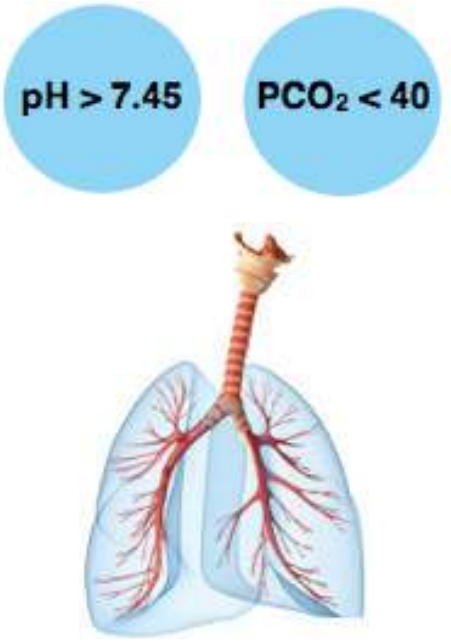
Acute: $\Delta pH\ 0.008 \times (40 - PaCO_2)$

Chronic: $\Delta pH\ 0.003 \times (40 - PaCO_2)$

Notes


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## C. Respiratory Alkalosis pH: > 7.45; PaCO2: < 35




**Common Causes**
- Hyperventilation syndrome
- Anxiety, pain
- Fever, psychosis
- Pulmonary embolism
- Heart failure
- Mechanical ventilation
- Stroke
- Pregnancy
- Hyperthyroidism
- Salicylate toxicity

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## D. Metabolic Acidosis pH: < 7.35; HCO3: < 22

pH < 7.35
Serum bicarbonate < 22 mEq/L

**Physiologic compensation**

**Blood buffers**
Nonvolatile buffers absorb excess $H^+$

**Respiratory compensation** occurs over minutes to hours
Increased respiratory rate (eliminate $CO_2$)

**Renal compensation** occurs over 3–5 days
Secrete $H^+$ and reabsorb and generate $HCO_3^-$

1 **Determination of appropriate respiratory compensation**

**Winter's formula**

$pCO_2 = 1.5\ (HCO_3^-) + 8 +/- 2$
If measured $pCO_2$ is > Winter's formula $pCO_2$ then concomitant respiratory acidosis
If measured $pCO_2$ is < Winter's formula $pCO_2$ then concomitant respiratory alkalosis

2 **Anion gap = Na - [$Cl^-$ + $HCO_3^-$]**
> 11 is elevated

3 **Delta ratio** = $\frac{\Delta \text{ Anion gap}}{\Delta\ HCO_3^-}$

Δ Anion gap = Measured anion gap - Normal anion gap
Δ $HCO_3^-$ = Normal [$HCO_3^-$] - Measured [$HCO_3^-$]

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### E. Major Causes for Metabolic Acidosis According to Mechanism and Anion Gap

| Mechanism of acidosis | Increased anion gap | Normal anion gap |
|---|---|---|
| Increased acid production | Lactic acidosis | - |
| | Ketoacidosis | - |
| | Diabetes mellitus | - |
| | Starvation | - |
| | Alcohol associated | - |
| | Ingestions | |
| | Methanol | - |
| | Ethylene glycol | - |
| | Aspirin | - |
| | Toluene (if early or if kidney function is impaired) | Toluene ingestion (if late and if renal function is preserved; due to excretion of sodium and potassium hippurate in the urine) |
| | Diethylene glycol | - |
| | Propylene glycol | - |
| | D-lactic acidosis | A component of non-AG metabolic acidosis may coexist due to urinary excretion of D-lactate as Na and K salts (which represents potential HCO3) |
| | Pyroglutamic acid (5-oxoproline) | - |
| Loss of bicarbonate or bicarbonate precursors | - | Diarrhea or other intestinal losses (e.g., tube drainage) |
| | | Type 2 (proximal) renal tubular acidosis |
| | | Posttreatment of ketoacidosis |
| | | Carbonic anhydrase inhibitors |
| | | Ureteral diversion (e.g., ileal loop) |
| Decreased renal acid excretion | Chronic kidney disease | Chronic kidney disease and tubular dysfunction (but relatively preserved glomerular filtration rate) |
| | | Type 1 (distal) renal tubular acidosis |
| | | Type 4 renal tubular acidosis (hypoaldosteronism) |

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**F. Causes of High-Anion Gap metabolic Acidosis**

**Anion gap = Na - [$Cl^-$ + $HCO_3^-$]**

A CAT MUDPILE

**A**spirin

**C**arbon monoxide, **C**yanide, **C**affeine

**A**cetaminophen

**T**heophylline

**M**ethanol, **M**etformin

**U**remia

**D**iabetic ketoacidosis (Alcoholic ketoacidosis)

**P**ropylene glycol

**I**soniazid, **I**buprofen, **I**ron

**L**actic acidosis

**E**thylene glycol



Notes


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**G. Metabolic Alkalosis** pH: **> 7.45; HCO3: > 26**

| Major Causes of Metabolic Alkalosis |
|---|
| **Gastrointestinal hydrogen loss** |
| Vomiting or nasogastric suction |
| Congenital chloride diarrhea |
| **Renal hydrogen loss** |
| Primary mineralcorticoid excess |
| Mineralcorticoid excess-like states<br>• Licorice ingestion<br>• Liddle syndrome<br>• Apparent mineralcorticoid excess |
| Loop or thiazide diuretics |
| Bartter or Gitelman syndrome |
| Posthypercapnic alkalosis |
| Hypercalcemia and the milk-alkali syndrome |
| **Intracellular shift of hydrogen** |
| Severe hypokalemia<br>Villous adenoma<br>Laxative abuse |
| **Alkali administration with reduced renal function** |
| **Contraction alkalosis** |

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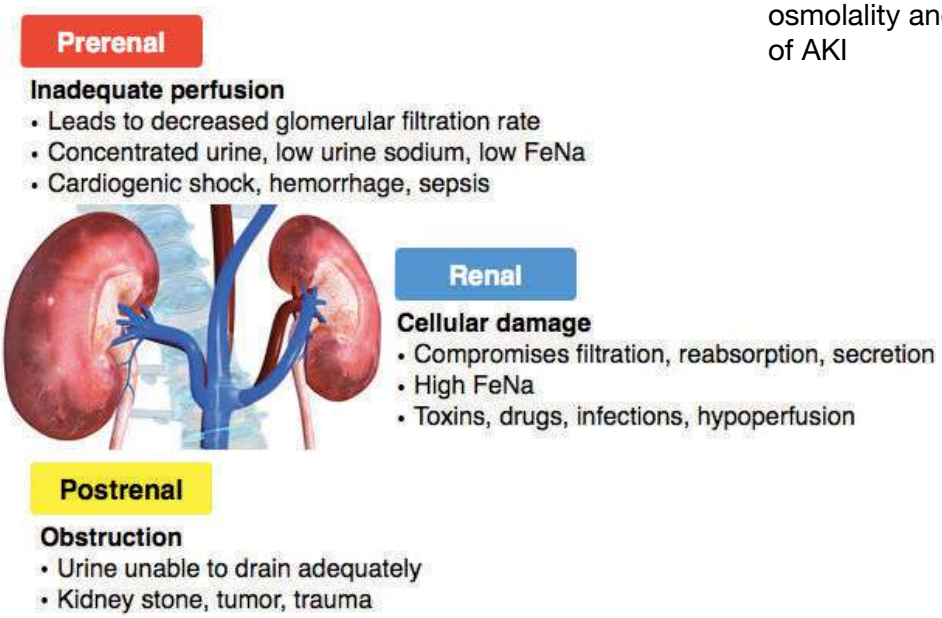
# II. Acute Renal Failure

### A. Acute Kidney Injury

Pathophysiology
- Acute reduction in glomerular filtration rate (GFR)
- Can be prerenal, intrarenal, or postrenal
- **Prerenal** causes
  - Decreased renal perfusion (Ex: hemorrhage, congestive heart failure, diuretic use)
- **Intrarenal** causes
  - Acute tubular necrosis
  - Ischemia and toxic causes
  - Interstitial nephritis
  - Glomerulonephritis
  - Vasculitis
  - Hemolytic uremic syndrome
  - Cholesterol emboli
- **Postrenal** causes
  - Urinary flow obstruction (e.g., benign prostatic hyperplasia and nephrolithiasis)
  - Postoperative secondary to bladder manipulation and anesthesia

Patient
- History of HTN, CKD, dehydration and volume depletion, diabetes, chronic liver or lung disease

Diagnostic Studies
- Renal ultrasound
- **Increase** in serum creatinine and blood urea nitrogen (BUN) to creatinine ratio
- Fractional excretion of sodium (FENa), urine osmolality and Na+ are helpful in determining type of AKI




Notes


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# III. Cystitis

### A. Cystitis (Urinary Tract Infection)

Pathophysiology
• **Most commonly** caused by ***Escherichia coli***

Presentation
• Low-grade fever, increased urinary frequency, dysuria, and suprapubic or abdominal pain

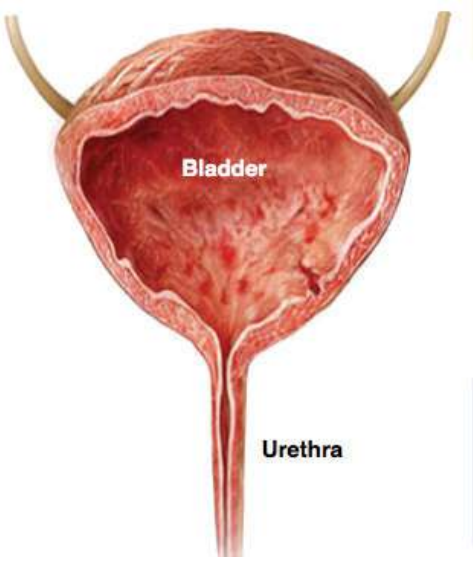
**Diagnosis**
• Urine culture > 100,000 colonies (from reliable source)

**Etiology**
• *E. coli*
• *Klebsiella*
• *Proteus*
• *Enterococcus*
• *Staph saprophyticus* (Female adolescents)
• Group B Strep (Neonates)
• *Pseudomonas*
• *Adenovirus*

**Clinical**
• Suprapubic pressure and flank pain (referred)
• Vomiting
• Dysuria, urgency, frequency
• Enuresis

Diagnostic Studies
• **Leukocyte esterase** and **nitrites** present on UA

Management
• Acute uncomplicated cystitis: TMP-SMX, nitrofurantoin, or fluoroquinolone for **three to five** days
• Acute uncomplicated cystitis with **comorbid conditions**: same antibiotics for **seven days**




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# IV. Epididymitis

### A. Epididymitis

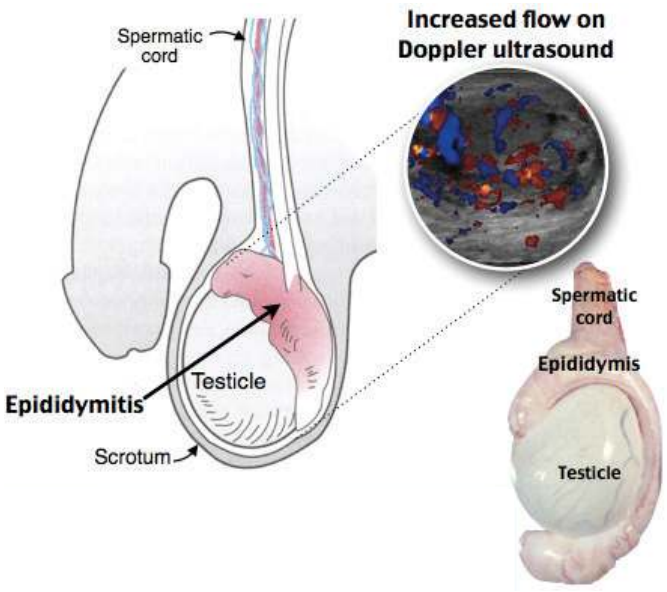
Pathophysiology
- < 35 years old: **most commonly *C. trachomatis***
- > 35 years old: **most commonly *E. coli***

Presentation
- **Gradual-onset** unilateral scrotal pain

Physical Exam
- **Relief** with testicular elevation (**Prehn sign**)

Diagnostic Studies
- **Increased color flow** on Doppler

Management
- < 35 years old: ceftriaxone/doxycycline
- > 35 years old: ciprofloxacin




**Clinical**
- Increasing **dull**, **unilateral** scrotal pain
- **Erythematous**, **painful**, swollen scrotum
- **Relief** with testicular **elevation** (Prehn sign)

Notes

**Sexually transmitted (< 35 years of age)**
- *Chlamydia trachomatis*
- *Neisseria gonorrhoeae*
- Rx: **Ceftriaxone/doxycycline**

**Nonsexually transmitted (> 35 years of age)**
- *E. coli* and other coliforms
- *Pseudomonas*
- Rx: **Trimethoprim and sulfamethoxazole or levo/ciprofloxacin**


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# V. Fluid and Electrolyte Disorders

## A. Dehydration

| Symptoms Associated with Dehydration (Pediatric) | | | |
|---|---|---|---|
| **Symptom** | **Minimal or no dehydration (< 3% loss of body weight)** | **Mild to moderate dehydration (3% to 9% loss of body weight)** | **Severe dehydration (> 10% loss of body weight)** |
| **Mental status** | Well, alert | Normal, fatigued or restless, irritable | Apathetic, lethargic, unconscious |
| **Thirst** | Drinks normally, might refuse liquids | Thirsty; eager to drink | Drinks poorly; unable to drink |
| **Heart rate** | Normal | Normal to increased | Tachycardia, with bradycardia in most severe cases |
| **Quality of pulses** | Normal | Normal to decreased | Weak, thready, or impalpable |
| **Breathing** | Normal | Normal; fast | Deep |
| **Eyes** | Normal | Slightly sunken | Deeply sunken |
| **Tears** | Present | Decreased | Absent |
| **Mouth and tongue** | Moist | Dry | Parched |
| **Skin fold** | Instant recoil | Recoil in < 2 sec | Recoil in > 2 sec |
| **Capillary refill** | Normal | Prolonged | Prolonged; minimal |
| **Extremities** | Warm | Cool | Cold; mottled; cyanotic |
| **Urine output** | Normal to decreased | Decreased | Minimal |

Notes


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**B. Hyperkalemia**

Patient
• History of **renal failure**, **DKA**, **rhabdomyolysis**, **tumor lysis**
• Missed dialysis session

Presentation
• Lethargy, weakness, paralysis

Physical Exam
• Bradycardia, hypotension, **cardiac dysrhythmia**

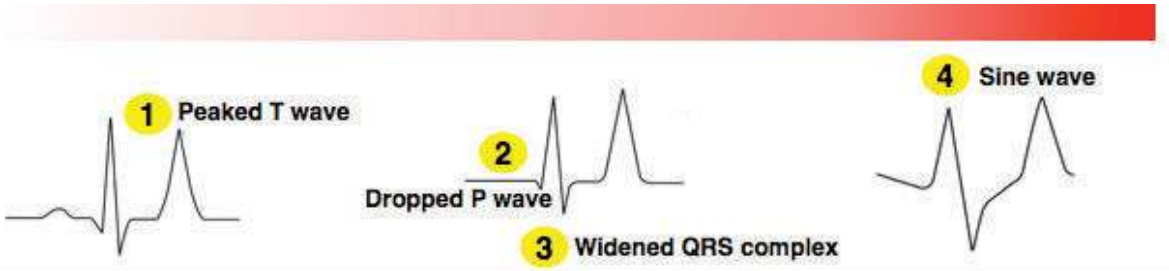
Diagnostic Studies
• ECG will show **peaked T waves**, **prolonged PR**, **wide QRS** when serum potassium is > **5.5** mmol/L

Management
• **Calcium gluconate** first to stabilize cardiac membrane followed by medications that will **redistribute** or **eliminate** potassium stores (insulin, albuterol, kayexalate, bicarbonate, hemodialysis)

Notes


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### C. ECG Changes Due to Hyperkalemia




| Hyperkalemia Treatment | | | |
|---|---|---|---|
| **Intervention** | **Onset** | **Duration** | **Mechanism** |
| **Membrane stabilization** | | | |
| Calcium gluconate or chloride | Immediate | 30–60 min | Membrane stabilization |
| **Redistribution** | | | |
| Insulin | 20–30 min | 4–6 hr | Shifts potassium into cell |
| Albuterol | 15–30 min | 2–4 hr | Upregulates cAMP & shifts potassium into cells |
| Sodium bicarbonate | 5–10 min | 1–2 hr | Shifts potassium into cell |
| **Elimination** | | | |
| Furosemide | 15–30 min | 2–3 hr | Increases renal potassium excretion |
| Sodium polystyrene | 1–4 hr | 4–6 hr | Increases GI potassium excretion |
| Hemodialysis | Immediate | 2–6 hr | Removes potassium |

Notes


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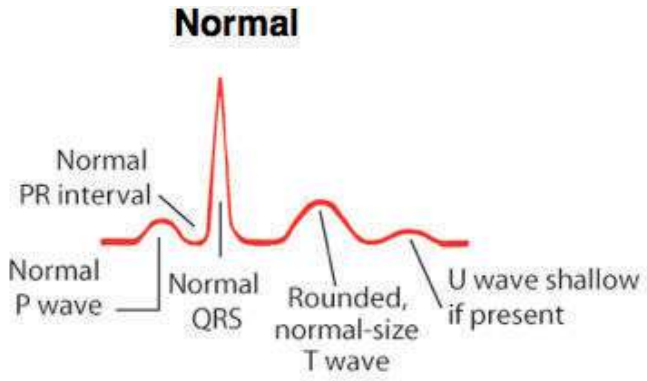
**D. Hypokalemia**

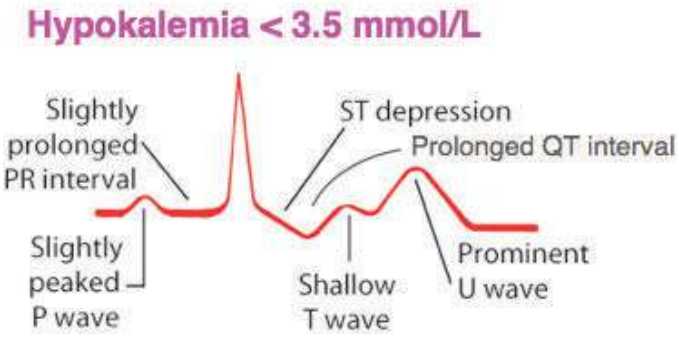
Patient
• **Diuretic use**, **diarrhea**, **vomiting**

Presentation
• **Weakness**, **hyporeflexia**, **cramping**, paresthesias

Diagnostic Studies
• ECG will show **U waves**, **T wave flattening**, ST depression, **QT prolongation** when serum potassium **< 3.5** mmol/L

Management
• Potassium and **magnesium** replacement




Notes


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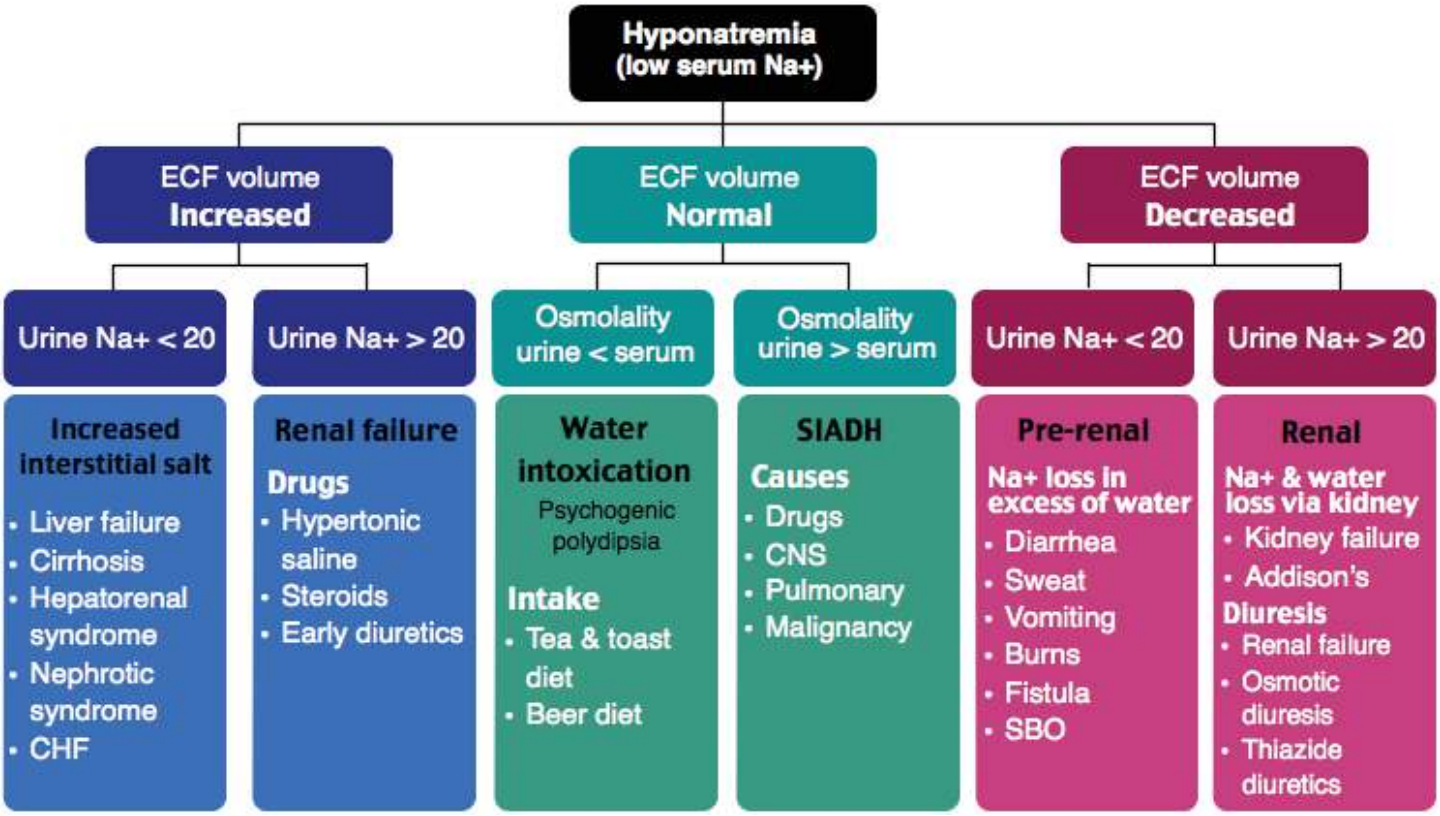
### E. Hyponatremia

Presentation
• Patient will be complaining of nausea, vomiting, paresthesias, AMS

Diagnostic Studies
• Labs will show serum sodium **< 135**

Management
• Treatment is **hypertonic saline** for **symptomatic** patients

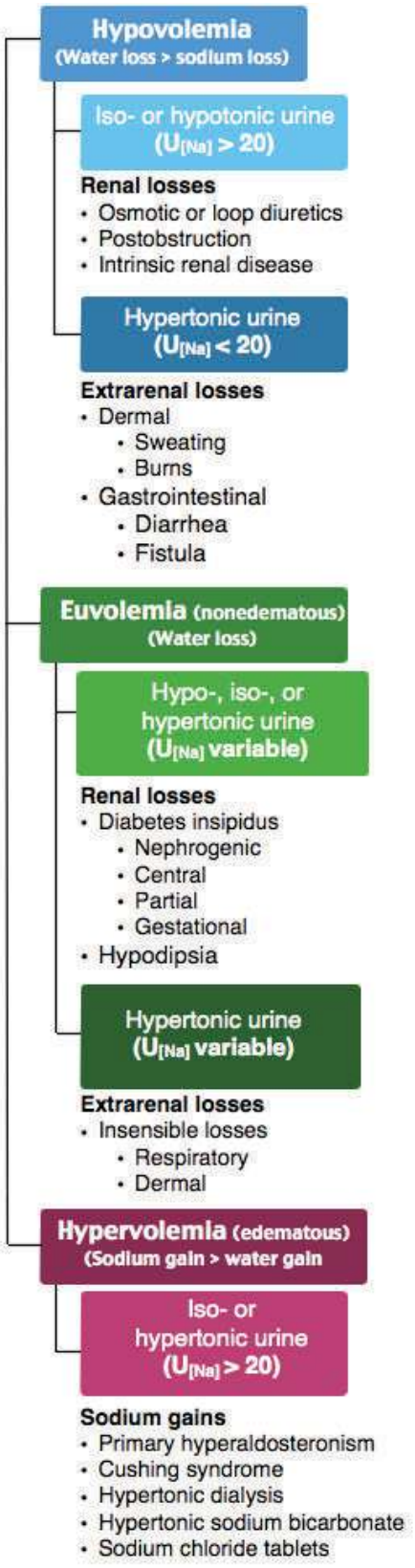
Comments
• **Central pontine myelinolysis** if corrected too fast




Notes


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## F. Hypernatremia




Notes

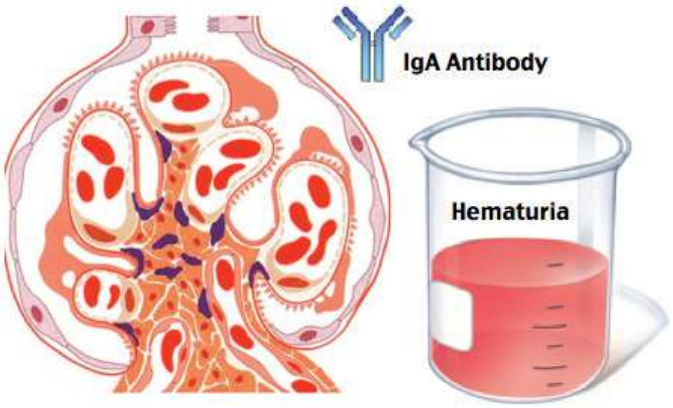

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# VI. Glomerulonephritis

### A. IgA Nephropathy (Berger disease)

Patient
• Often affects **young males** within days of URI or GI infection

Comments
• **Most common** cause of acute glomerulonephritis worldwide




• **Most common glomerulonephritis** worldwide
• **Deposition of IgA antibodies** in the glomerulus
• Episodic **hematuria** begins **1-2 days after URI** (**Poststreptococcal** glomerulonephritis occurs **weeks** after initial infection)

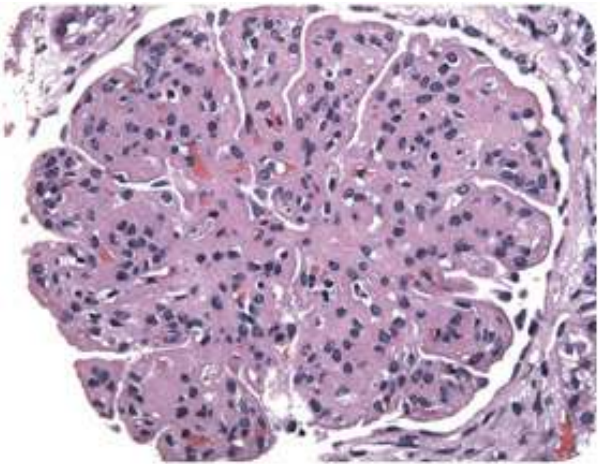
Notes


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### B. Membranoproliferative Glomerulonephritis

Patient
- **Most common** in **nondiabetic adults**

Comments
- Associated with **malignancies**



**Pathologic findings**
- Mesangial hypercellularity
- Endocapillary proliferation
- Capillary wall remodeling
- Double contour formation
- Duplication of basement membranes

**Etiology**

***Immune complex-mediated (Type I)***
- Infection (e.g., Hep B and C)
- Autoimmune (e.g., Lupus)
- Monoclonal gammopathy

***Complement-mediated (Type II)***
- Positive staining for C3 (or C4d)
- Normal C3 levels do not rule out

Notes

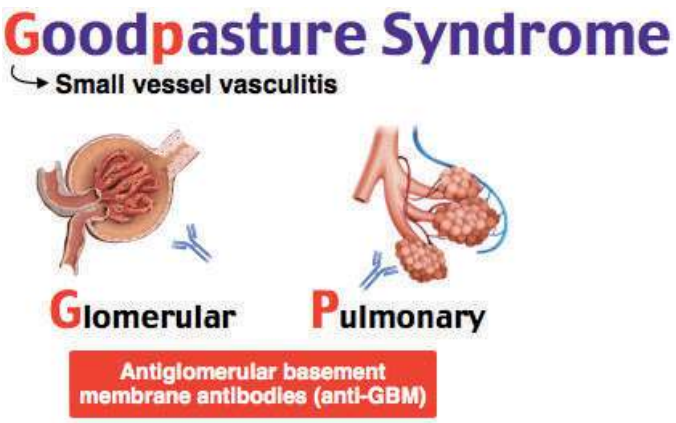

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### C. Goodpasture syndrome

Pathophysiology
• Small vessel vasculitis

Diagnostic Studies
• **Anti-GBM** antibodies
• **Linear IgG deposits** on biopsy

Management
• Treat with high-dose **steroids**, plasmapheresis, and **cyclophosphamide**




**Clinical**
• Cough
• Dyspnea
• Hemoptysis
• Glomerulonephritis

**Diagnosis**
• Renal and lung biopsy showing **antiglomerular basement membrane antibodies (anti-GBM)**

**Management**
• Supportive
• Prednisone
• Cyclophosphamide
• Plasmapheresis

Notes


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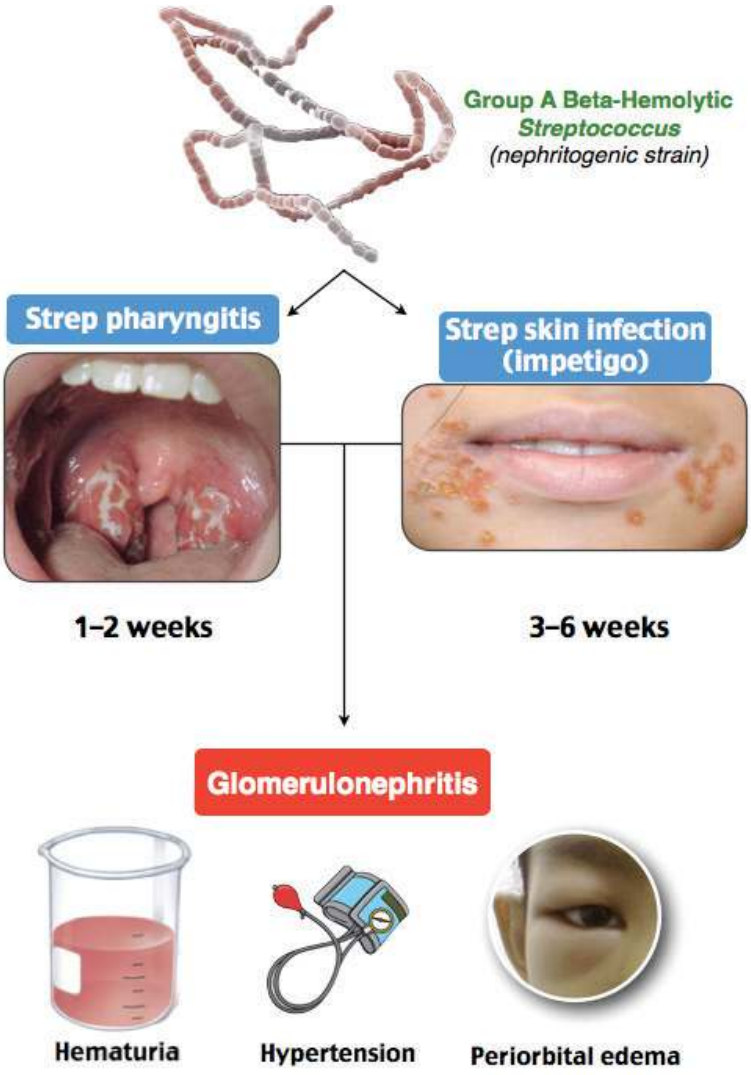
**D. Poststreptococcal Glomerulonephritis**

Pathophysiology
• **Most commonly** caused by group **A beta-hemolytic *Streptococcus***

Physical Exam
• **Hypertension**, **hematuria**, and **periorbital edema**

Diagnostic Studies
• **Proteinuria** and **red blood cell casts** in the urine

Comments
• **Most common** infectious cause of acute glomerulonephritis




Notes


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# VII. Hematuria

**A. Hematuria Rapid Associations**

Trauma: **bladder injury**, **ureter injury**, **retroperitoneal injury**

Anemia, thrombocytopenia, renal insufficiency: **HUS**, TTP

Flank pain: **nephrolithiasis**

UA with blood on dipstick, no RBCs: **rhabdomyolysis**

Dysuria, frequency: **UTI**

Hearing loss: **Alport syndrome**

Hemoptysis: **Goodpasture syndrome**

Immigrant: **Schistosomiasis**

Nephrotic syndrome, flank pain: **renal vein thrombosis**

Proteinuria, RBC casts: **glomerulonephritis**

Recent URI: **glomerulonephritis**, **IgA nephropathy**

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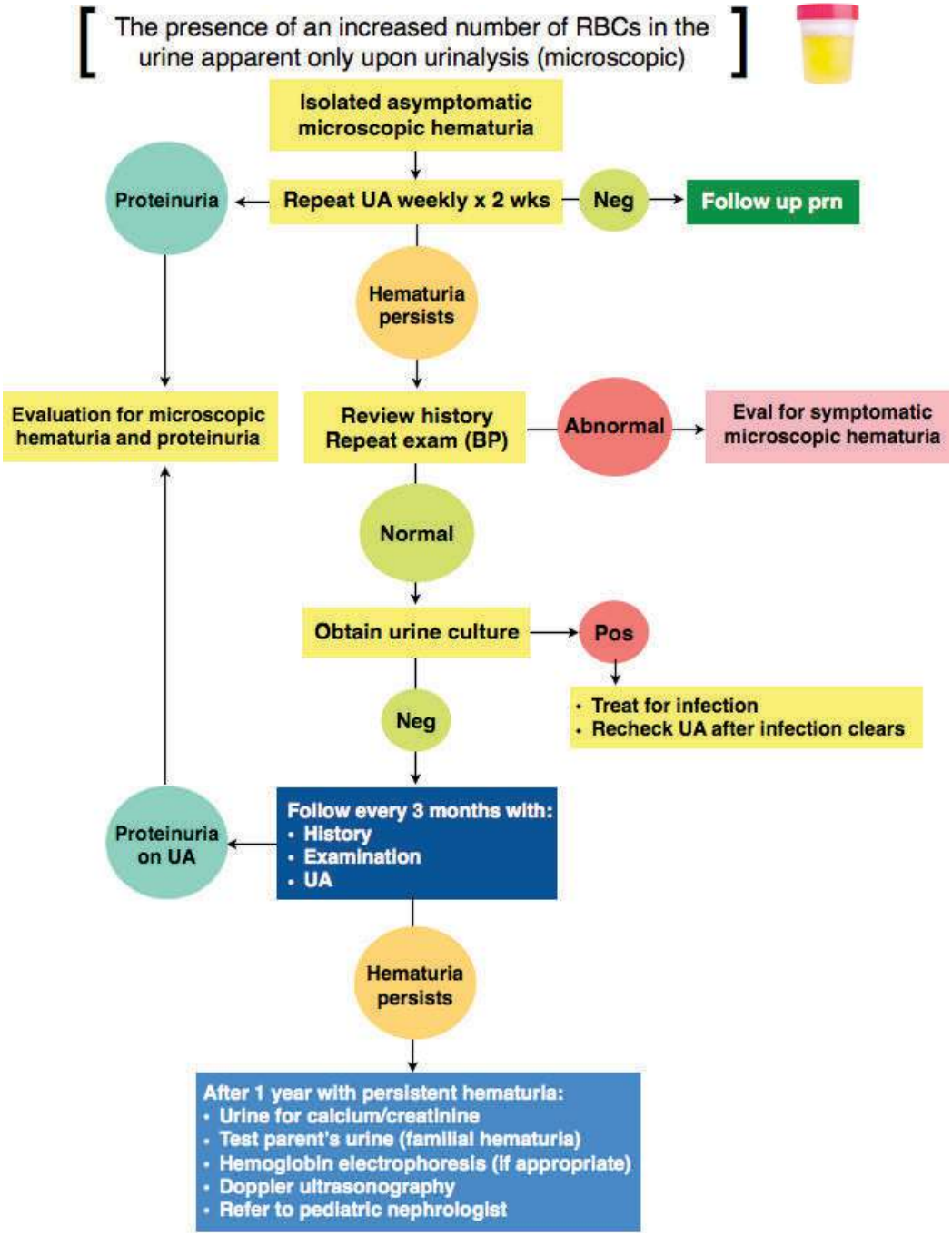
**B. Common Causes of Hematuria by Age**

| | |
|---|---|
| < 20 years | • UTI<br>• Glomerulonephritis<br>• Irritation of urethral meatus<br>• Congenital urinary tract anomaly with obstruction |
| 20–40 years | • UTI<br>• Bladder cancer<br>• Urolithiasis |
| 40–60 years | • UTI<br>• Bladder cancer<br>• Urolithiasis |
| > 60 years | • UTI<br>• Bladder cancer<br>• Benign prostatic hyperplasia (Men) |

Notes


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### C. Microscopic Hematuria




Notes


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# VIII. Hernias

### A. Inguinal Hernias

Pathophysiology
- Indirect:
  - **Most common**
  - Protrudes through internal ring, lateral to the inferior epigastric artery (**IEA**)
- Direct:
  - Protrudes **directly** through **Hesselbach triangle** and medial to the IEA
  - Bulge **improves** upon reclining

Patient
- **Bimodal** distribution ( < 1 year old, > 40 years old)

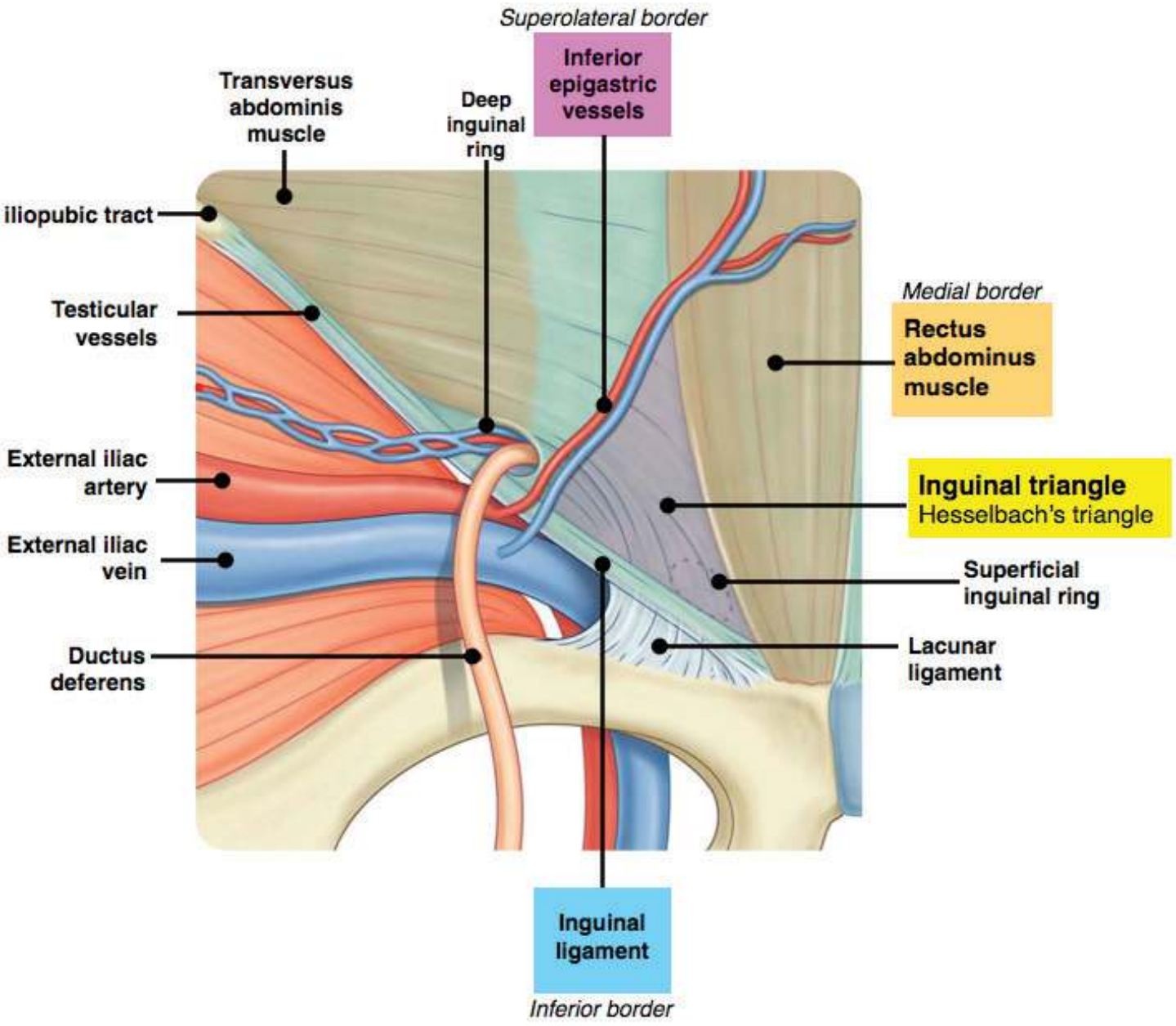
Management
- Nonreducible: **emergent** surgical consultation

Comments
- Strangulation risk **higher** with indirect hernia versus direct hernia

Notes


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**B. Inguinal Triangle (Hesselbach's triangle)**




Notes


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### C. Types of Hernias

| Hernias | | | |
|---|---|---|---|
| **Type** | **Pathophysiology** | **Location** | **Characteristics** |
| **Direct inguinal** | Defect of the transversalis fascia in Hesselbach triangle | Groin, **medial** to the inferior epigastric vessels | Middle-aged or **elderly men** |
| **Indirect inguinal** | Persistent processus | Groin, **lateral** to the inferior epigastric vessels | Congenital, trauma, can descend into **scrotum** |
| **Femoral** | Through femoral canal | **Upper thigh**, medial to the femoral vein | Most common in **women**, risk of incarceration and strangulation |
| **Spigelian (lateral ventral)** | Defect through the spigelian fascia (aponeurotic layer b/w rectus abdominus (medially), and semilunar line (laterally) | **Lateral** to rectus abdominus | Rare, small, high strangulation risk, right side |
| **Incisional/ ventral** | Breakdown of fascial closure from prior surgery | Site of **previous surgery** | Usually **asymptomatic**, increase in size with straining |
| **Umbilical** | Through the fibromuscular umbilical ring | Umbilicus | Repair if persists beyond 5 years |
| **Obturator** | Through the large obturator canal | Deep structures, not visualized externally | **Female** > male |
| **Epigastric** | Through **defects in aponeurosis of rectus sheath** | Midline between umbilicus and xiphoid process | Middle-aged and young children |

Notes


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# IX. Incontinence

### A. Urge Incontinence

Pathophysiology
• **Most commonly** caused by **overactive bladder** (**detrusor** overactivity)

Patient
• Woman

Presentation
• **Sudden**, **urgent** need to urinate resulting in urinary leakage
• Nocturia

Management
• **Anticholinergics** (oxybutynin)

### B. Stress Incontinence

Pathophysiology
• **Most commonly** caused by **muscle weakness**

Patient
• Woman

Presentation
• Small amount of urine loss when **coughing**, **laughing**, or **sneezing**

Management
• **Kegel** exercises, estrogen replacement, surgery

### C. Overflow Incontinence

Patient
• Woman

Presentation
• Feeling of incomplete bladder emptying
• Fullness, pressure, **dribbling**

Management
• **Alpha-blockers**

Notes


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### D. Types of Urinary Incontinence

| Type | Common Causes | Common Symptoms | Pharmacologic Treatments |
|---|---|---|---|
| **Urge incontinence** (detrusor overactivity) | • Stroke<br>• Alzheimer disease<br>• Parkinson disease<br>• BPH with overflow | • Urgency and frequency, day or night | • Anticholinergic drugs |
| **Stress incontinence** (outlet incompetence) | • Urologic procedures<br>• History of multiple childbirths | • Small volumes of urine loss with coughing or sneezing | • Topical estrogen |
| **Overflow incontinence** | • BPH<br>• Fecal impaction | • Poor stream<br>• Incomplete emptying | • Alpha-adrenergic blockers |
| **Atonic bladder** | • Severe diabetic neuropathy<br>• Stroke | • Complete loss of bladder control | • Intermittent catheterizations |
| **Functional incontinence** | • Inability to get to the bathroom | • Symptoms will vary | • Eliminate causes |

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# X. Nephrolithiasis/Urolithiasis

### A. Nephrolithiasis

Pathophysiology
- **Most commonly** caused by **calcium oxalate**
  - **Struvite**: **s**taghorn calculi, **urease**-producing bacteria
  - **U**ric acid: radiol**U**cent on X-ray, gout
  - **C**ystine: **c**hildren with metabolic diseases
- **Most common** location is the ureterovesicular junction (**UVJ**)

Presentation
- **Flank pain radiating** to **groin**

Physical Exam
- Restless, uncomfortable appearing patient

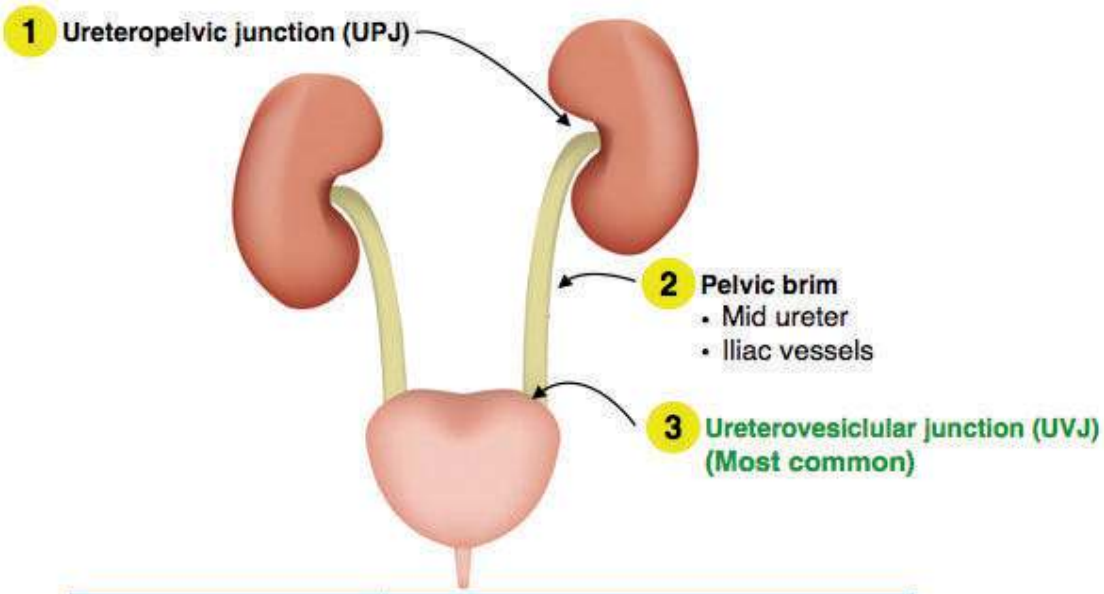
Diagnostic Studies
- **Helical CT** is diagnostic

Management
- < 5 mm: likely to pass spontaneously
- > 8 mm: unlikely to pass; **lithotripsy**
- **Emergent stenting** if stone is obstructing (associated with infection)

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**B. Kidney Stone**




| Size of Stone (mm) | Likelihood of Spontaneously Passing |
|---|---|
| < 5 | 90% |
| 5 to 8 | 15% |
| > 8 | 5% |

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# XI. Orchitis

## A. Orchitis

Pathophysiology
- **Inflammation** of the testes
- **Viral** **more common** than bacterial

Patient
- Orchitis without epididymitis **more common** in adolescents and children
  - In adults consider **epididymo-orchitis** (see "epididymitis" above for management)

Presentation
- **Acute** onset of scrotal swelling and pain

Physical Exam
- Glossy-appearing erythema
- Normal cremasteric reflex



**Inflammation of one or both testicles**

**Etiology (Children and adolescents)**
**Viral**
- Mumps
- Rubella
- Coxsackie
- Echovirus
- Lymphocytic choriomeningitis virus
- Parvovirus

**Bacterial**
- Brucellosis

**Clinical**
- Scrotal swelling, pain, tenderness
- Scrotal erythema




Notes


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# XII. Prostatitis

### A. Prostatitis

Pathophysiology
• < 35 years old: **most commonly *N. gonorrhoeae***, ***C. trachomatis***
• > 35 years old: **most commonly *E. coli***

Presentation
• **Fever**, **chills**, **perineal/back pain**, and **dysuria**

Physical Exam
• **Warm**, **exquisitely tender** prostate

Management
• < 35 years old: ceftriaxone or ofloxacin and doxycycline
• > 35 years old: ciprofloxacin or TMP-SMX
• Chronic prostatitis: fluoroquinolones or TMP-SMX for **6–12 weeks**

Comments
• **Avoid** vigorous prostatic **massage** as this can lead to **septicemia**

Notes


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## B. Acute Prostatitis

↳ **Reflux of urine from urethra into intraprostatic ducts**

**Microbiology**
- *E. coli*
- *Proteus spp.*
- Other Enterobacteriaceae
- *Pseudomonas aeruginosa*

**Clinical**
- Fever
- Dysuria
- Low back and perineal pain
- Urinary retention
- Tender boggy prostate
- Pyuria

**Treatment**
- Trimethoprim-sulfamethoxazole
- Fluoroquinolone



**Sexually active men**

**Microbiology**
- *Neisseria gonorrhoeae*
- *Chlamydia trachomatis*

**Treatment**
- Ceftriaxone
- Doxycycline

**Treat for 4–6 weeks**

Notes


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### C. Chronic Bacterial Prostatitis

↳ **Reflux of urine from urethra into intraprostatic ducts**

**Microbiology**
- *E. coli* (most common)
- *Proteus mirabilis*
- *Enterococcus faecalis*
- *Klebsiella pneumoniae*
- *Psuedomonas aeruginosa*

**Clinical**
- Recurrent UTI
- Low back and perineal pain
- Urinary retention
- Tender boggy prostate

**Treatment**
- Trimethoprim-sulfamethoxazole
- Fluoroquinolone






Notes


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# XIII. Pyelonephritis

### A. Acute Pyelonephritis

Pathophysiology
• **Most commonly** caused by ***E. coli***

Presentation
• **Fever**, dysuria, **flank pain**, nausea and vomiting

Physical Exam
• CVA tenderness

Management
• **Fluoroquinolone** or TMP-SMX
• If an inpatient or pregnant: ampicillin/gentamicin or third-gen cephalosporin

**Microbiology**

*Ascending (most common)*
• *E. coli* (75% to 95%)
• *Proteus mirabilis*
• *Klebsiella pneumoniae*
• *Staphylococcus saprophyticus*

*Descending (less common)*
• Septicemia
• Infective endocarditis

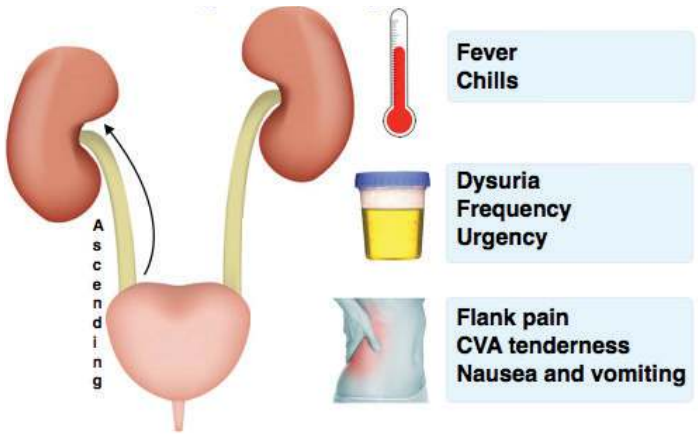
**Management**

*Oral (outpatient)*
• Ciprofloxacin, levofloxacin
• Trimethoprim-sulfamethoxazole

*Parenteral (mild to moderate)*
• Ceftriaxone
• Ciprofloxacin, levofloxacin
• Aztreonam

*Parenteral (severe)*
• Cefepime
• Pipercillin-tazobactam
• Meropenem

Notes





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# XIV. Testicular Torsion

### A. Testicular Torsion

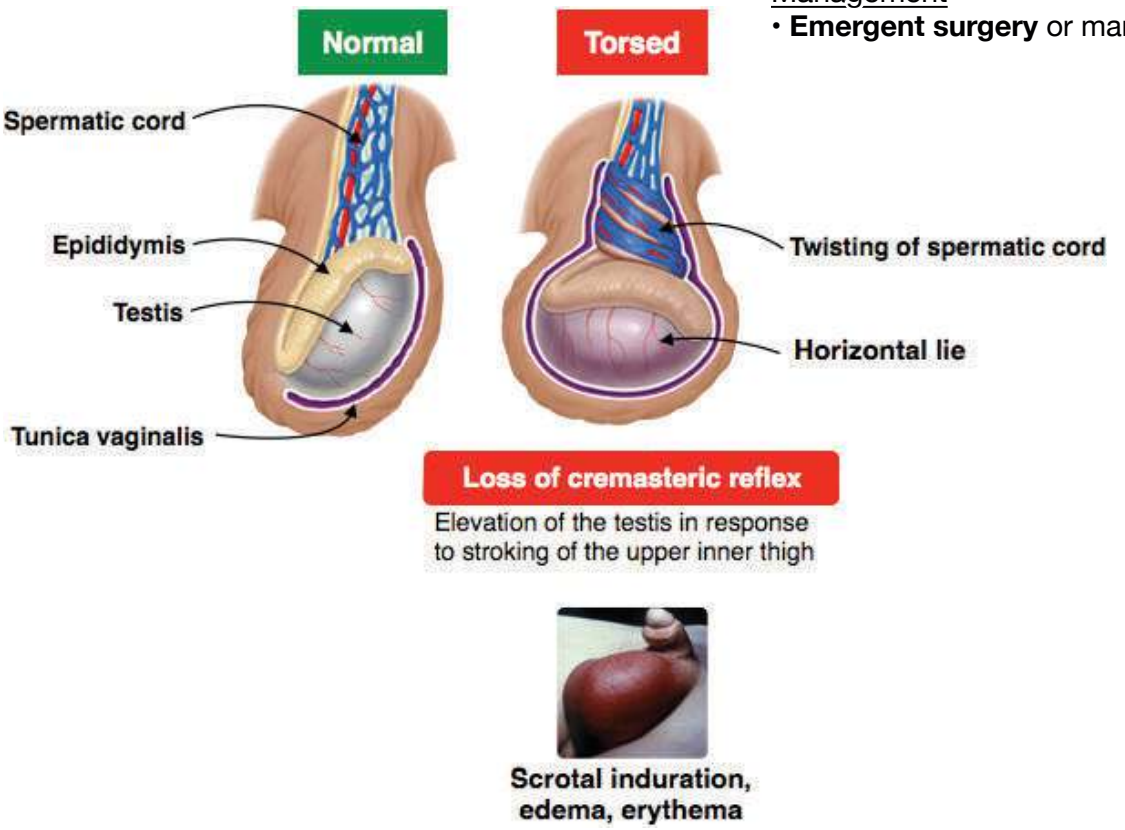
Patient
• Young male

Presentation
• **Intense** scrotal pain, can be intermittent

Physical Exam
• Horizontal, high-riding testicle, exquisite tenderness, and **absent cremasteric reflex**

Diagnostic Studies
• Ultrasound with Doppler

Management
• **Emergent surgery** or manual detorsion




**Loss of cremasteric reflex**
Elevation of the testis in response to stroking of the upper inner thigh



**Scrotal induration, edema, erythema**

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# XV. Urethritis

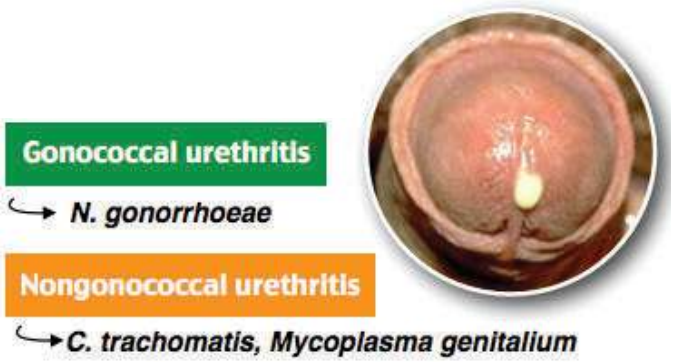
### A. Gonococcal Urethritis

Pathophysiology
• **Most commonly** caused by ***Neisseria gonorrhoeae***

Presentation
• **Purulent** urethral discharge and dysuria

Diagnostic Studies
• **Gold standard** diagnosis with urine culture (**gram-negative diplococci**)
• PCR both sensitive and specific

Management
• Ceftriaxone 250 mg IM **and** azithromycin 1 g PO **or** doxycycline 100 mg BID for seven days




**Clinical**
• Dysuria
• Pruritus
• Discharge at urethral meatus

**Treatment**
• Ceftriaxone 250 mg IM AND
• Azithromycin 1 g PO or doxycycline 100 mg BID x 7 days

Notes


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CHAPTER 8

# Dermatology

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CHAPTER 8

# Dermatology


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# I. Bullous Pemphigoid

### A. Bullous Pemphigoid

Pathophysiology
• **Most commonly** caused by a chronic **autoimmune** blistering disease

Patient
• Age > **60 years old**

Presentation
• **Intensely pruritic papules** that become large, tense **blisters** and **bullae**

Physical Exam
• **Tense** and **firm** blisters that **do not** extend with lateral pressure (**Negative Nikolsky sign**)

Management
• **Corticosteroids** and **immunosuppressants**





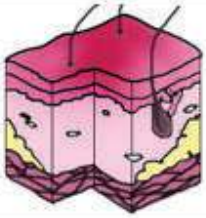
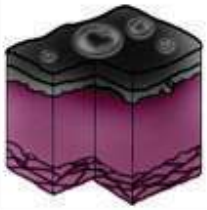


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# II. Burns

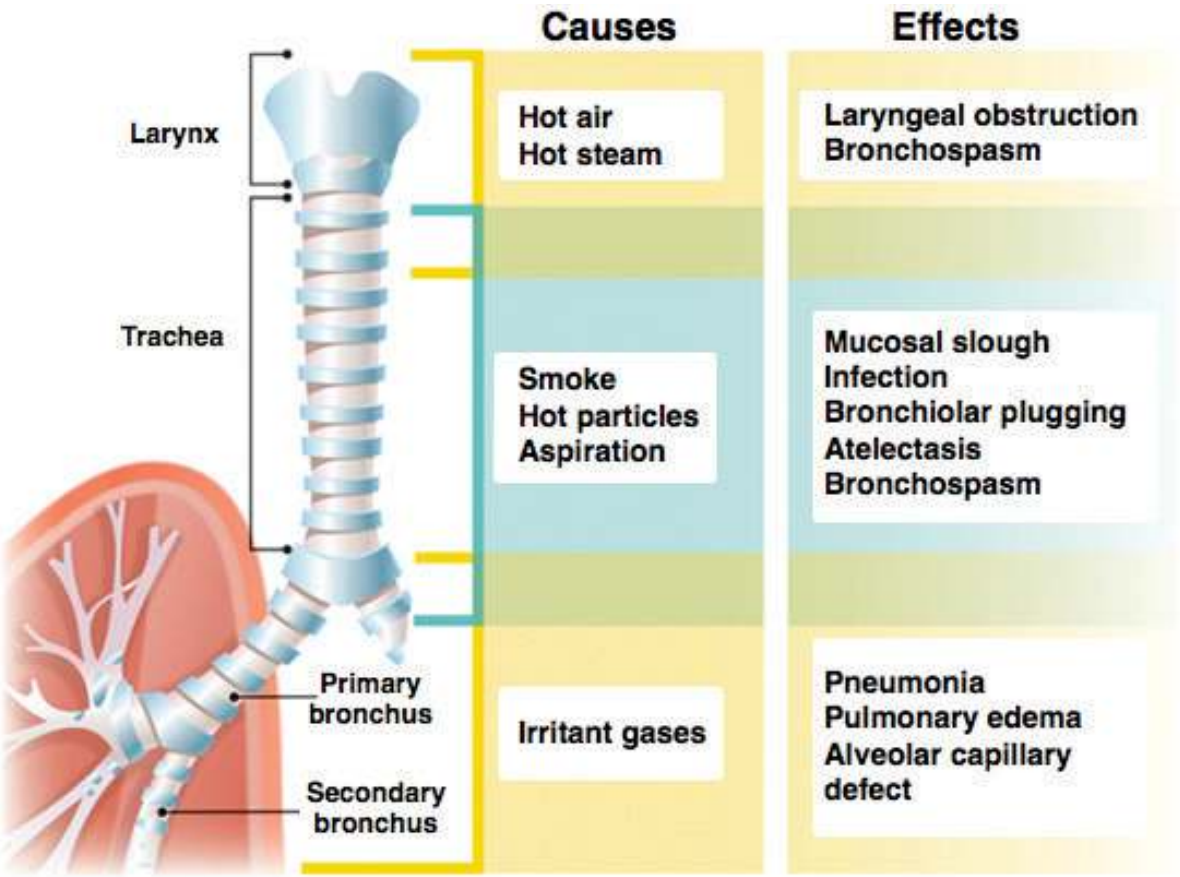
### A. Burn Classification

| Clinical appearance | Thickness | Degree | Depth | Characteristics |
|---|---|---|---|---|
|   | Superficial | 1st | Epidermis | Pain, redness, mild swelling |
|   | Superficial Partial | 2nd | Dermis: papillary region | Pain, blisters, splotchy skin, severe swelling |
|  | Deep partial | | Dermis: reticular region | White, leathery, relatively painless |
|   | Full | 3rd | Hypodermis (subcutaneous tissue) | Charred, insensate, eschar formation |

Notes


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**B. Respiratory Injury from Burns**

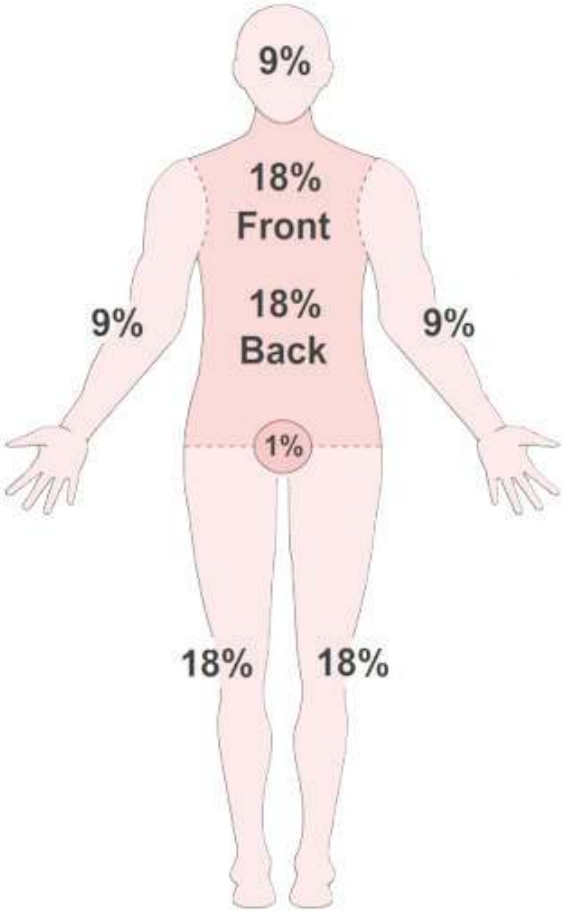


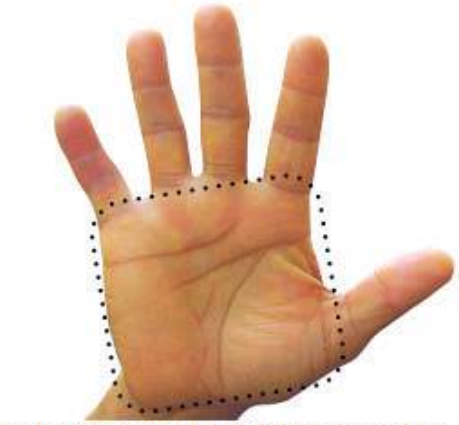

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### C. Rule of Nines

**Only for 2nd and 3rd degree burns**



Patient's PALM approximates 1% total body surface area

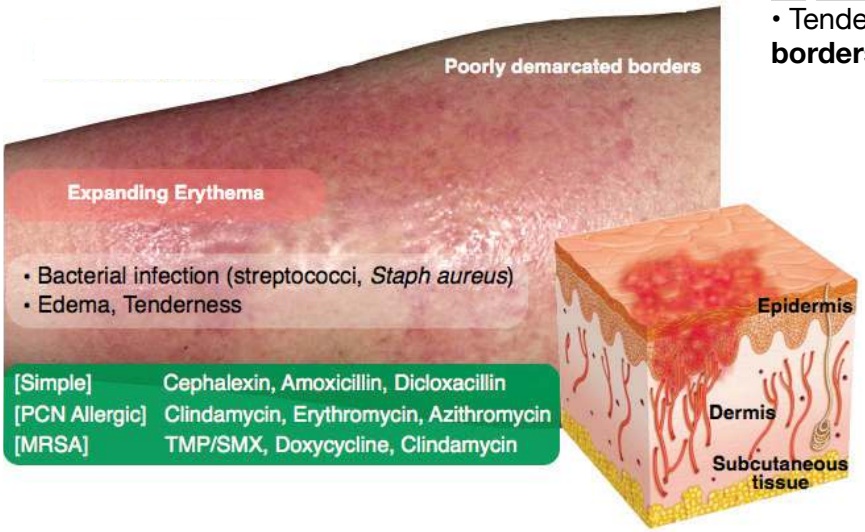
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# III. Cellulitis

## A. Cellulitis

Pathophysiology
• **Most commonly** caused by ***S. aureus*** and streptococci




Presentation
• **Pain**, **redness**, **swelling**

Physical Exam
• Tenderness, **erythema with poorly demarcated borders**, lymphedema

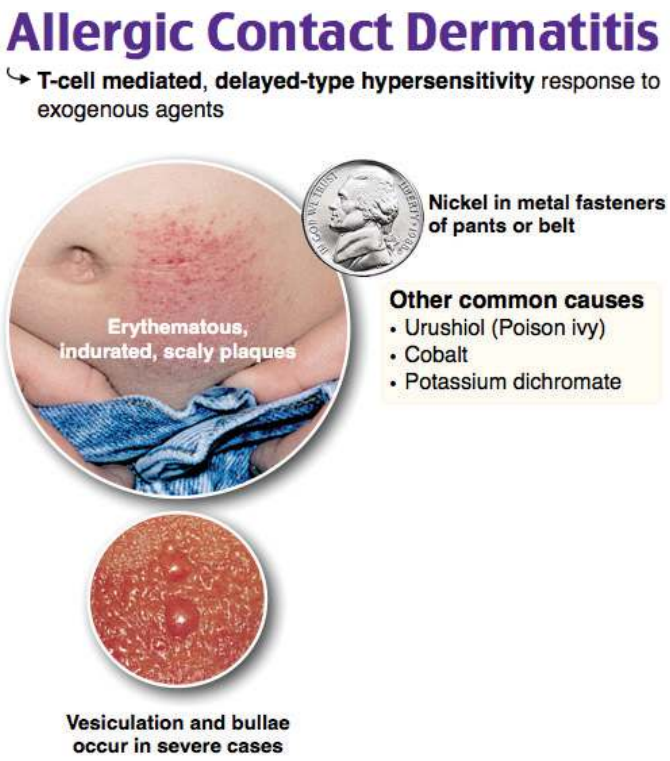
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# IV. Dermatitis (Eczema, Contact)

### A. Contact Dermatitis

• Well-demarcated erythema, erosions, vesicles




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**B. Atopic Dermatitis (Eczema)**

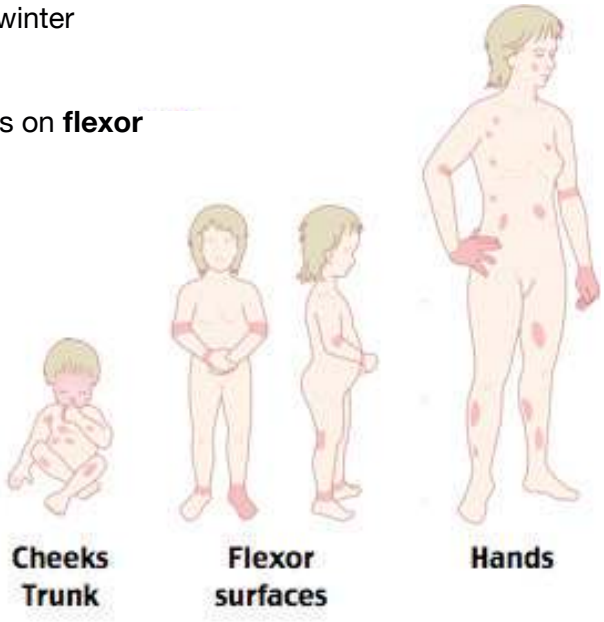
Patient
• History of **asthma** or **allergic rhinitis**

Presentation
• **Itchy**, **scaly** rash often **worse** in the winter

Physical Exam
• Thick, leathery, hyperpigmented areas on **flexor** surfaces

Management
• **Topical corticosteroids** and emollients




***Sine qua non*** → **PRURITUS**

→ **Vicious cycle of itch-scratch-rash-itch**

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### C. Nummular Eczema (Discoid Eczema)

• Coin-shaped and discoid plaques




**Clinical**
• Highly pruritic, round, coin-shaped patches (1 to 10 cm in diameter)
• Acute: lesions are dull red, exudative, and crusted
• Over time: dry, scaly, with occasional central clearing
• Legs and upper extremities most commonly involved
• Chronic and relapsing

**Management**
• Measures to reduce skin dryness and exposure to irritants
• Corticosteroids
• Phototherapy (severe or refractory)
• Methotrexate, cyclosporin

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# V. Drug Eruptions

### A. Drug Eruptions

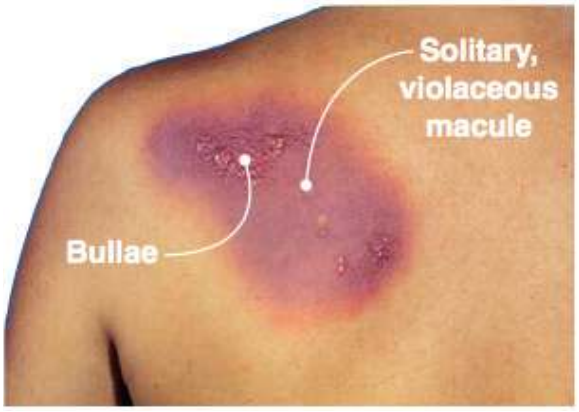
Pathophysiology
• An adverse cutaneous reaction in response to administration of a drug
• Severity can range from mild eruptions that resolve after the removal of the inciting agent to severe skin damage with multiorgan involvement

Comments
• Skin reactions are the **most common** adverse drug reactions

Notes


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### B. Fixed Drug Eruption




**Eliciting Drugs**
- Trimethoprim-sulfamethoxazole, tetracyclines, penicillins, quinolones, dapsone
- NSAIDs, acetylsalicylic acid
- Acetaminophen
- Barbiturates
- Antimalarials

**Clinical**
- Systemic symptoms are usually absent
- Pruritus and a burning or stinging sensation are common
- May occur anywhere on body

**Management**
- Drug withdrawal and avoidance
- Supportive care (e.g., diphenhydramine)

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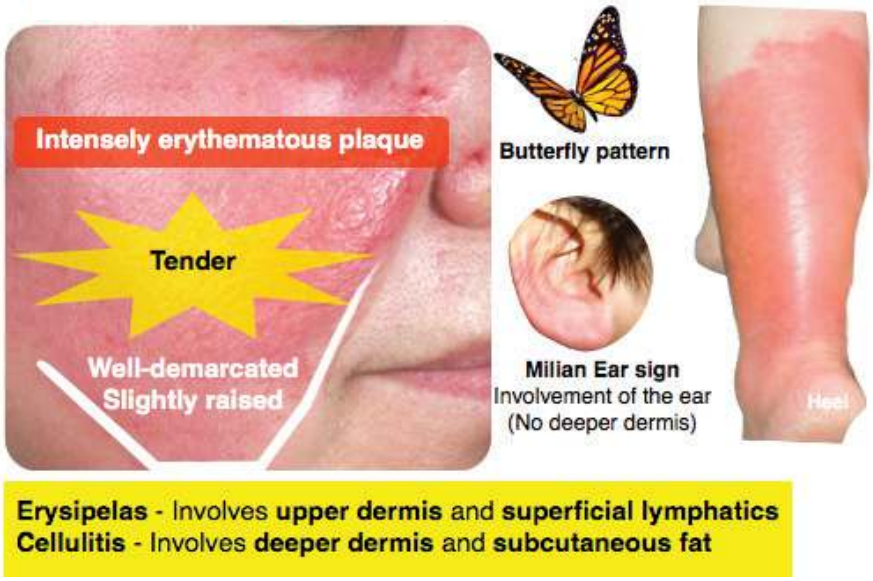
# VI. Erysipelas

## A. Erysipelas

Pathophysiology
• **Most commonly** caused by ***Streptococcus pyogenes*** infection (group A beta-hemolytic *Strep*)

Presentation
• Malaise, fever, chills, nausea

Physical Exam
• Intensely erythematous, **sharply demarcated**, **slightly raised** plaque




**Erysipelas** - Involves **upper dermis** and **superficial lymphatics**
**Cellulitis** - Involves **deeper dermis** and **subcutaneous fat**

Management
• Amoxicillin or cephalexin (mild to moderate disease)
• Ceftriaxone or cefazolin (systemic symptoms)

**Clinical**
• Tend to have acute onset with systemic manifestations
• Fever and chills
• Cellulitis tends to be more indolent

**Management**
• Elevation of affected area (e.g., limb)
• Ceftriaxone, cefazolin (systemic symptoms)
• Amoxicillin, cephalexin (mild to moderate)

Most commonly caused by
**Beta-hemolytic streptococci**
(*Streptococcus* pyogenes)

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# VII. Herpes Zoster

### A. Herpes Zoster (Shingles)

Pathophysiology
• Caused by reactivation of latent **varicella-zoster virus**

Patient
• Elderly

Presentation
• Painful, papulovesicular rash **preceded by tingling** or **hyperesthesia**

Physical Exam
• Rash with unilateral dermatomal distribution that **does not cross midline**

Diagnostic Studies
• **Tzanck smear** will reveal **multinucleated giant cells**

Management
• Acyclovir

Comments
• **Postherpetic neuralgia**: persistent pain > three months
• **Vaccination** indicated at age 60

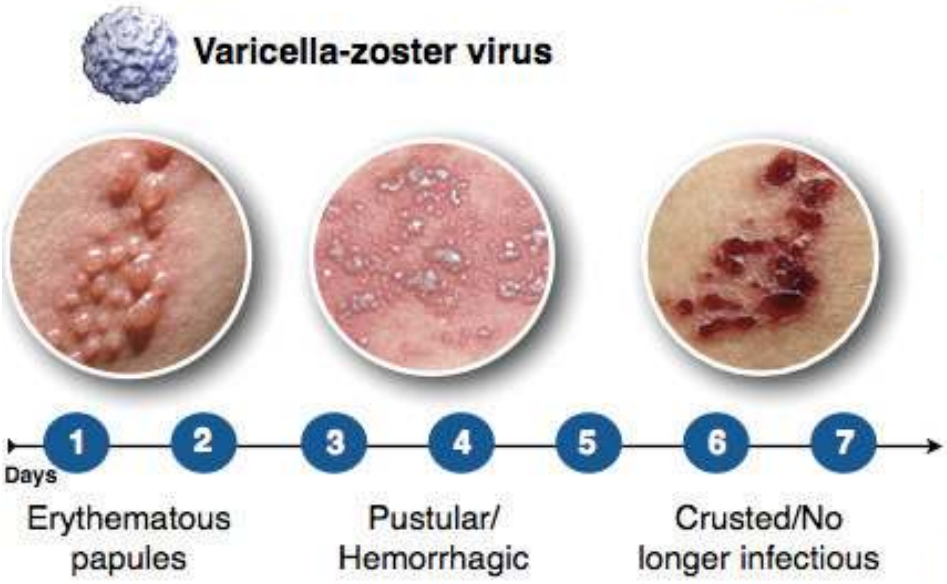
**Clinical**
• Rash
• Acute neuritis (pain)

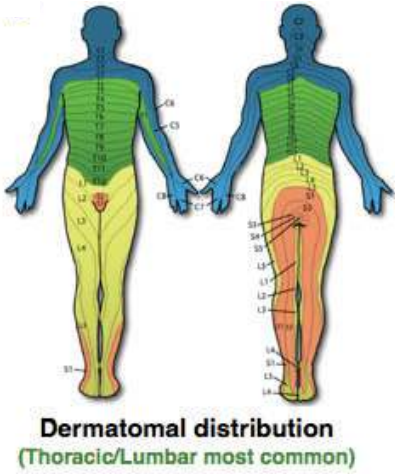
**Complications**
• Postherpetic neuralgia (8%)
• Bacterial skin infection (2%)
• Uveitis/Keratitis (1.5%)
• Motor neuropathy (1%)
• Meningitis (0.5%)
• Zoster oticus (0.2%)

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**Herpes Zoster (Shingles) cont.**






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# VIII. Impetigo

## A. Impetigo

Pathophysiology
• **Most commonly** caused by ***S. aureus***, *Streptococcus*

Patient
• Child younger than six years of age

Presentation
• **Nonpainful**, **pruritic lesions** on face

Physical Exam
• **Honey-colored** weeping lesions with **crusting**

Management
• Topical **mupirocin**

Comments
• Complications include **poststreptococcal glomerulonephritis**

**Treatment**
• Limited: Topical **mupirocin** or retapamulin
• Extensive: Systemic antibiotics (dicloxacillin, cephalexin)
• If MRSA suspected: Trimethoprim-sulfamethoxazole

**Microbiology**
• *Staphylococcus aureus* (most common)
• Beta-hemolytic streptococci

**1 Nonbullous impetigo**



• Papules, vesicles, and pustules
• Rapidly break down
• Form golden adherent crusts
• Often located on face or extremities

**2 Bullous impetigo**



• Flaccid, fluid-filled bullae
• Rupture
• Leaves a thin brown crust
• Often located on trunk

**3 Ecthyma**



• "Punched-out" ulcers
• Overlying crust
• Raised violaceous borders

Notes


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# IX. Lice

### A. Pediculosis Capitis (Head Lice)

Pathophysiology
• Caused by ***Pediculus humanus capitis***

Patient
• Child

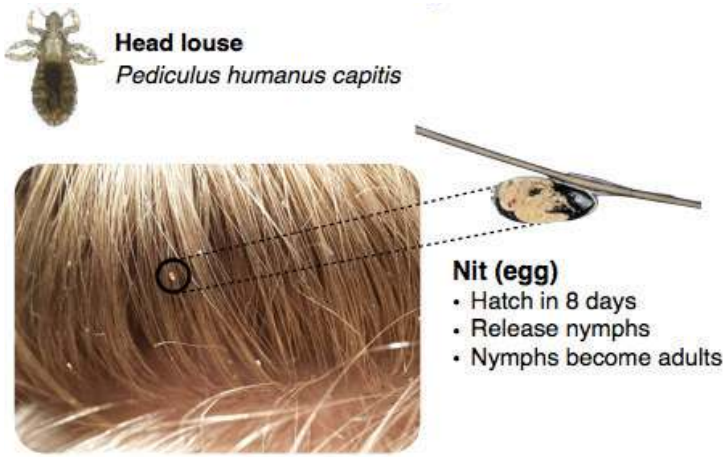
Presentation
• Scalp itching

Physical Exam
• Nits (eggs) or actual lice

Management
• Topical pediculicides, such as **permethrin**

**Treatment**
• Wet combing (young infants)
• Topical pediculicides
  • Permethrin
  • Malathion
  • Benzyl alcohol
  • Spinosad
  • Topical ivermectin

Comments
• A second treatment should be applied on **day nine** after the first treatment to ensure eradication




**Transmission**
• Direct contact with head of infested person
• Lice do not jump, fly, or use pets as vectors

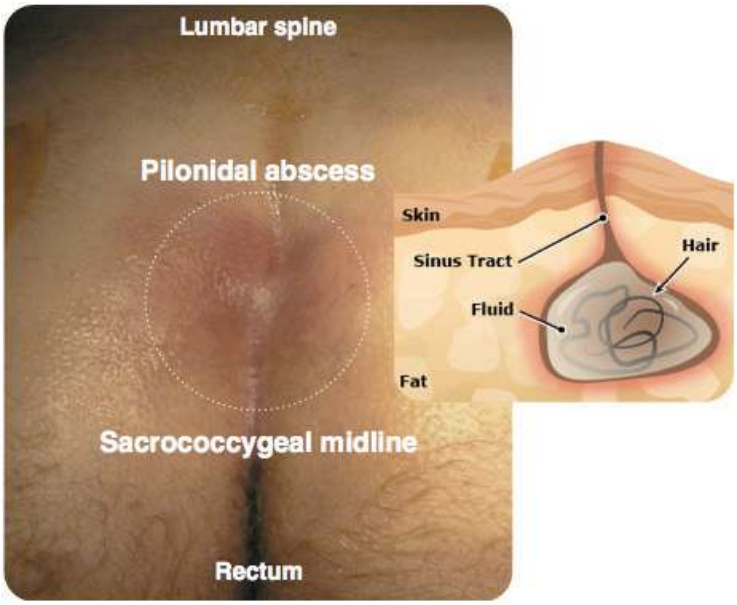
**Clinical**
• Pruritus (reaction to saliva injected during feeding)

Notes


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# X. Pilonidal Disease

### A. Pilonidal Disease




Patient
• **Man < 40 years old**

Presentation
• Painful area over tailbone

Physical Exam
• **Tender**, **fluctuant** area in the **sacrococcygeal cleft**

Management
• **Acute**: incision and drainage
• **Definitive**: surgical excision

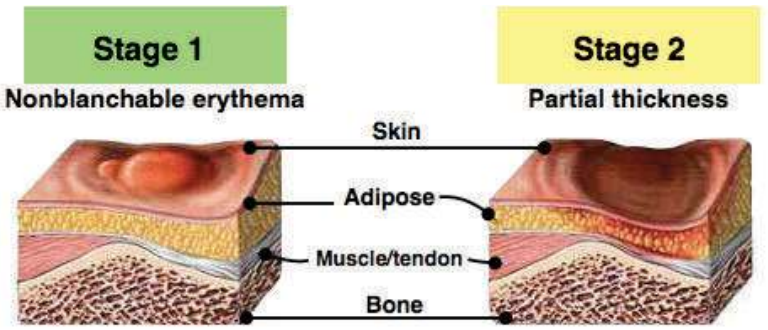
**Management**
• Incision and drainage (acute)
• Surgical excision (definitive)

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# XI. Pressure Sores/Rash

### A. Pressure Ulcer Stages




- Intact skin
- May be painful

- Shallow open ulcer
- Red/pink wound bed




- Subcutaneous fat may be visible
- Bone, muscle, tendon not exposed
- May include tunneling

- Exposed bone, tendon, or muscle
- Slough or eschar may be present
- Undermining and tunneling

Notes


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## B. Rash

| Classic Drug Reaction Patterns | | |
|---|---|---|
| **Type** | **Characteristics** | **Example** |
| **Drug-induced exanthems** | • Most common cutaneous drug reaction |  |
| **Symmetrical drug-related intertriginous and flexural exanthema** | • Sharply demarcated<br>• Involvement of flexural or intertriginous fold<br>• No systemic symptoms |  |
| **Urticaria** | • Pruritic<br>• Raised<br>• Central pallor |  |
| **Cutaneous small vessel vasculitis** | • A single organ vasculitis |  |
| **Exfoliative dermatitis** | • Chronic erythema and scale involving > 90% of body surface |  |
| **Stevens-Johnson syndrome** | • Severe mucocutaneous reaction<br>• < 10% body surface |  |
| **Toxic epidermal necrolysis** | • Severe mucocutaneous reaction<br>• > 30% body surface |  |
| **Erythema multiforme** | • Target lesions |  |
| **Drug reaction with eosinophilia and systemic symptoms (DRESS)** | • Idiosyncratic reaction<br>• Fever, malaise, lymphadenopathy |  |
| **Fixed drug reaction** | • Erythematous and edematous plaques with grayish center or frank bullae |  |


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# XII. Scabies

### A. Scabies

Pathophysiology
• **Most commonly** caused by *Sarcoptes scabiei hominis*

Presentation
• **Severe pruritus** that is **worse** at **night**

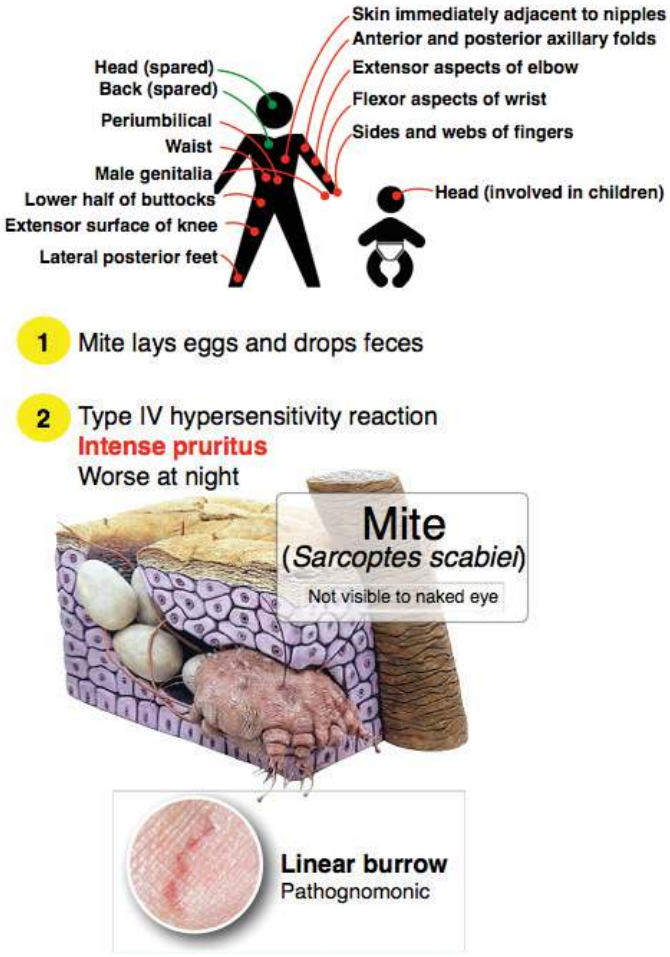
Physical Exam
• Small papules, **vesicles**, **and linear burrows in the webbed spaces of the fingers and toes**

Diagnostic Studies
• Microscopic visualization

Management
• **Permethrin**

**Clinical - Rash**
• Small, erythematous, nondescript papules
• Often excoriated, hemorrhagic crusts

**Management**
*Eradication of mites*
• Permethrin 5% cream (safe for infants)
• Oral Ivermectin (ideal for nursing home outbreaks)




Notes


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# XIII. Spider, Snake, Scorpion Bites

| Organism | Location (USA) | Unique Physical Characteristics | Incidence of Toxicity | Classic Findings | Antivenin Available |
|---|---|---|---|---|---|
|  Scorpion | Southwest | Tan colored | Frequent systemic sxs | • Fasciculations<br>• Disconjugate gaze<br>• Temperature reversal | Yes |
|  Brown recluse | Midwest | Fiddle/Violin on dorsal cephalothorax  | 10% develop necrotic ulcer | • Papule to necrotic blister | No |
|  Black widow | Nationwide | Red hourglass on ventral abdomen  | Systemic sxs common | • Local papule with halo<br>• Muscle fasciculations<br>• Abdominal pain<br>• Diaphoresis | Yes |
|  Pit viper | None in Maine, Alaska, Hawaii | Triangular head and elliptical pupils  | 75% positive for envenomation | • Local swelling<br>• Oozing blood from wound<br>• Coagulopathy | Yes |
|  Eastern coral | Southeast | Red on yellow stripes with black in between | 60% positive for envenomation | • Muscle paralysis | Yes |

Notes


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# XIV. Stevens-Johnson Syndrome

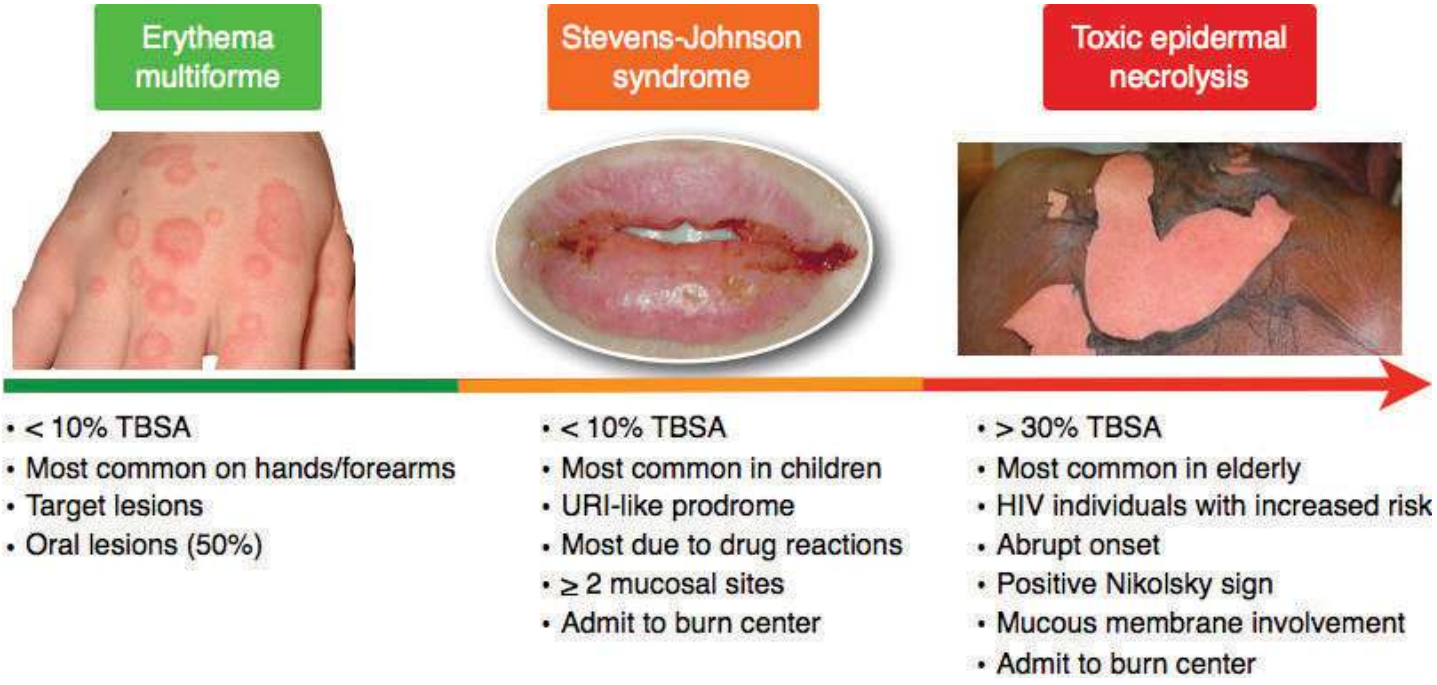
### A. Stevens-Johnson Syndrome

Pathophysiology
• **Most commonly** caused by a reaction to medications

Physical Exam
• Vesicles and bullae **involving < 10% of the body surface area including mucous membranes**

Management
• Referral to **burn center**

Comments
• **SCORTEN** score is used to determine the prognosis and clinical setting for treatment




Notes


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# XV. Toxic Epidermal Necrolysis

## A. Toxic Epidermal Necrolysis

Patient
• Elderly, increased risk if **HIV** positive

Physical Exam
• Vesicles and bullae **involving > 30% of the body surface area including mucous membranes**
• **Positive Nikolsky** sign (extension of lesion with lateral pressure)

Management
• Referral to **burn center**

**Management**
• Supportive
• Transfer to Burn Center

**Etiology**
*Drugs*
• Sulfonamides
• Phenobarbital, carbamazepine, lamotrigine
• Allopurinol
• NSAIDs
*Infection*
• *Mycoplasma pneumoniae*

**Clinical**
• Prodrome (influenza-like)
• Cutaneous lesions (coalescing erythematous macules with purpuric centers)
• Mucosal lesions (can precede or follow skin eruptions)
• Urethritis




Notes


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# XVI. Urticaria

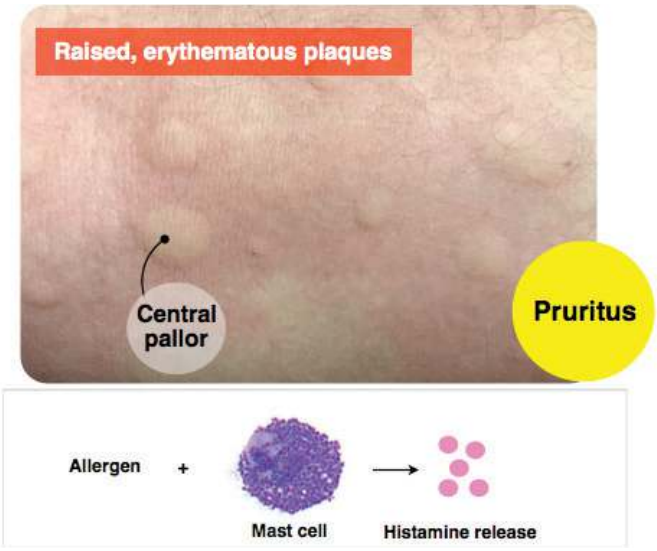
### A. Urticaria

Presentation
- **Blanchable**, edematous, pink papules, wheals, or plaques
- **Angioedema**: painless, deeper form of urticaria affecting the lips, tongue, eyelids, hands, and genitals

Physical Exam
- **Darier sign**: localized urticaria appearing where the skin is rubbed (histamine release)

Management
- If anaphylaxis: **epinephrine** 0.3–0.5 mg; use 1 : 1,000 dilution for IM route (Peds: epinephrine 0.01 mg/kg IM)

**Management**
- Supportive care
- Antihistamines
- Glucocorticoids (if associated with angioedema)




Notes


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**B. Common Causes of Urticaria**

| | | |
|---|---|---|
| **Drugs** | • Penicillin<br>• Sulfa<br>• Aspirin<br>• Local anesthetics<br>• Diuretics | • NSAIDs<br>• Morphine<br>• Codeine<br>• Progesterone |
| **Infection** | • Epstein-Barr virus<br>• Hepatitis B virus | • Coxsackie virus<br>• Parasitic infections |
| **Environmental** | • Heat<br>• Cold<br>• Exercise | • Metals<br>• Animal saliva |
| **Food** | • Fish<br>• Eggs<br>• Nuts | • Shellfish<br>• Fruits |
| **Other** | • Latex<br>• Pregnancy<br>• Malignancy | |

Notes


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# XVII. Viral Exanthems

## A. "Erythema" Rashes




Notes


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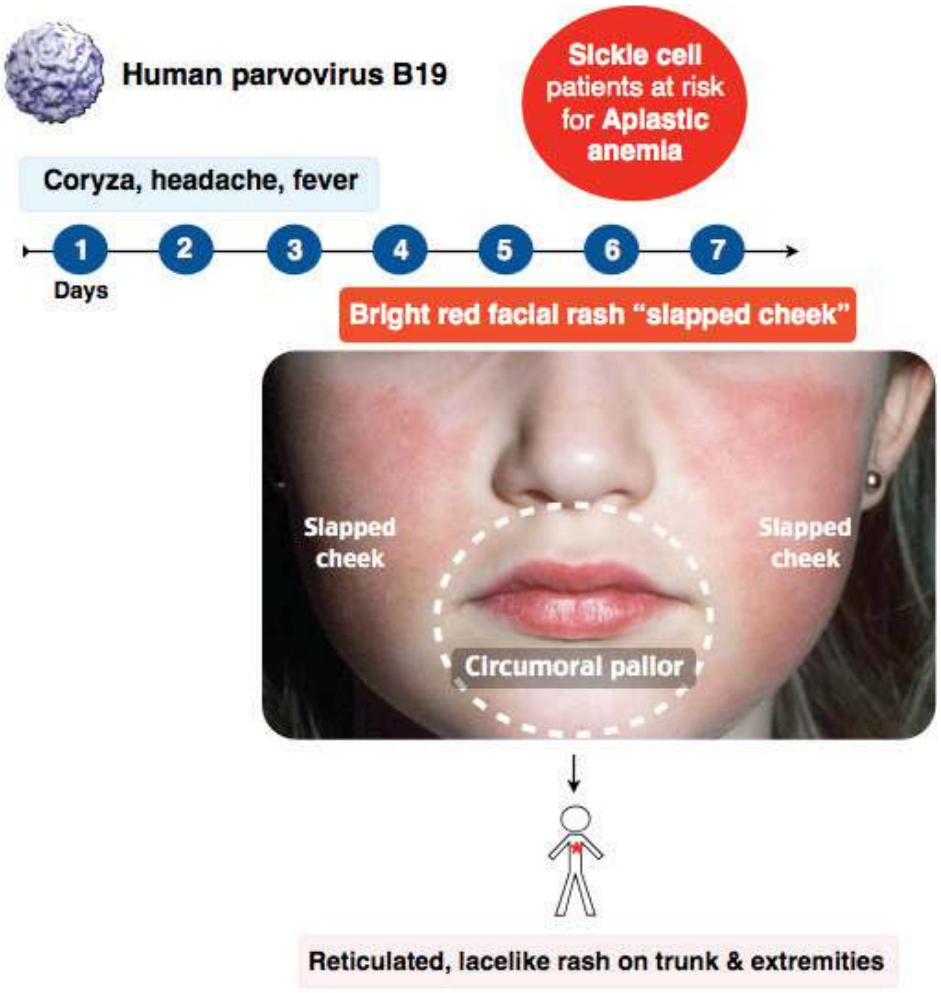
### B. Erythema Infectiosum (Fifth Disease)

Pathophysiology
• **Most commonly** caused by **parvovirus B19**

Patient
• History of **URI symptoms** 3–4 days prior of rash onset

Physical Exam
• **"Slapped cheek"** rash

Comments
• Complications may include **aplastic crisis** in sickle cell patients




Notes


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### C. Hand, Foot, and Mouth Disease

Pathophysiology
• **Most commonly** caused by **Coxsackievirus A**

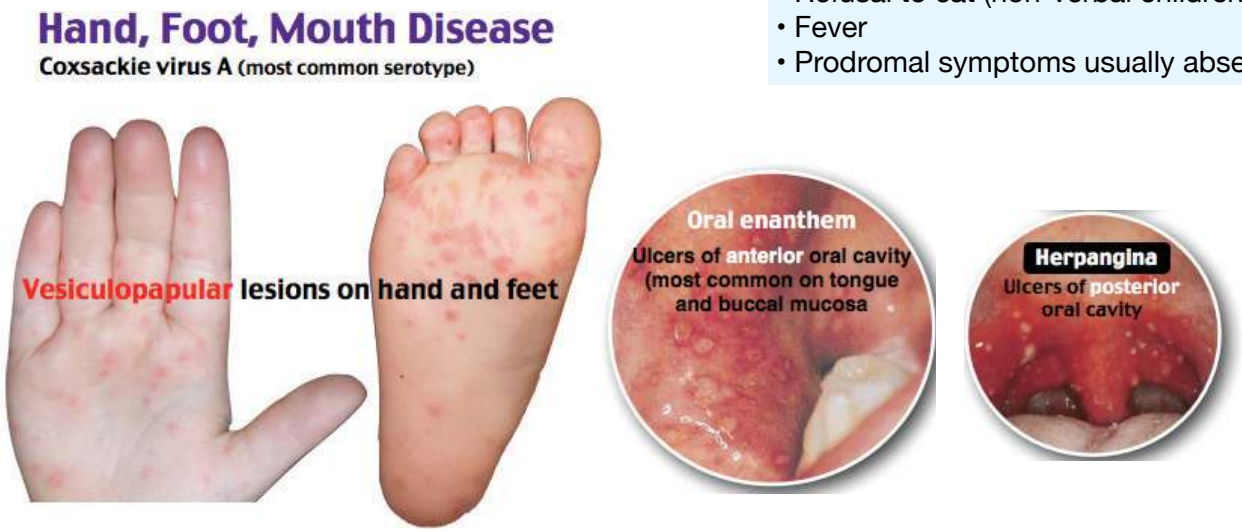
Patient
• Child younger than five years of age

Presentation
• Decreased appetite and fever

Physical Exam
• **Oral exanthem** plus a **macular**, **maculopapular**, or **vesicular** rash on the **hands and feet**

Management
• Supportive care

**Clinical**
• Mouth or throat pain (verbal children)
• Refusal to eat (non-verbal children)
• Fever
• Prodromal symptoms usually absent




Notes


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### D. Measles (Rubeola)

Patient
• **Unvaccinated** young child

Presentation
• High **fever**, **cough**, **conjunctivitis**, **coryza**
• **Maculopapular rash** starting on the **head** and spreading towards the **feet**

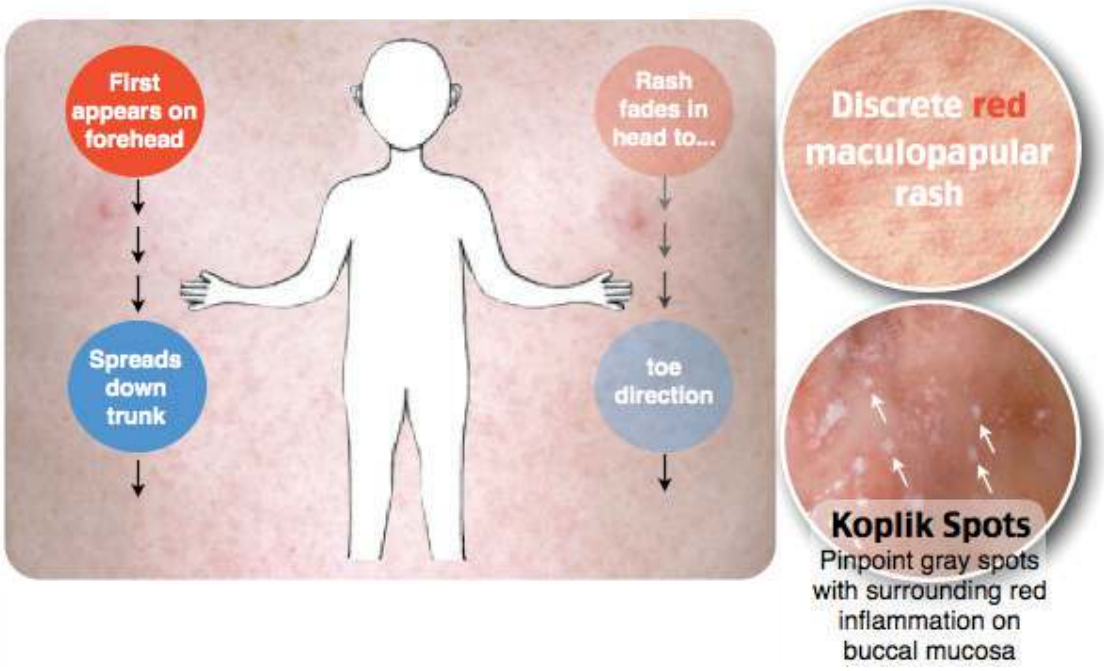
Physical Exam
• **Red spots** with **blue-white** center on **buccal mucosa** (**Koplik spots**)

Diagnostic Studies
• Diagnosis is made **clinically**

Management
• Supportive care

**Clinical**
• High fever
• **C**ough, **C**oryza, **C**onjunctivitis (The **3 Cs**)
• Rash
• Koplik spots

**Complications**
• Diarrhea (most common)
• Pneumonia (most common cause of mortality)
• Acute disseminated encephalomyelitis (ADEM)
• Subacute sclerosing panencephalitis (SSPE)




Notes


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CHAPTER 9

# Endocrinology

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CHAPTER 9

# Endocrinology


414 **ADRENAL INSUFFICIENCY**

415 **CUSHING DISEASE**

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431 **HYPOTHYROIDISM**

435 **NONKETOTIC HYPERGLYCEMIA**

437 **PALPITATIONS**

438 **THYROIDITIS**

440 **TREMORS**



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# I. Adrenal Insufficiency

### A. Primary Adrenal Insufficiency (Addison Disease)

Pathophysiology
• **Most common** cause is **autoimmune**

Presentation
• Adrenal crisis with abdominal pain, nausea, vomiting, diarrhea, fever, and confusion

Physical Exam
• **Hyperpigmentation** of the skin and mucous membranes
• Hypotension

Diagnostic Studies
• **Hyponatremia**, **hyperkalemia**, **hypoglycemia**

Management
• **Hydrocortisone**




**Clinical**
• Hypotension/shock
• Vomiting, diarrhea
• Fever
• Abdominal pain
• Hyponatremia, hypoglycemia, hyperkalemia

**Management**
• Hydrocortisone
• Supportive

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# II. Cushing Disease

### A. Cushing Syndrome

Pathophysiology
• **Most commonly** caused by **exogenous steroids** or **hypercortisolism** from **ACTH-secreting pituitary tumor**

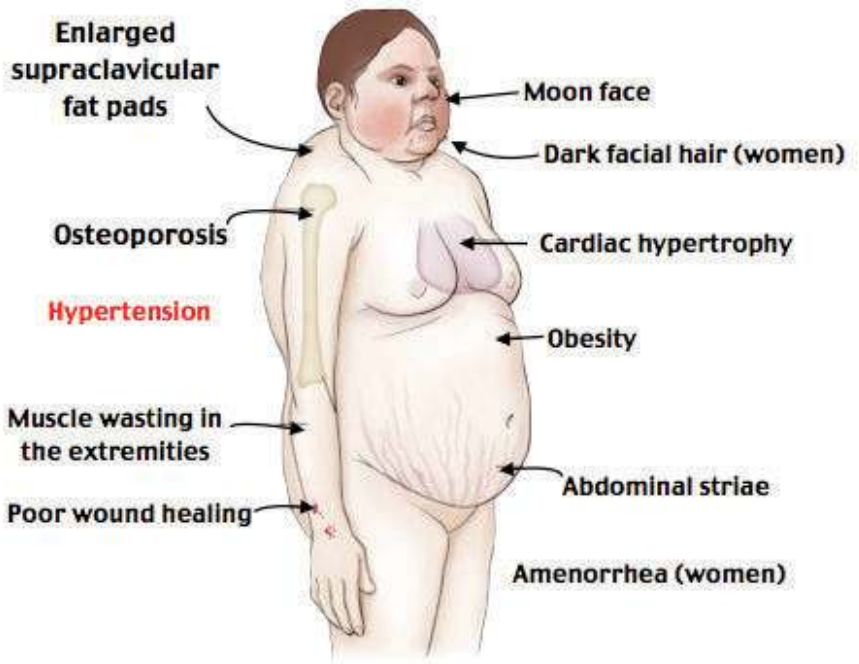
Presentation
• Amenorrhea, **central obesity**, depressive symptoms, easy bruising

Physical Exam
• **Purple striae**, **"moon facies"** (facial adiposity), **"buffalo hump"** (increased adipose tissue in the neck and upper back), and **hypertension**

**Cushing Syndrome** – Due to **excess cortisol-like medication** (prednisone) or **tumor** that produces or results in production of **excessive cortisol** **[Cases due to a pituitary adenoma = Cushing disease]**

Diagnostic Studies
• **24-hour urine cortisol**
• Serum ACTH

Comments
• If caused by a pituitary tumor, the condition is termed **Cushing disease**




Notes


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# III. Diabetes Insipidus

## A. Diabetes Insipidus (DI)

Presentation
- **Polyuria**, **polydipsia**

Diagnostic Studies
- **Increase** in plasma osmolality and **decrease** in urine osmolality

### 1. Central DI

Pathophysiology
- **Most commonly** caused by **decreased** **ADH production**

Diagnostic Studies
- Water deprivation test: **> 50% increase in urine osmolality**

Management
- Intranasal **DDAVP**

### 2. Nephrogenic DI

Pathophysiology
- Caused by **renal unresponsiveness** to ADH

Patient
- **Lithium** use

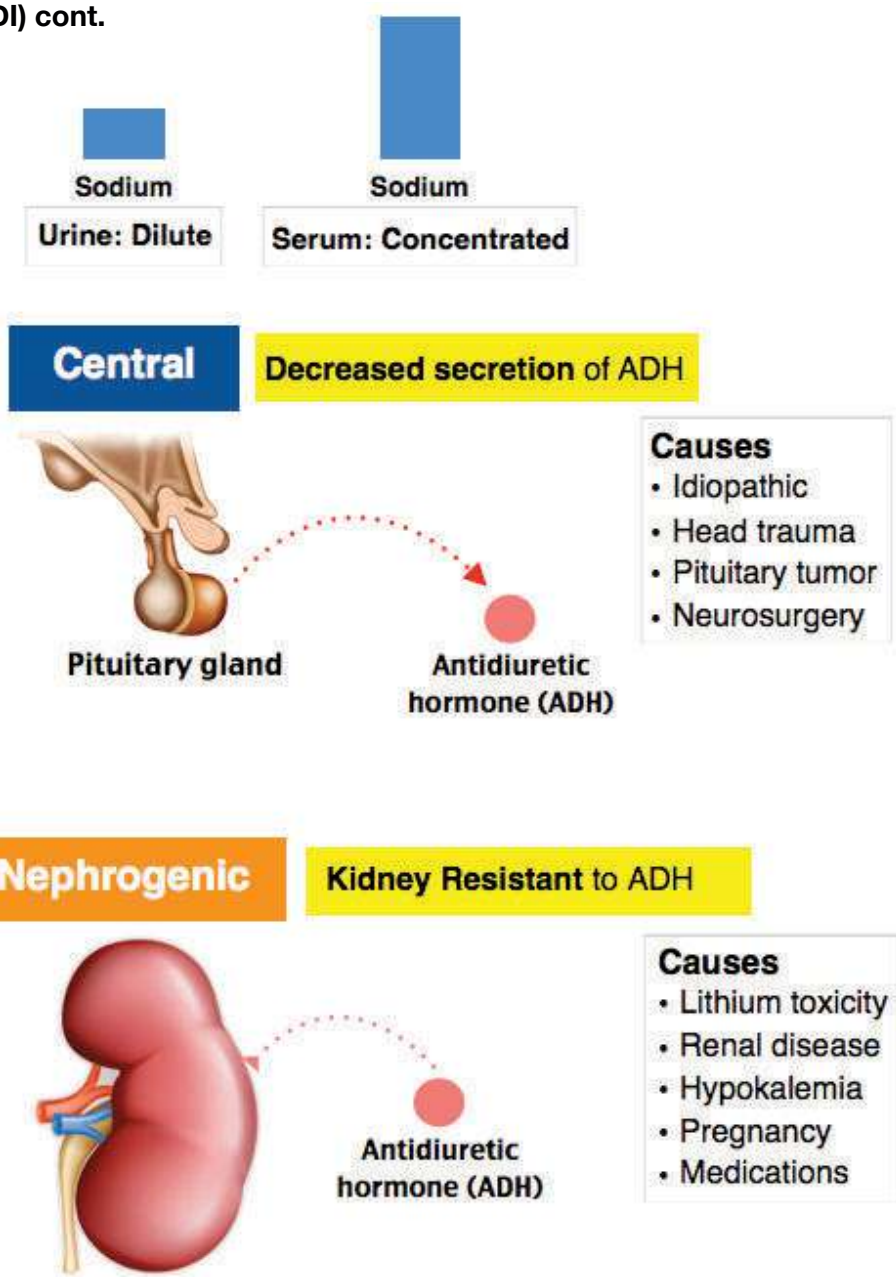
Diagnostic Studies
- Water deprivation test: **no change** in **urine osmolality**

Management
- HCTZ, amiloride, indomethacin

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**Diabetes Insipidus (DI) cont.**




Notes


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# IV. Diabetes Mellitus

## A. Diabetes Mellitus Type 1

Pathophysiology
• **Most commonly** caused by **autoimmune destruction** of **pancreatic beta cells**

Patient
• Child

Presentation
• **Polydipsia**, **polyphagia**, **polyuria**, **weight loss**

Diagnostic Studies
• **ADA Diagnostic Criteria**
• Symptoms plus one of the following:
  • **Random** plasma glucose of **≥ 200 mg/dL**
  • **Fasting** plasma glucose of **≥ 126 mg/dL** on **two** separate occasions
  • Glycated hemoglobin (A1C) of **≥ 6.5%** (adults only)
  • Plasma glucose of **> 200 mg/dL two hours** after a **75 g glucose load** during an oral glucose tolerance test

Management
• **Insulin**

**Clinical presentation**
• Polyphagia, polydipsia, polyuria
• Typically presents in the first two decades of life
• Can present in adulthood

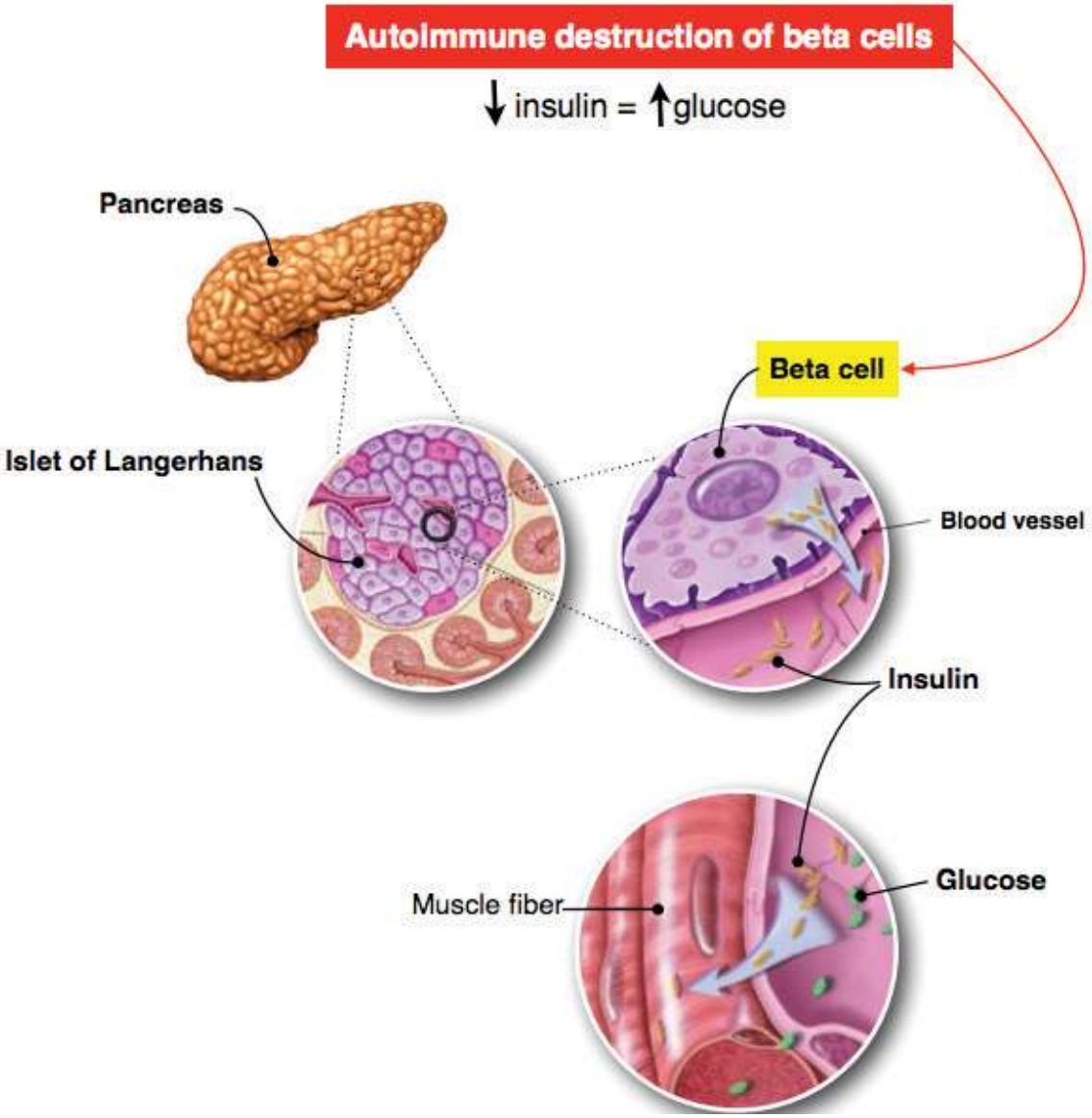
**Complications**
*Acute*
• DKA

*Chronic*
• Nephropathy
• Retinopathy
• Neuropathy
• Cardiovascular disease
• Skin manifestations (acanthosis nigricans, necrobiosis lipoidica)

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**Diabetes Mellitus Type 1 cont.**




Notes


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**B. Diabetes Mellitus Type 2**

Pathophysiology
- **Most commonly** caused by **insulin resistance**

Patient
- Middle-aged, **obese**

Presentation
- **Polydipsia**, **polyphagia**, **polyuria**

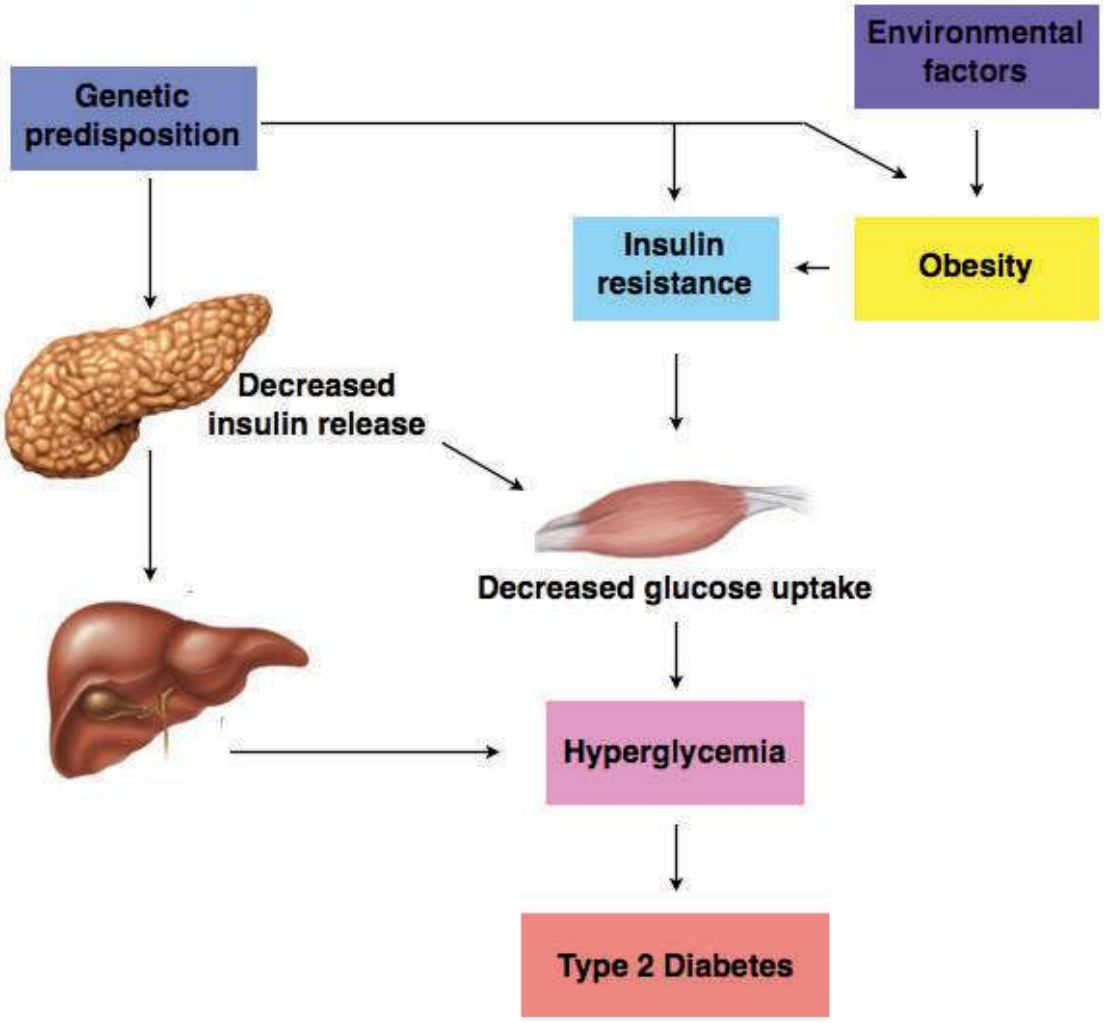
Diagnostic Studies
- **ADA Diagnostic Criteria**
- Symptoms plus one of the following:
  - **Random** plasma glucose of **≥ 200 mg/dL**
  - **Fasting** plasma glucose of **≥ 126 mg/dL** on **two** separate occasions
  - Glycated hemoglobin (A1C) of **≥ 6.5%**
  - Plasma glucose of **> 200 mg/dL two hours** after a **75 g glucose load** during an oral glucose tolerance test

Management
- Lifestyle modifications, then medication (**metformin** is first line)

Notes


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**Diabetes Mellitus Type 2 cont.**




Notes


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### C. Diabetes Type 2 Medication Classes

| Class | Common Drugs | Mechanism | Hypoglycemia |
|---|---|---|---|
| **Biguanides** | Metformin | Decreases hepatic gluconeogenesis | No |
| **Sulfonylureas** | Glyburide, Glipizide | Increases insulin secretion | Yes |
| **Thiazolidinediones** | Pioglitazone, Rosiglitazone | Increases insulin sensitivity in muscle and fat | No |
| **Meglitinides** | Repaglinide, Nateglinide | Increases insulin secretion | Yes |
| **α-glucosidase inhibitors** | Acarbose, Miglitol | Decreases intestinal absorption of carbohydrates | No |
| **DPP-4 inhibitors** | Sitagliptin, Saxagliptin | Increases insulin secretion | No |
| **Glucagon-like peptide-1 agonists** | Exenatide, Lixisenatide, Liraglutide, Dulaglutide | Increases insulin secretion and decreases glucagon secretion | No |
| **Insulin** | Regular, Lispro, Aspart, NPH, Glargine | Increases glucose uptake | Yes |
| **Gliflozins** Sodium/glucose cotransporter 2 (SGLT2) inhibitors | Dapagliflozin, Canagliflozin, Empagliflozin | Reduces reabsorption of filtered glucose in the proximal renal tubules resulting in increased urinary excretion of glucose and reducing plasma glucose | No |

Notes


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### D. American Diabetes Association Diagnostic Criteria for Diabetes Mellitus

- Symptoms of diabetes plus a random plasma glucose concentration of ≥ 200 mg/dL
- A fasting plasma glucose of ≥ 126 mg/dL on two separate occasions
- A plasma glucose of > 200 mg/dL two hours after a 75-g glucose load during an oral glucose tolerance test
- HgbA1C ≥ 6.5% (Adults only)

Notes


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# V. Diabetic Ketoacidosis

### A. Diabetic Ketoacidosis

Patient
• Diabetic with recent **I**nfection, **I**schemia (cardiac, mesenteric), **I**nfarction, **I**gnorance (poor control), **I**ntoxication (**FIVE Is**)

Presentation
• Complaining of abdominal pain, vomiting, and fatigue

Physical Exam
• Fruity-smelling breath, dehydration, and AMS

Diagnostic Studies
• **Hyperglycemia**, ketonemia, and an **anion gap metabolic acidosis**

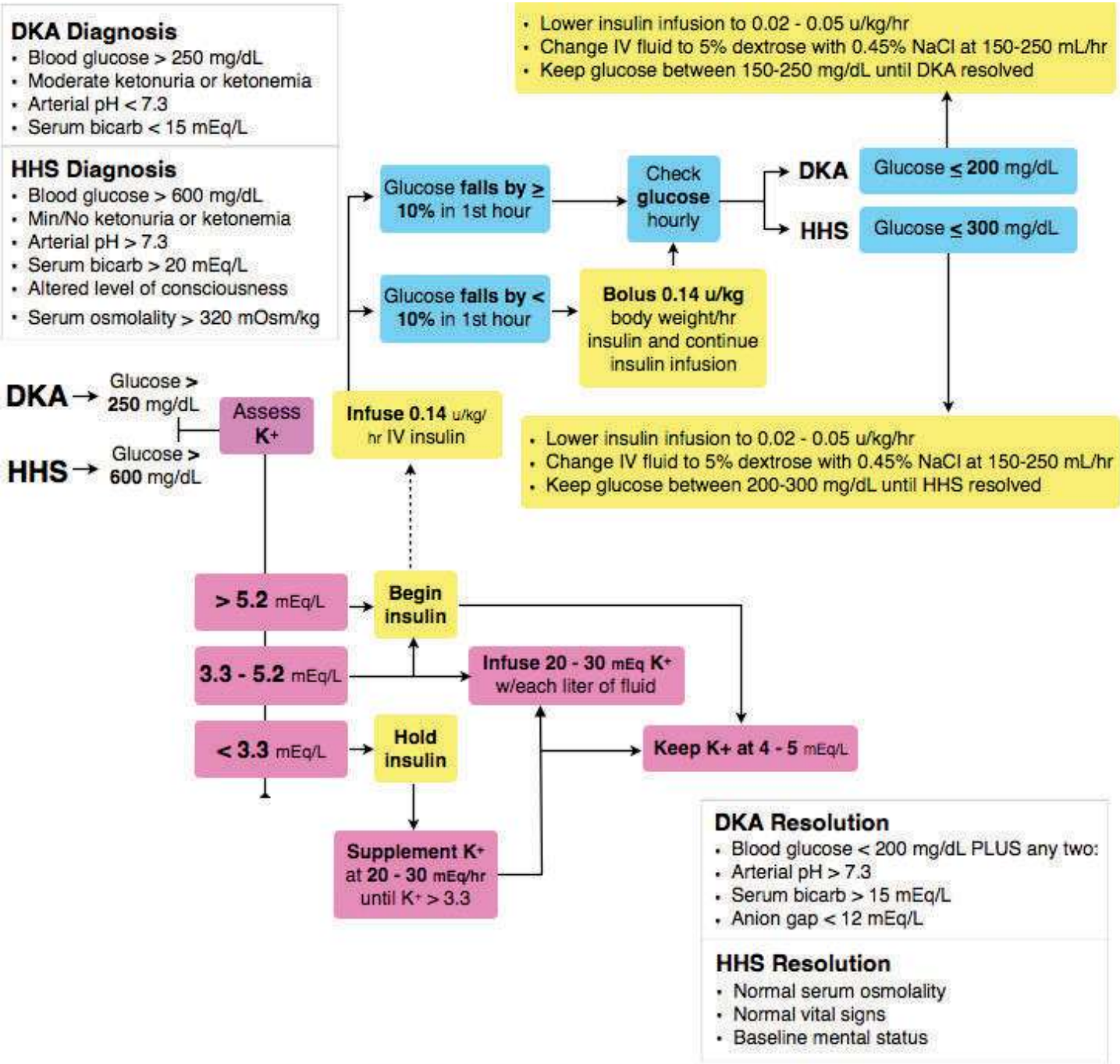
Management
• **IV fluids** and insulin infusion
• **Potassium** supplementation

Comments
• Corrected sodium: add 1.6 mEq/L for each 100 mg/dL in serum glucose
• **HHS** = hyperglycemic hyperosmolar syndrome

Notes


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## Diabetic Ketoacidosis cont.




Adapted from Van Ness-Otunnu R, Hack JB. Hyperglycemic crisis. *J Emerg Med*. 2013;45(5):797-805.


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# VI. Heat/Cold Intolerance

**A. Heat/Cold Intolerance**: See Hyper/Hypothyroidism

Notes


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# VII. Hyperparathyroidism

### A. Hyperparathyroidism

Pathophysiology
• **Most commonly** caused by an **adenoma** with unregulated **overproduction of PTH**

Diagnostic Studies
• **High** PTH, **high** calcium, **low** phosphorus




Notes


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# VIII. Hyperthyroidism

### A. Hyperthyroidism

Pathophysiology
• **Most commonly** caused by **Graves disease** (autoimmune, against TSH receptors)

Presentation
• **Heat intolerance**, **palpitations**, weight loss, and anxiety

Physical Exam
• **Tachycardia**, hyperreflexia, **goiter**, **exophthalmos**, pretibial edema

Diagnostic Studies
• **Low** TSH and **high** free T4

Management
• **Methimazole** or PTU

Comments
• **P**TU if **P**regnant

Notes


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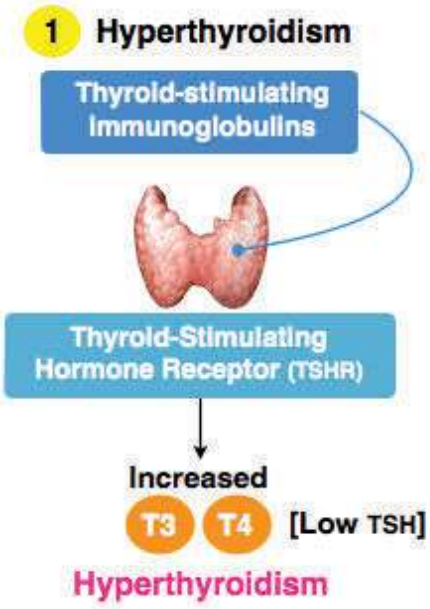
### B. Clinical Manifestations of Hyperthyroidism

| | |
|---|---|
| **Skin** | • Warm<br>• Sweating<br>• Onycholysis<br>• Hyperpigmentation<br>• Pruritus and hives<br>• Vitiligo and alopecia areata<br>• Thinning of hair |
| **Eyes** | • Stare<br>• Lid lag<br>• Proptosis |
| **Cardiovascular** | • Increased cardiac output<br>• Tachycardia<br>• Widened pulse pressure<br>• Systolic hypertension<br>• Atrial fibrillation |
| **Respiratory** | • Dyspnea<br>• Increased pulmonary arterial systolic pressure<br>• Respiratory muscle weakness |
| **Gastrointestinal** | • Weight loss<br>• Increased gut motility |
| **Hematologic** | • Normochromic, normocytic anemia |
| **Genitourinary** | • Urinary frequency<br>• Nocturia |
| **Bone** | • Bone resorption<br>• Osteoporosis |
| **Neuropsychiatric** | • Psychosis<br>• Agitation<br>• Depression |
| **Neurologic** | • Tremor |

Notes

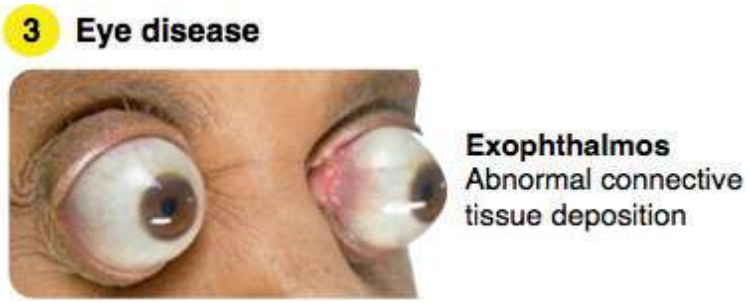

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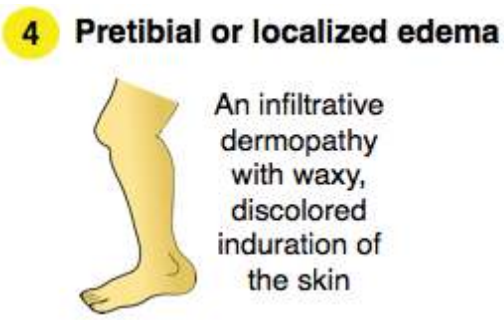
**B. Graves Disease**










Notes


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# IX. Hypothyroidism

**A. Hypothyroidism**

Pathophysiology
• **Most commonly** caused by **Hashimoto thyroiditis**

Presentation
• Generalized weakness, **fatigue**, facial swelling, constipation, **cold intolerance**, **weight gain**

Physical Exam
• **Periorbital edema**, **dry skin**, and **coarse**, **brittle hair**

Diagnostic Studies
• **High** TSH and **low** free T4, **antithyroid peroxidase**, and **antithyroglobulin** autoantibodies

Management
• Levothyroxine

Comments
• Monitor TSH serially; takes six weeks to see the effect
• **Hashimoto** thyroiditis is a risk factor for non-Hodgkin lymphoma




**1 Slowing of metabolic processes**
- Fatigue and weakness
- Cold intolerance
- Dyspnea on exertion
- Weight gain
- Cognitive dysfunction
- Constipation
- Bradycardia
- Delayed deep tendon reflexes
- Slow movement and speech

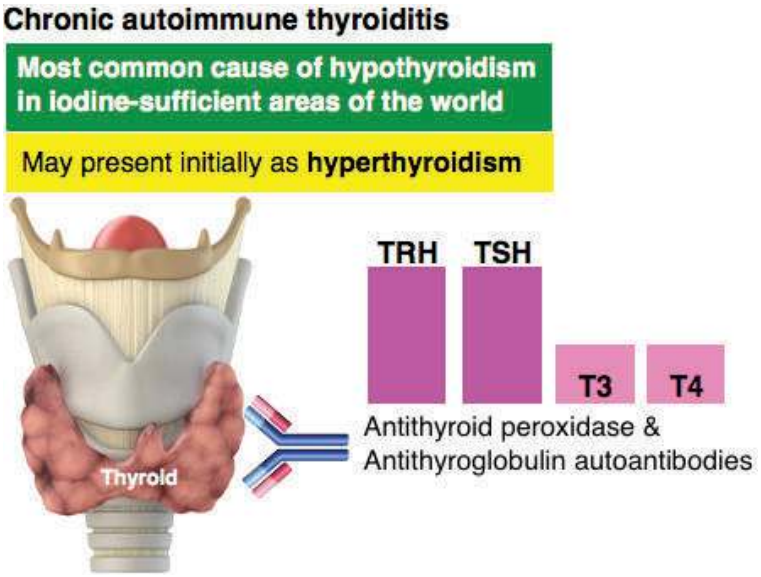
**2 Accumulation of matrix glycosaminoglycans**
- Dry skin
- Hoarseness
- Edema
- Coarse skin
- Puffy facies and loss of eyebrows
- Periorbital edema
- Enlargement of tongue

**Treatment**
• Synthetic thyroxine (T4, levothyroxine)

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### B. Hashimoto Thyroiditis



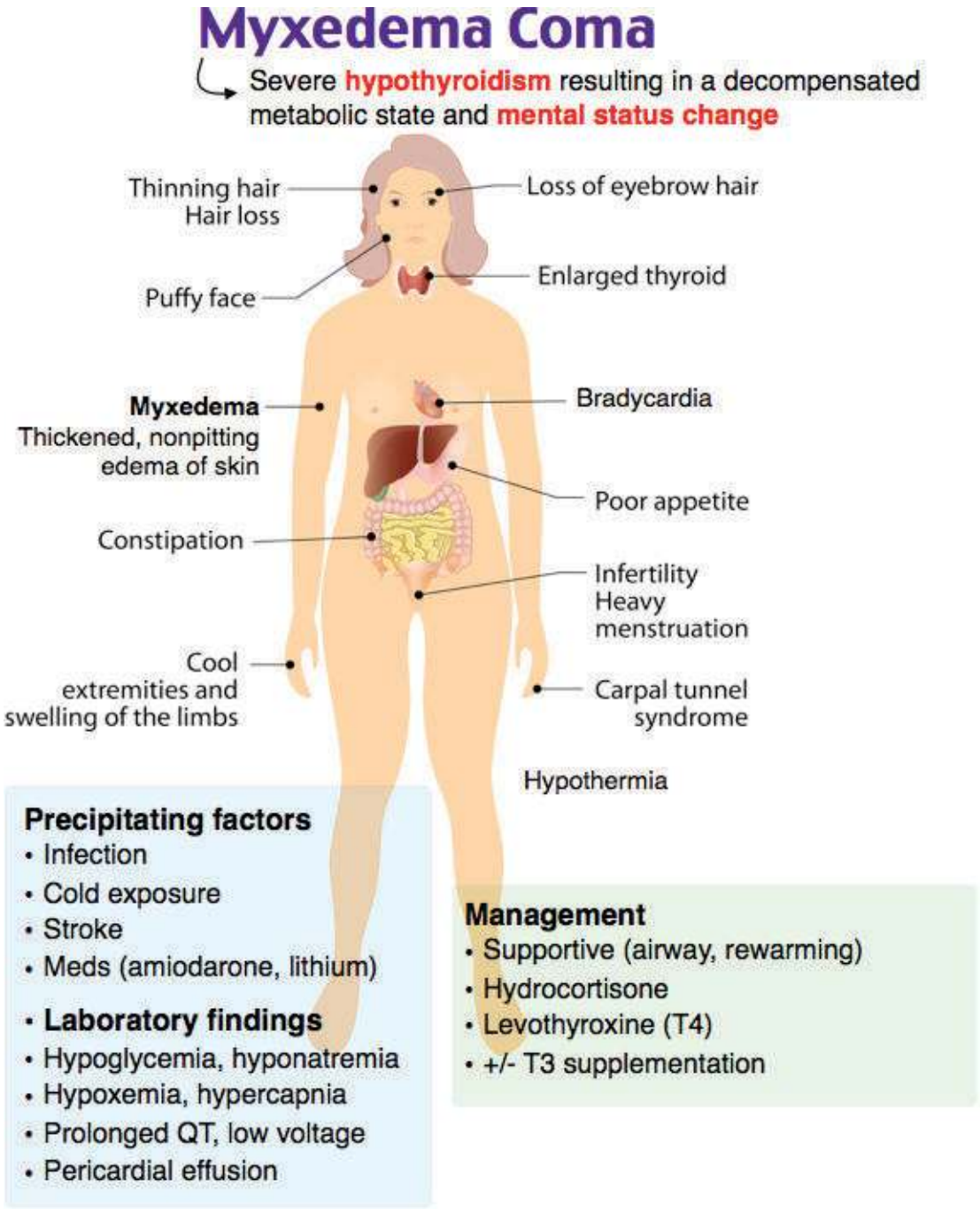

**Clinical**
- Fatigue
- Weight gain
- Cold intolerance
- Dry skin
- Constipation
- Menstrual irregularities
- Depression

**Treatment**
- Levothyroxine

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**C. Myxedema Coma**




Notes


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### D. Thyroid Function Test

| Thyroid Function Test Interpretation | | | |
|---|---|---|---|
| TSH | Free T4 | Free T3 | Condition |
| Normal | Normal | Normal | • None |
| Low | High | High | • **Hyperthyroidism** |
| Low | Normal | Normal | • Subclinical hyperthyroidism |
| Low | Normal | High | • T3 toxicosis |
| Low | High | Normal | • Thyroiditis<br>• T4 ingestion<br>• Hyperthyroidism in the elderly or with comorbid illness |
| Low | Low | Low | • Euthyroid sick syndrome<br>• Central hypothyroidism |
| High | Normal | Normal | • Subclinical hypothyroidism<br>• Recovery from euthyroid sick syndrome |
| High | Low | Low | • **Primary hypothyroidism** |
| High | High | High | • TSH producing pituitary adenoma |

Notes


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# X. Nonketotic Hyperglycemia

### A. Hyperosmolar Hyperglycemic State

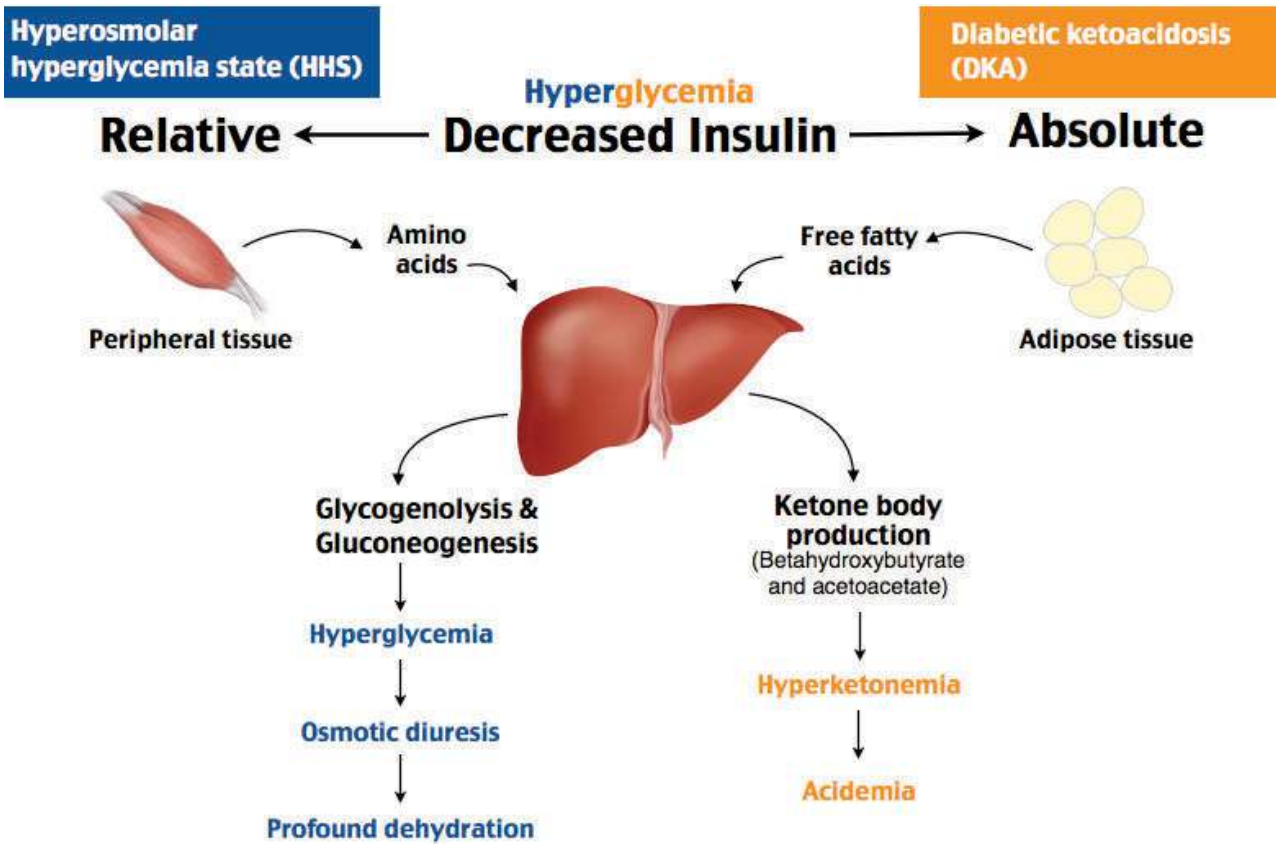
Patient
• History of Type 2 DM

Presentation
• Complaining of AMS

Physical Exam
• Profound dehydration

Diagnostic Studies
• Labs will show **glucose > 600** and **negative ketones**

Management
• **IVF**




Notes


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| Lab Finding | Hyperglycemic Hyperosmolar State (HHS) | Diabetic ketoacidosis (DKA) |
|---|---|---|
| Plasma glucose | > 600 | > 250 |
| Serum bicarbonate | > 15 | < 18 |
| Urine ketones | -/+ | +++ |
| Anion gap | < 12 | > 10 |

Notes


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# XI. Palpitations

**Definition**: A noticeably rapid, strong, or irregular heartbeat

| Causes of Palpitations | |
|---|---|
| **Cardiac** | • Any dysrhythmia<br>• Cardiac and Extracardiac shunts<br>• Valvular heart disease<br>• Pacemaker<br>• Atrial myxoma<br>• Cardiomyopathy |
| **Psychiatric disease** | • Panic attack and disorder<br>• Generalized anxiety disorder<br>• Somatization<br>• Depression |
| **Medications** | • Sympathomimetic agents<br>• Vasodilators<br>• Anticholinergic drugs<br>• Beta blocker withdrawal |
| **Habits** | • Cocaine<br>• Amphetamines<br>• Caffeine<br>• Nicotine |
| **Metabolic disorders** | • Hypoglycemia<br>• Thyrotoxicosis<br>• Pheochromocytoma<br>• Mastocytosis<br>• Scombroid food poisoning |
| **High output states** | • Anemia<br>• Pregnancy<br>• Paget's disease<br>• Fever |
| **Catecholamine excess** | • Stress<br>• Exercise |

Notes


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# XII. Thyroiditis

### A. Thyroid Storm

Pathophysiology
• Precipitated by an **acute event** (infection, trauma, etc.)

Patient
• History of hyperthyroidism

Presentation
• Tachycardia, hyperpyrexia, agitation, anxiety

Physical Exam
• **Goiter**, **lid lag**, hand tremor, and warm, moist skin

Diagnostic Studies
• **Low** TSH and **high** free T4 or T3

Management
1. Beta-blocker (propranolol)
2. Thionamide (propylthiouracil or methimazole)
3. Iodine solution
4. Glucocorticoids

Notes


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**Thyroid Storm cont.**

## Hyperthyroid state + Acute event

### Risk Factors (Acute event)

- Surgery
- Trauma
- Infection
- Acute iodine load
- Parturition

### Clinical



- Tachycardia (HR > 140)
- Heart failure
- Hypotension
- Dysrhythmia (Atrial fibrillation)



- Hyperpyrexia (104°F-106°F)
- Agitation
- Psychosis, stupor
- Coma

### Labs




### Treatment

1. Beta-blockers (Propranolol)
2. Thionamides (Propylthiouracil)
3. Iodine (Given after thionamide)
4. Glucocorticoid (Hydrocortisone)
5. Bile acid sequestrants (Cholestyramine)

Notes


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# XIII. Tremors

| Tremors | | | |
|---|---|---|---|
| **Type** | **Description** | **Physical Exam** | **Examples** |
| **Resting** | Tremor at rest | • Observe at rest<br>• Frequency 4-6 Hz<br>• Associated with hypokinesia or rigidity (parkinsonism) | Parkinson's disease<br>Parkinsonism tremor (medications) |
| **Postural** | Tremor with sustained posture of an extremity | • Ask patient to extend arms and hold | Essential tremor<br>Increased physiologic tremor<br>Wilson's disease |
| **Intention** | Tremor during movement that increases as a target is approached | • No tremor at rest<br>• May be associated with cerebellar signs | Cerebellar disease<br>Multiple sclerosis<br>Chronic alcohol use |

Notes


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CHAPTER 10

# Obstetrics/ Gynecology

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CHAPTER 10

# Obstetrics/Gynecology


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458 **OVARIAN CYSTS**

461 **PELVIC INFLAMMATORY DISEASE**

464 **PELVIC PAIN/DYSMENORRHEA**

465 **PLACENTA ABRUPTION**

466 **PLACENTA PREVIA**

467 **PRELABOR RUPTURE OF MEMBRANES**

468 **SPONTANEOUS ABORTION**

470 **VAGINAL DISCHARGE/VAGINITIS**



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# I. Amenorrhea

### A. Amenorrhea

Primary
• Absence of menses by **age 16** with secondary sexual development present, or the absence of secondary sexual characteristics by age 14
  • Turner Syndrome: XO karyotype, webbed neck, broad chest
  • Androgen insensitivity: breast development only
  • Imperforate hymen: observed on speculum exam
  • Müllerian agenesis: secondary sex characteristics, no uterus

Secondary
• Previously had menses, amenorrhea for six months
  • **Most common** cause is **pregnancy**
  • Also caused by weight changes, hypothyroidism, prolactinoma

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### B. Causes of Amenorrhea

| Causes of Primary Amenorrhea | |
|---|---|
| **Hypothalamic/Pituitary** | |
| Hypothalamic dysfunction | Organic illness, vigorous exercise, stressful life events, anorexia nervosa |
| Genetic gonadotropin deficiency | Kallmann syndrome |
| Hypothalamic lesions | Craniopharyngioma |
| Pituitary tumors | Prolactinoma |
| Endocrinopathies | Cushing syndrome, hypothyroidism |
| Hyperandrogenism | From adrenal, ovarian, or exogenous sources |
| **Ovarian** | Turner syndrome, ovarian failure due to autoimmunity, ovarian steroidogenic enzyme deficiencies |
| **Uterine** | Congenital absence or malformation of the uterus, imperforate hymen |
| **Pseudohermaphroditism** | Absent uterus and intra-abdominal or cryptorchid testes due to either an enzymatic defect in testosterone synthesis or to complete androgen resistance |
| **Causes of Secondary Amenorrhea** | |
| **Hypothalamic/Pituitary** | Emotional stress, illness, dieting, medications, anorexia, rigorous exercise |
| | Cushing syndrome, hypothyroidism |
| | Pituitary tumor (prolactinoma), pituitary infarction (Sheehan syndrome) |
| | Granulomatous disease (sarcoidosis) |
| **Ovarian** | Polycystic ovarian disease |
| | Premature ovarian failure |
| | Hyperandrogenism |
| **Uterine** | Pregnancy, Asherman syndrome, cervical scarring |

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# II. Abnormal Uterine Bleeding

## A. Abnormal Uterine Bleeding (formerly Dysfunctional Uterine Bleeding)

Presentation
• Abnormal menstrual bleeding with regards to **quantity**, **duration**, or **schedule** in a **nonpregnant** woman of reproductive age

Pathophysiology
• **Structural** (adenomyosis, uterine fibroids, malignancy, and polyps) and **nonstructural** causes
• If AUB due to anovulatory state:
  • **Increased** estrogen and **decreased** progesterone leads to endometrial hyperplasia and unpredictable bleeding
• Ovulatory state:
  •Predictable bleeding

Diagnostic Studies
• Diagnosis of exclusion if cause is nonstructural

Management
• Combination OCPs
• Unstable bleeding: IV estrogen
• Surgery if structural cause is identified

Comments
• Most common cause of abnormal vaginal bleeding in women of reproductive age

Notes


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## B. Causes of Abnormal Uterine Bleeding

**(FIGO Classification for nongravid women of reproductive age)**

**Abnormal uterine bleeding (AUB)**
**Heavy menstrual bleeding (AUB/HMB)**
**Intermenstrual bleeding (AUB/IMB)**

### PALM: Structural Causes

**P**olyp (AUB-P)
**A**denomyosis (AUB-A)
**L**eiomyoma (AUB-L)
  **S**ubmucosal myoma (AUB-$L_{SM}$)
  **O**ther myoma (AUB-$L_O$)
**M**alignancy and hyperplasia (AUB-M)




### COEIN: Nonstructural Causes

**C**oagulopathy (AUB-C)
**O**vulatory dysfunction (AUB-O)
**E**ndometrial (AUB-E)
**I**atrogenic (AUB-I)
**N**ot yet classified (AUB-N)




Notes


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# III. Ectopic Pregnancy

## A. Ectopic Pregnancy

Pathophysiology
• **Most commonly** implanted in a **fallopian tube**

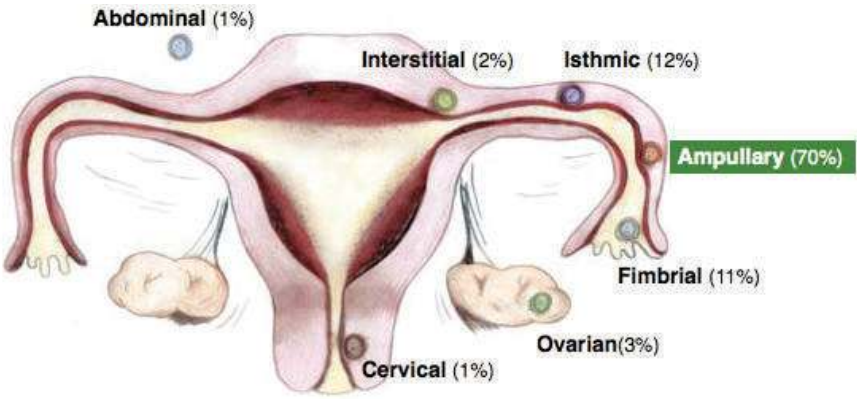
Patient
• History of **prior ectopic pregnancy**, PID, tubal surgery, IVF, IUD

Presentation
• Vaginal bleeding, abdominal pain, amenorrhea

Physical Exam
• Adnexal tenderness or **unexplained hypotension**

Diagnostic Studies
• Positive pregnancy test and **lower than expected serum beta-hCG levels**
• Ultrasound

Management
• Methotrexate or surgery




| Risk Factors for Ectopic Pregnancy |
|---|
| Previous ectopic pregnancy |
| Tubal surgery |
| Pelvic inflammatory disease |
| Tobacco use |
| Advanced maternal age |
| Prior spontaneous abortion |
| Medically induced abortion |
| History of infertility |
| Intrauterine device |

Notes


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# IV. Endometriosis

### A. Endometriosis

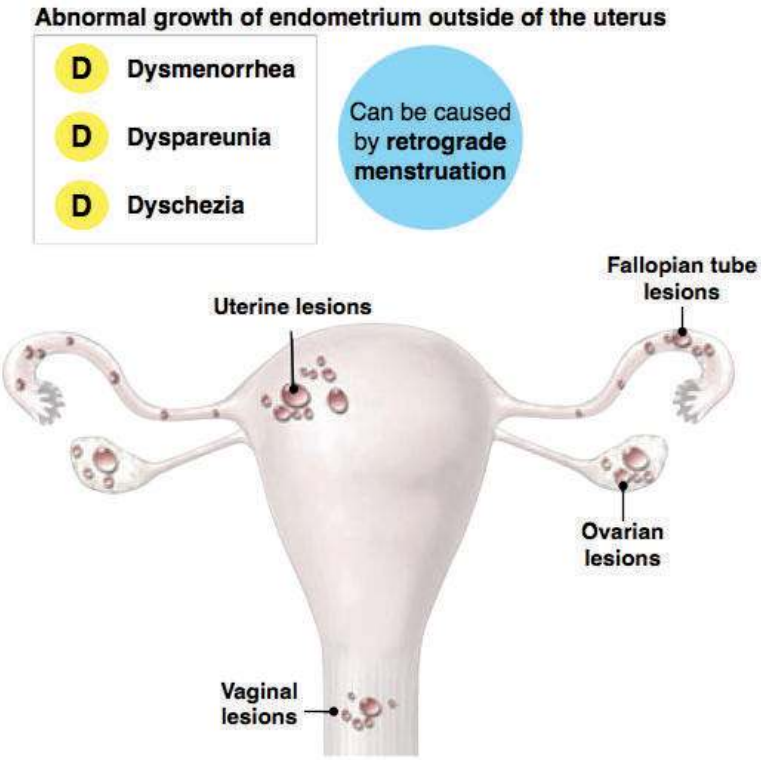
Pathophysiology
• **Most common** site of endometrial implants is the **ovaries**

Presentation
• Pre- or mid-cycle **dysmenorrhea**, **dyspareunia**, **dyschezia** (painful bowel movement)

Physical Exam
• Uterosacral nodularity or a **fixed** or **retroverted uterus**

Diagnostic Studies
• **Laparoscopy** is diagnostic

**Management**
• NSAIDs
• Hormonal contraceptives
• Gonadotoprin-releasing hormone (GnRH) agonists
• Aromatase inhibitors




Notes


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### B. Classification of Endometriosis

| Stage | Characterization | Example |
|---|---|---|
| **1: Minimal** | • Isolated implants<br>• No significant adhesions |  |
| **2: Mild** | • Superficial implants that are < 5 cm in aggregate and are scattered on the peritoneum and ovaries<br>• No significant adhesions are present |  |
| **3: Moderate** | • Exhibits multiple implants, both superficial and deeply invasive<br>• Peritubal and periovarian adhesions may be evident |  |
| **4: Severe** | • Characterized by multiple superficial and deep implants, including large ovarian endometriomas<br>• Filmy and dense adhesions are usually present |  |

Notes


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# V. Fetal Distress

### A. Components of FHR Evaluation

- Rate (normal = 110–160 bpm):
  - FHR < 110 bpm: bradycardia
  - FHR > 160 bpm: tachycardia

- Variability:
  - **Absent**: Indicates severe fetal distress
  - **Minimal**: < 6 bpm; indicates fetal hypoxia or the effects of opioids, magnesium, or sleep cycle
  - **Normal**: 6–25 bpm
  - **Marked**: > 25 bpm; may indicate fetal hypoxia; may occur before a **decrease** in variability
  - **Sinusoidal**: Points to serious fetal anemia; a pseudosinusoidal pattern may also occur during maternal meperidine use

- Accelerations: Onset of an **increase** in FHR > 15 beats above baseline to a peak in < 30 seconds. Reassuring because they indicate fetal ability to appropriately respond to the environment.

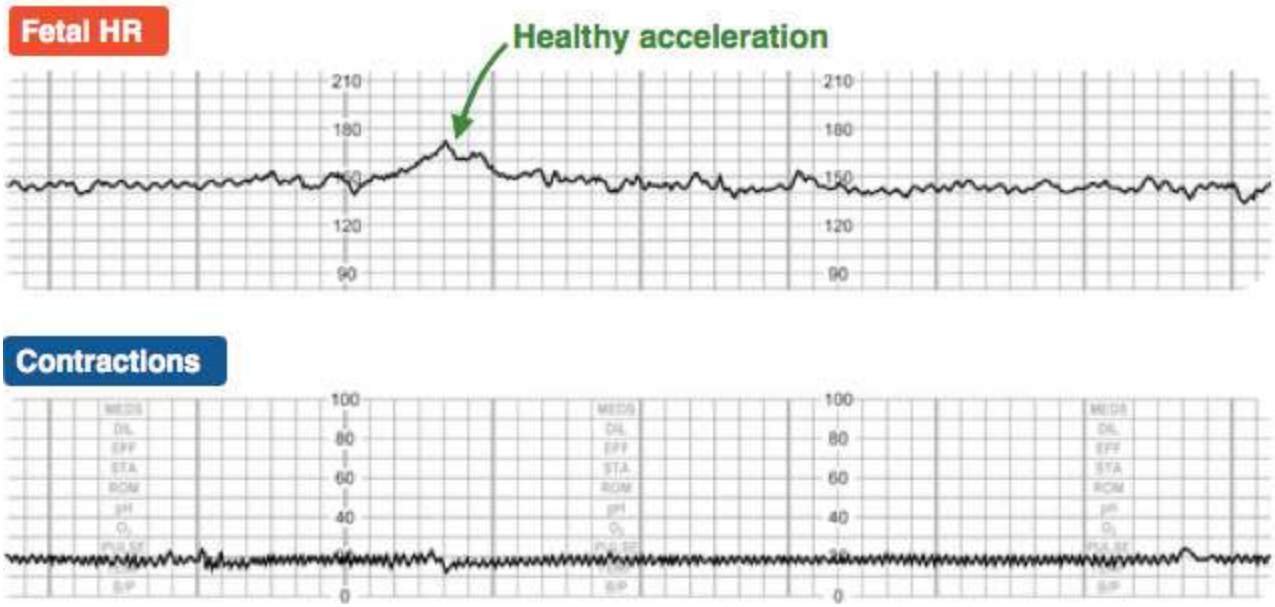
- Decelerations:
  - **Early**: Visually apparent, gradual (onset to nadir in > 30 seconds) **decrease** in FHR with a return to baseline that **mirrors the uterine contraction**
  - **Late**: Visually apparent, gradual (onset to nadir in > 30 seconds) **decrease** in FHR with return to baseline whose onset, nadir, and recovery occur after the beginning, peak, and end of uterine contraction, respectively
  - **Variable**: Abrupt (onset to nadir in < 30 seconds), visually apparent **decrease** in FHR 15 bpm below baseline lasting ≥ 15 seconds but < 2 minutes

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### B. Reassuring Pattern

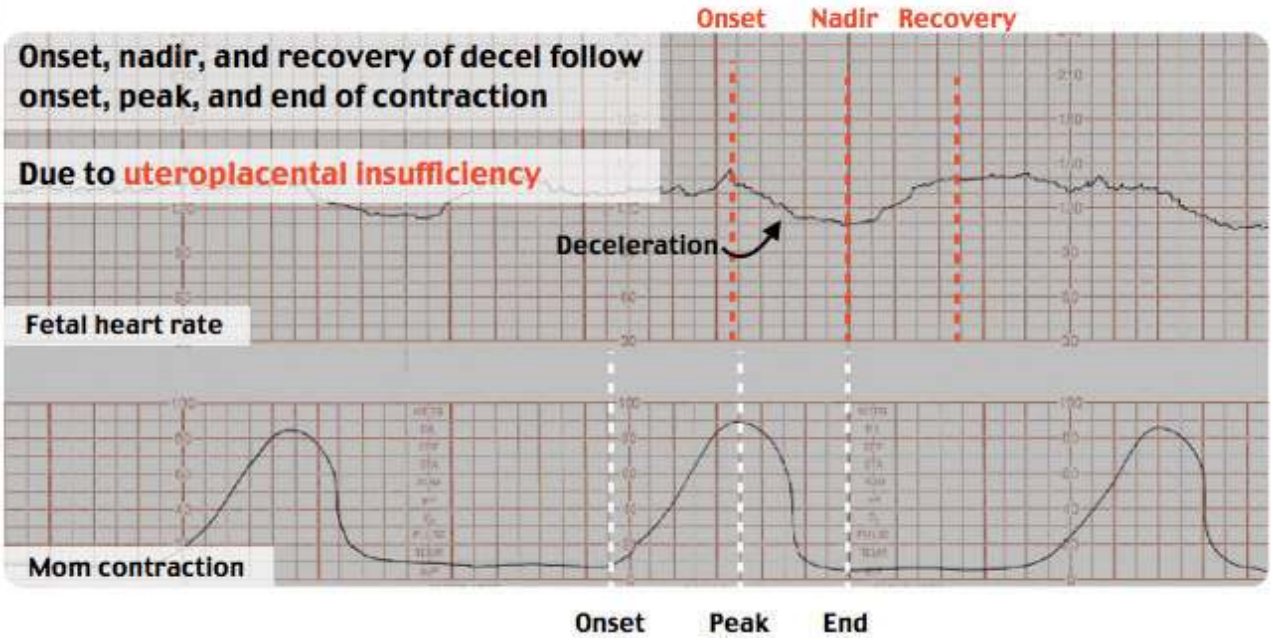
- Baseline fetal heart rate is 120-160
- Preserved beat-to-beat and long term variability
- Accelerations last ≥ 15 seconds above baseline
- Accelerations peak ≥ 15 beats per minute




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### C. Late Deceleration

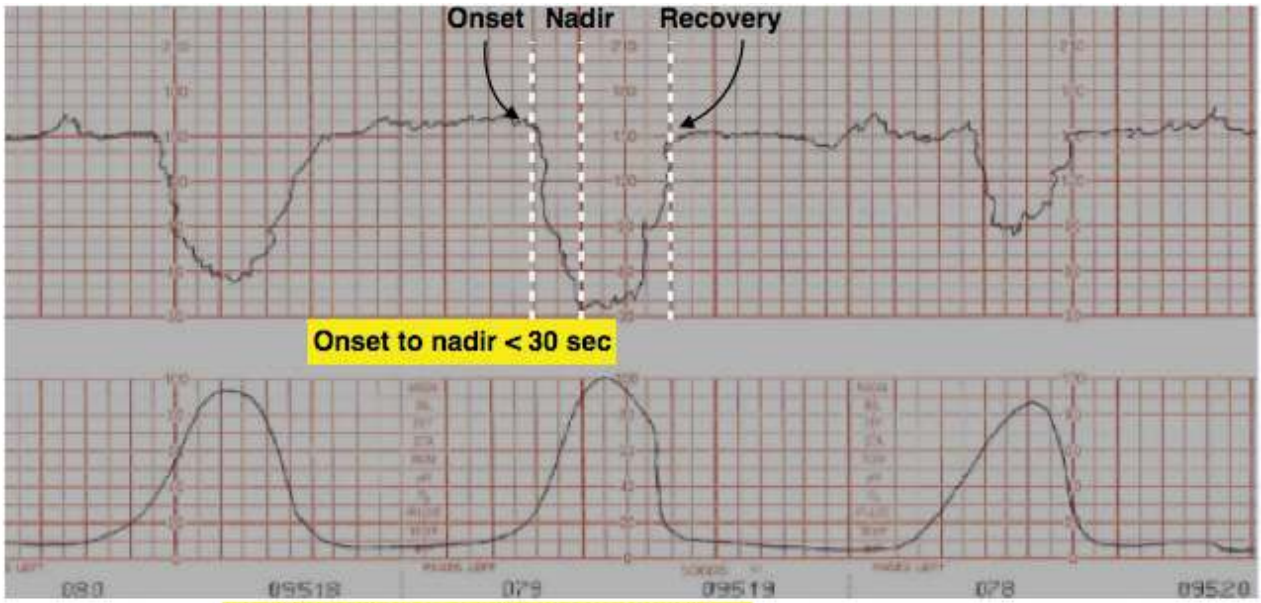



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### D. Variable Decelerations

• Onset to nadir < 30 seconds
• Drop of 15 bpm below baseline for > 15 seconds but < 2 minutes
• Relationship to contraction is variable




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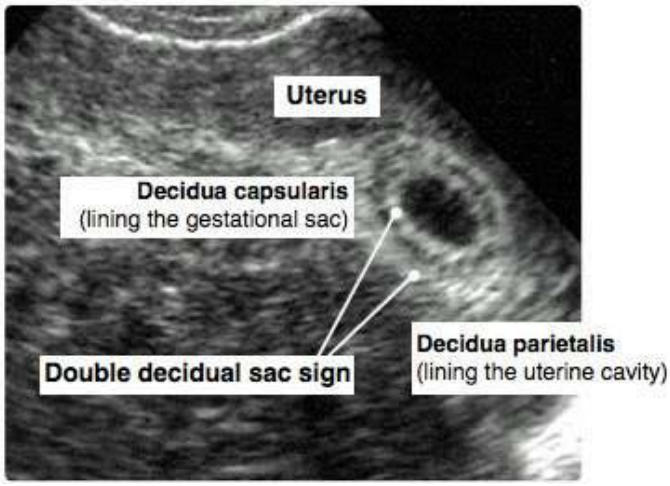
# VI. Intrauterine Pregnancy

### A. Intrauterine pregnancy

• Normal pregnancy, usually diagnosed by a positive pregnancy test and demonstration of a gestational sac and yolk sac in the uterus on ultrasound

### B. Double Decidual Sac Sign

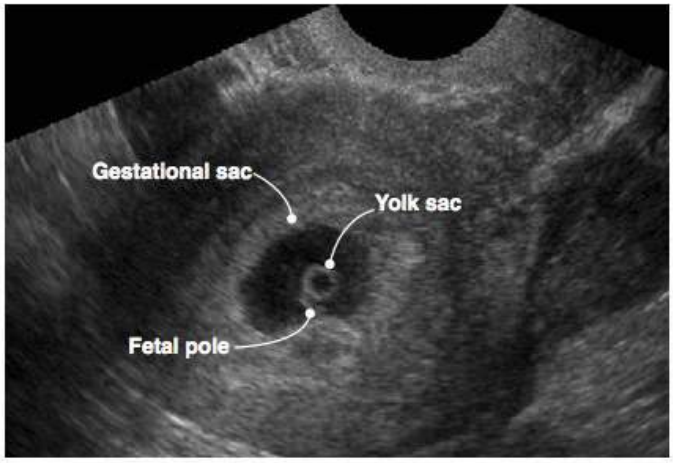
• Ultrasound finding that confirms early intrauterine pregnancy

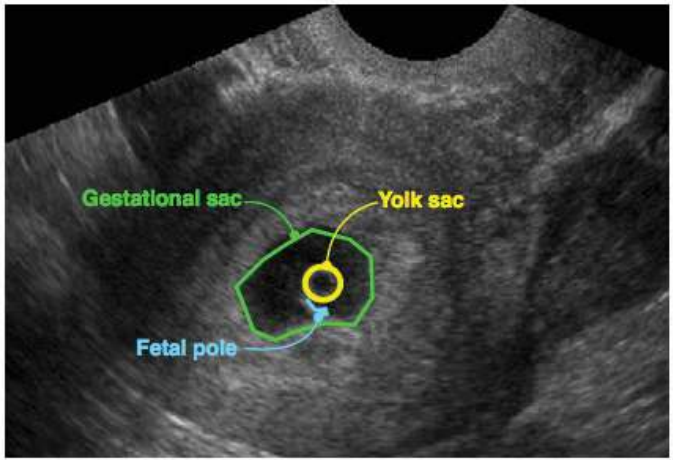



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## C. Structures of 1st Trimester Pregnancy




**Yolk sac**
Bright ring with anechoic center located inside the gestational sac at 5 weeks gestational age and persists to 11-12 weeks

**Embryo/fetus**
Seen by TVUS as thickening of yolk at 6 weeks gestational age

**Presence of cardiac activity**
Usually seen around the time fetal pole is present, further confirming viability

Notes


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# VII. Mastitis/Breast Abscess

### A. Mastitis

Pathophysiology
• **Most commonly** caused by ***Staphylococcus aureus***

Patient
• Breastfeeding mother

Presentation
• Breast **erythema**, **tenderness**, **fever**

Management
• **Warm compresses** (helps soften and drain blocked milk), cold compresses, **dicloxacillin**, amoxicillin
• **Continue breastfeeding** to avoid progression to abscess




**Clinical**
• Most commonly caused by *S. aureus*, coag-negative *Staph*, or *Strep. viridans*
• Colonized infant is asymptomatic
• Pain, erythema, tenderness, fever
• Must consider inflammatory breast cancer

**Management**
• Continue breastfeeding
• Cool compresses
• Anti-staphylococcal antibiotics [dicloxacillin, amoxicillin, cephalexin]

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## B. Breast Abscess

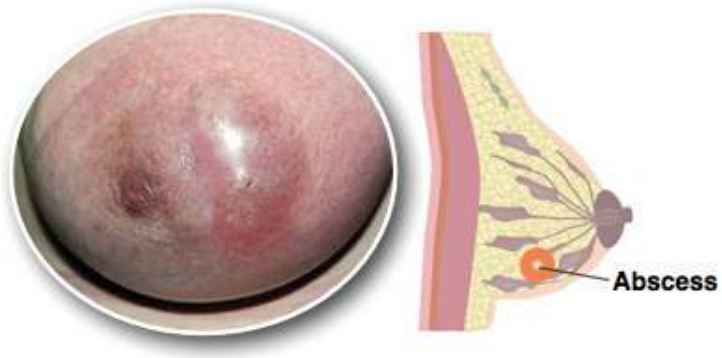
Pathophysiology
• **Most commonly** caused by ***Staphylococcus aureus***

Presentation
• Fever, malaise, painful breast lump

Physical Exam
• Fluctuant, tender, palpable mass

Management
• Antibiotics (**dicloxacillin**), continue breastfeeding, I&D or aspiration

Comments
• Usually a complication of **mastitis**




**Etiology**
• Primary abscess is usually complication of mastitis

**Microbiology**
• *Staphylococcus aureus* (most common)

**Clinical**
• Localized, painful inflammation of the breast
• Fever, malaise
• Fluctuant, tender, palpable mass

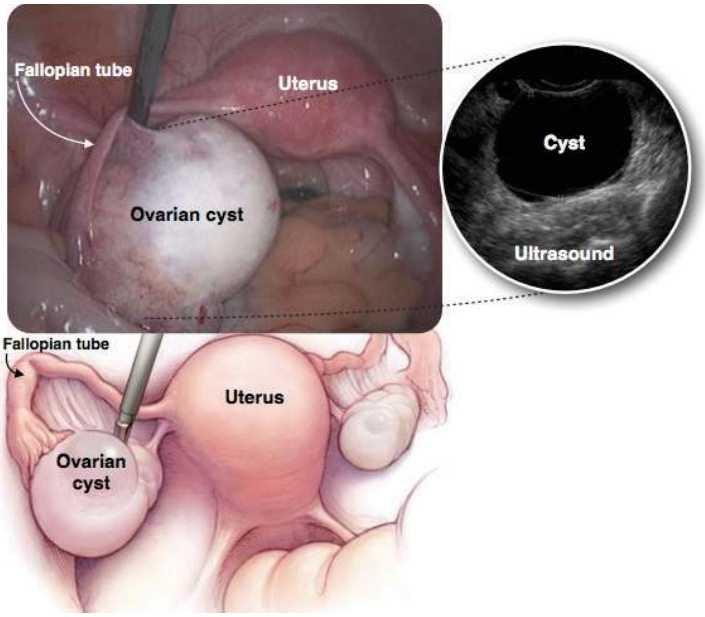
**Management**
• Drainage (needle aspiration, I&D)
• Antibiotics (e.g., dicloxacillin or cephalexin)
• If suspect MRSA (e.g., trimethoprim sulfamethoxazole, clindamycin)
• If breastfeeding, do not stop

Notes


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# VIII. Ovarian Cysts

## A. Ovarian Cysts




Subtypes
- Follicular: **most common** ovarian mass, non-neoplastic, regress spontaneously
- Corpus luteum: **most common** ovarian mass in pregnancy, non-neoplastic, regress spontaneously
- Dermoid: teratoma
- Theca lutein: bilateral ovarian enlargement
- Endometrioid: endometriosis within ovary, "chocolate" cyst

Diagnostic Studies
- Ultrasound

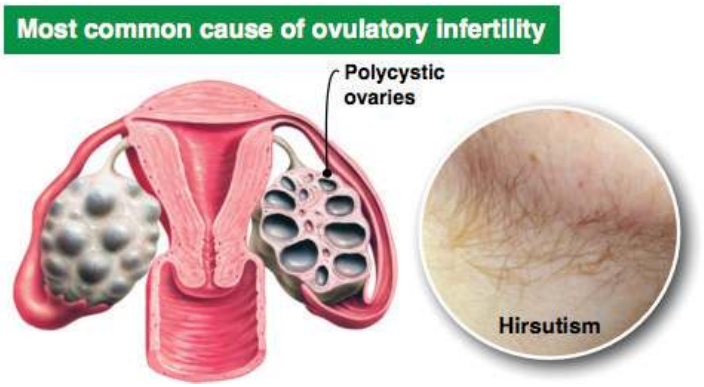
**Ultrasound findings concerning for malignancy**
- Cyst size > 10 cm
- Solid or papillary components
- Irregularity
- High color Doppler flow
- Presence of ascites

**Management**
- Observation (asymptomatic and malignancy risk is low)
- Surgical (minimally invasive when feasible)

Notes


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## B. Polycystic Ovarian Syndrome (PCOS)




Pathophysiology
• Peripheral **insulin resistance** leads to **hyperinsulinemia**
• Ovarian stimulation leading to excess androgen production
• Genetic factors

Patient
• **Obese** female

Presentation
• **Amenorrhea**, **obesity**, **hirsutism**

Physical Exam
• Bilateral ovarian enlargement, acanthosis nigricans

Diagnostic Studies
• **High LH** : **FSH**, androgen excess

Management
• Combination **oral contraceptive pills**, **lifestyle changes**, **metformin**

Comments
• **Most common** cause of **ovulatory infertility**

**Clinical**
• Hirsutism
• Menstrual irregularities
• Acanthosis nigricans
• Obesity
• Acne

**Additional comorbidities**
• Impaired glucose tolerance
• Metabolic syndrome
• Obstructive sleep apnea
• Fatty liver disease
• Endometrial hyperplasia

| Proposed Diagnostic Criteria for Polycystic Ovarian Syndrome |
|---|
| **2003 Rotterdam Criteria for Polycystic Ovarian Syndrome** |
| ***Two out of three of the following criteria are required to make the diagnosis*** |
| • Oligo- or anovulation or both |
| • Clinical or biochemical signs of hyperandrogenism or both |
| • Polycystic ovaries (by ultrasound) |
| • Exclusion of other conditions that mimic polycystic ovarian syndrome |
| **Ultrasound Criteria**: Ovarian volume > 10 $cm^3$/10 mL or ≥ 12 antral follicles |

Notes


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### C. Ovarian Torsion

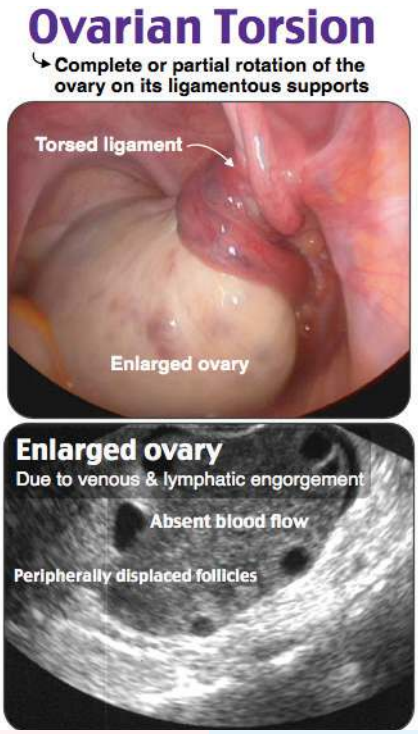
Pathophysiology
• **Most commonly** caused by an ovarian **cyst** or **tumor**

Patient
• **15–30 years old** or **postmenopausal**

Presentation
• **Sudden onset** of **unilateral** (**right** > left) **abdominal** and **pelvic pain**

Diagnostic Studies
• Transvaginal ultrasound
• **Gold standard**: **Laparoscopy**

Management
• Surgery




| Risk factor | Clinical |
|---|---|
| • **Ovarian mass ≥ 5 cm** | • Pelvic pain |
| • Reproductive age | • Adnexal mass |
| • Pregnancy | • Nausea/vomiting |
| • Ovulation induction | • Fever |
| • Prior torsion | • Vaginal bleeding |

| Imaging | Management |
|---|---|
| • Pelvic ultrasound | • Operative evaluation |

Notes


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# IX. Pelvic Inflammatory Disease

## A. Pelvic Inflammatory Disease (PID)

Pathophysiology
• **Most commonly** caused by ***Chlamydia trachomatis***

Patient
• History of **multiple sexual partners** or **unprotected sex**

Presentation
• Lower abdominal pain, painful sexual intercourse

Physical Exam
• **Mucopurulent cervical discharge**, **cervical motion tenderness** (**"chandelier sign"**)

Management
• **Ceftriaxone** with **doxycycline**

Comments
• **Fitz-Hugh-Curtis syndrome**: **perihepatitis** with PID

**Major Criteria**
• Lower abdominal pain in at risk population PLUS
• Uterine tenderness OR
• Adnexal tenderness OR
• Cervical motion tenderness

**Supporting Criteria**
• Fever > 38°C
• Abnormal vaginal discharge
• Positive culture for *N. gonorrhoeae* or *C. trachomatis*
• Elevated CRP or ESR

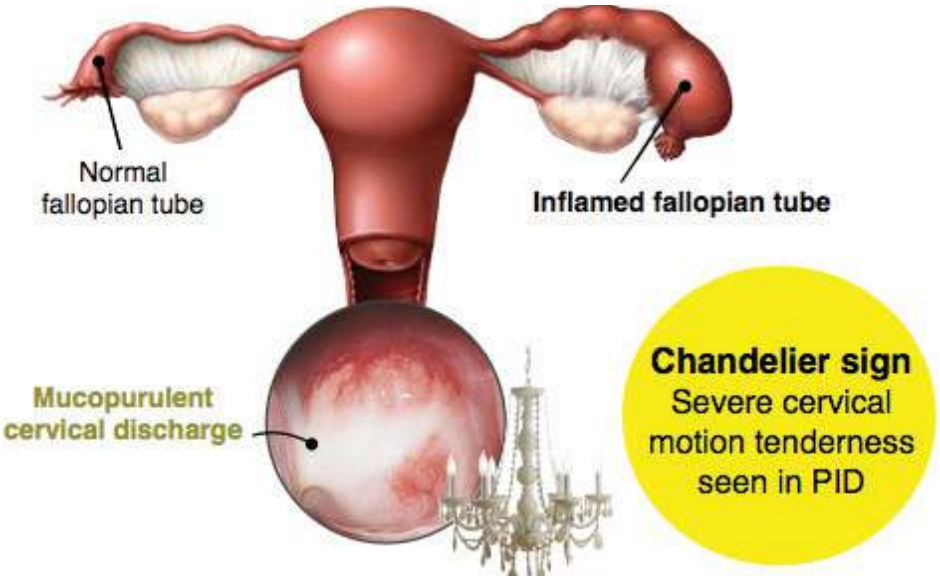
**Rx (outpatient)**
• Ceftriaxone 250 mg IM
• Doxycycline 100 mg PO BID x 14 d
• +/- Metronidazole 500 mg PO BID x 14 d

**Rx (inpatient)**
• Cefotetan 2 gm q12 hours
• Doxycycline 100 mg PO/IV q12 hours

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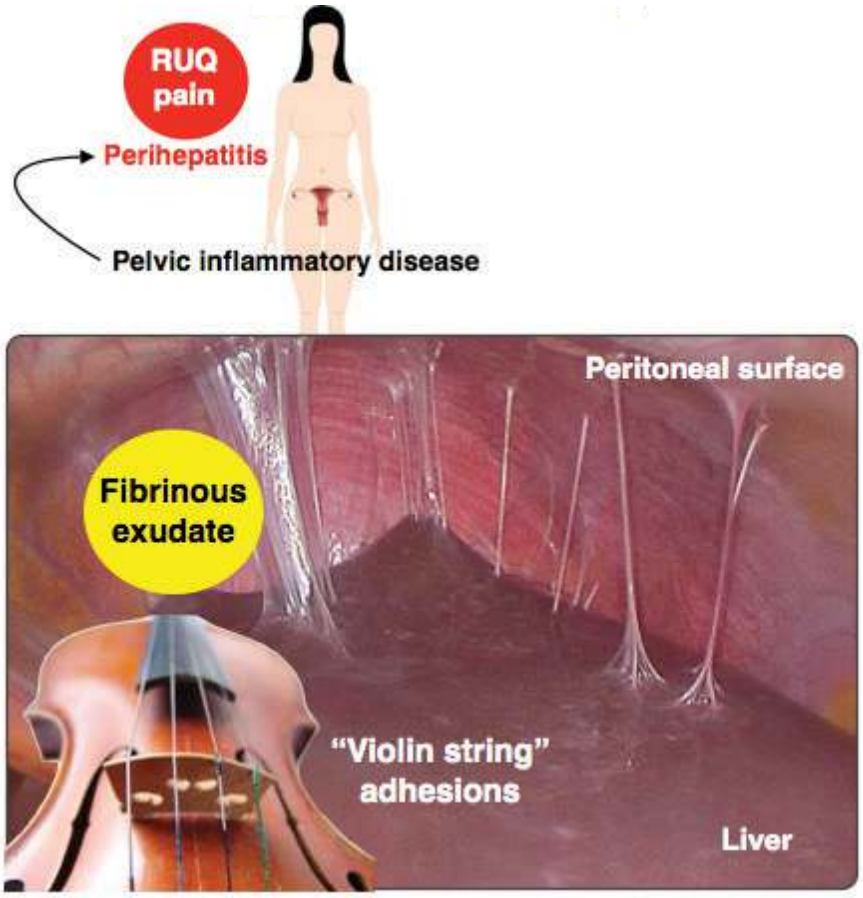
**Pelvic Inflammatory Disease (PID) cont.**




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**B. Fitz-Hugh-Curtis Syndrome**




**Management**
- Treat the PID infection
- Ceftriaxone and doxycycline

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# X. Pelvic Pain/Dysmenorrhea

## A. Uterine Fibroids (Leiomyoma)

**Most common solid pelvic tumor in women**

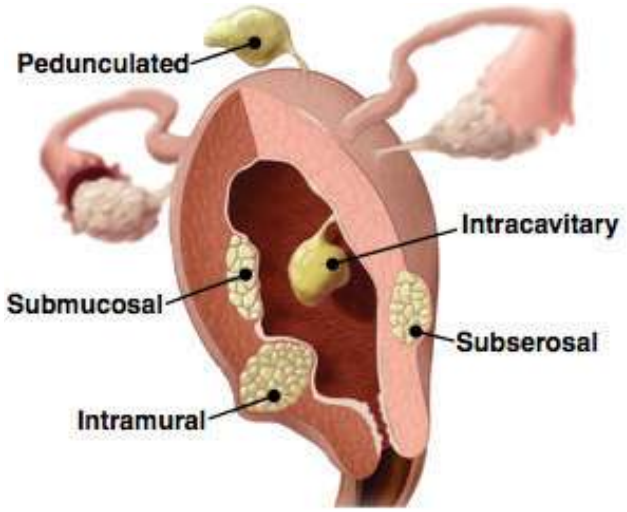
Patient
• **African-American** woman, **20–40 years old**

Presentation
• **Menorrhagia** and **dysmenorrhea**

Physical Exam
• **Palpable**, **asymmetric**, and **nontender** uterus

Diagnostic Studies
• Pelvic ultrasound

Management
• Majority do not require surgical or medical treatment
• Severe cases: **myomectomy** (fertility can be preserved) or **hysterectomy**




**Risk Factors**
• Advancing age
• Hyperestrogen states
• African-Americans
• Hypertension
• Nulliparity

**Management**
• Hormonal therapy
• Myomectomy
• Embolization
• Hysterectomy

**Clinical**
• Menorrhagia (fibroids most common cause)
• Pelvic pain
• Palpable uterus above symphysis pubis

Notes


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# XI. Placental Abruption

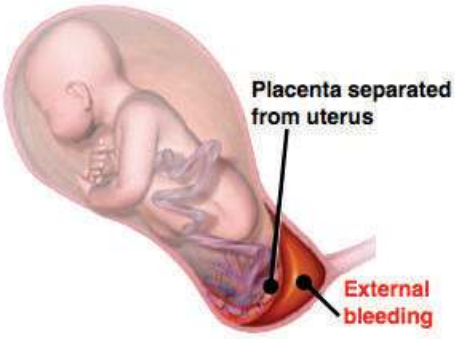
## A. Placental Abruption

Patient
• Pregnant woman in her **third trimester** with a history of hypertension, trauma, or cocaine use

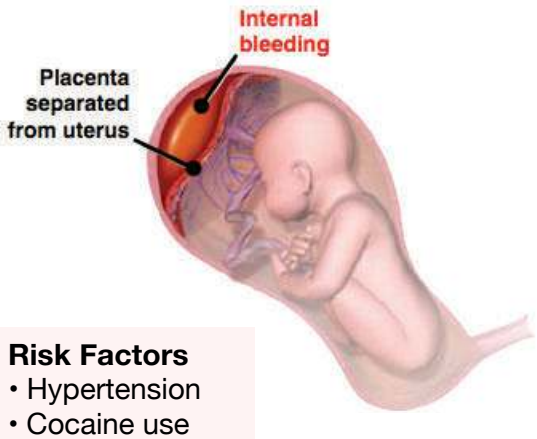
Presentation
• **Painful** vaginal bleeding

Diagnostic Studies
• **Hypofibrinogenemia**

**Placental Abruption** – **Premature separation** of the implanted **placenta** from the **uterine wall**




**PAINFUL** vaginal bleeding but...
Bleeding is **not** always present (may be internal)




**Risk Factors**
• Hypertension
• Cocaine use
• Trauma
• Multiparity
• Smoking

**Diagnosis**
• Clinical

Notes


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# XII. Placenta Previa

## A. Placenta Previa

Pathophysiology
• Placenta implants on cervix, **covering internal os**

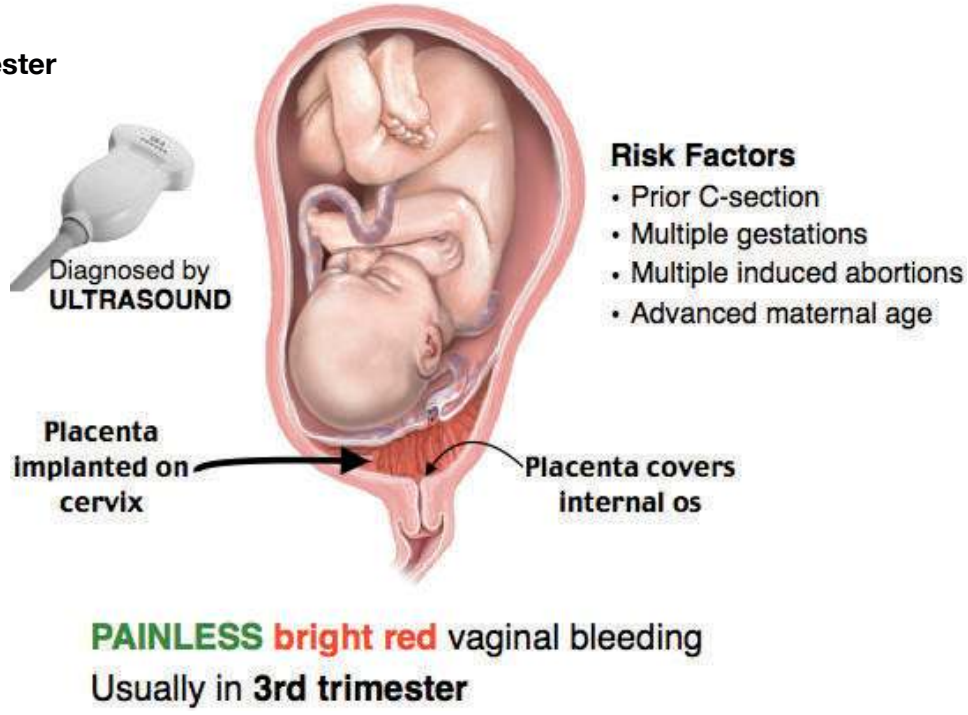
Patient
• Pregnant women in her **third trimester**

Presentation
• **Painless** vaginal bleeding

Physical Exam
• Do **not** do a digital vaginal exam

Diagnostic Studies
• **Transabdominal ultrasound**

**Placenta Previa** – **Placenta** overlaps and **implants** on the **cervix**, **covering the internal os** to varying degrees




Notes


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# XIII. Prelabor Rupture of Membranes

## A. Prelabor Rupture of Membranes

Pathophysiology
• Rupture of membranes at **≥ 37 weeks gestation** prior to the start of uterine contractions
• **Preterm** prelabor rupture of membranes (PPROM) describes PROM < 37 weeks gestation

Diagnostic Studies
• Need to confirm that fluid is truly **amniotic fluid**
  • Sterile speculum exam: fluid pooling in posterior fornix
  • **Nitrazine** test: blue (due to **elevated** pH) determine if this is amniotic fluid; **pH > 7.1** means it is positive
  • Microscope examination, **ferning**: take a specimen of the fluid, put it on a slide and let it air dry - will see "fern pattern" crystallization of the amniotic fluid (crystallization of estrogen and amniotic fluid)

Management
Depends on gestational age
  • > 37 weeks: Proceed to labor, GBS prophylaxis as indicated
  • 34–37 weeks: same as above
  • 24–33 weeks: Expectant management, antibiotics recommended to prolong latency if there are no contraindications, single-course corticosteroids
  • < 24 weeks: Patient counseling, expectant management or induction of labor
    • Antibiotics may be considered as early as 20 weeks of gestation
    • GBS prophylaxis, corticosteroids, tocolysis, and magnesium sulfate are **not** recommended before viability

Notes


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# XIV. Spontaneous Abortion

### A. Abortion

• Threatened abortion: vaginal bleeding with **closed os**
• Inevitable: vaginal bleeding with **open os**
• Incomplete: **partial** passage of products of conception (POC)
• Complete: **complete** passage of POC
• Missed: fetal death < 20 weeks **without POC passage**
• Missed abortion complications: infection, coagulopathy
• Rh-negative women: **RhoGAM**

Notes


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### B. Types of Spontaneous Abortion

| Types of Spontaneous Abortion | | | |
|---|---|---|---|
| **Abortion Classification** | **Characteristics** | **Cervical Os** | **Passage of Fetal Tissue** |
| **Threatened** | Abdominal pain or bleeding in the first 20 weeks of gestation | Closed | None |
| **Inevitable** | Abdominal pain or bleeding in the first 20 weeks of gestation | Open | None |
| **Incomplete** | Abdominal pain or bleeding in the first 20 weeks of gestation | Open | Yes (some products of conception still remain in uterus) |
| **Complete** | Abdominal pain or bleeding in the first 20 weeks of gestation | Closed | • Complete passage of fetal parts and placenta<br>• Uterus contracted |
| **Missed (delayed miscarriage)** | In-utero death of the embryo or fetus prior to 20 weeks of gestation with retention of pregnancy | Closed | None |
| **Septic** | Infection of uterus during miscarriage, fever and chills. Usually due to *Staph. aureus* | • Open with purulent cervical discharge<br>• Uterine tenderness | None (may be incomplete) |

Notes


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# XV. Vaginal Discharge/Vaginitis

## A. Atrophic Vaginitis

Pathophysiology
• Caused by a **decrease** in **estrogen**

Patient
• **Postmenopausal** woman

Presentation
• **Dyspareunia**, **dryness**, bleeding, itching

Physical Exam
• Pale, dry, **shiny** vaginal epithelium

Management
• Lubricants, moisturizers
• Topical estrogen (second line)

## B. Genitourinary Syndrome of Menopause

• Previously referred to as **atrophic vaginitis**




**Clinical**
• Vaginal dryness
• Postmenopausal women
• Due to loss of estrogens
• Dryness, burning, pruritus
• Dyspareunia, bleeding

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### C. Vulvitis, Vaginitis, Vulvovaginitis

**Vulvitis**
External genital pruritus, burning, redness, rash

**Vaginitis**
Inflammation of the vagina

**Vulvovaginitis**
Vaginal irritation, pain, and pruritus

**Non-Sexually Transmitted**

- Poor or excessive hygiene
- Chemical irritants (bubble bath, soap)
- Tight clothing
- Pinworms
- Group A beta hemolytic Strep, Staph, Candida

**Sexually Transmitted**

- Gonorrhea
- Chlamydia
- *Trichomonas vaginalis*
- Herpes simplex

Notes


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### D. Vulvovaginal Candidiasis

Pathophysiology
• **Most commonly** caused by Candida spp.

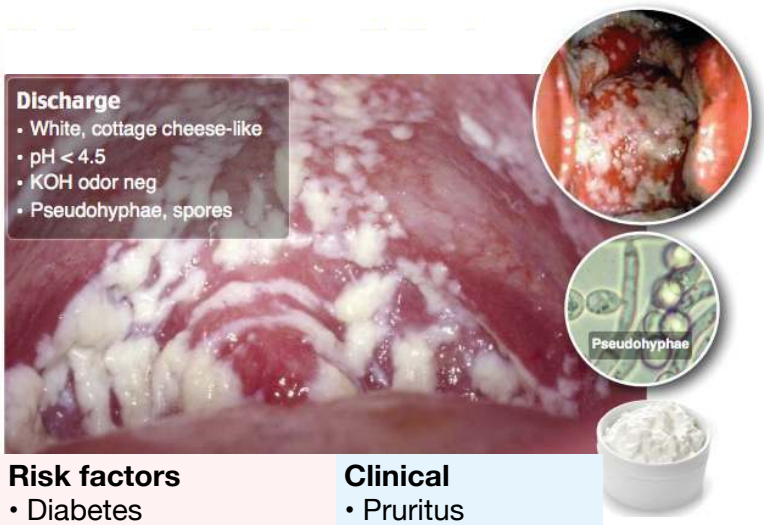
Patient
• History of **diabetes**, **HIV**, recent **antibiotic** use

Presentation
• **Pruritus**, **dysuria**, **dyspareunia**

Physical Exam
• **White**, **cottage cheese-like discharge**

Diagnostic Studies
• Vaginal **pH < 4.5**, **pseudohyphae**, **spores**

Management
• **Oral fluconazole**, **topical azole**




**Risk factors**
• Diabetes
• HIV
• Recent antibiotic use
• Pregnancy

**Clinical**
• Pruritus
• Dysuria
• Dyspareunia

**Management**
• Topical azole
• Oral fluconazole

Notes


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### E. Trichomoniasis

Pathophysiology
• **Most commonly** caused by ***Trichomonas vaginalis***

Presentation
• **Malodorous** vaginal discharge

Physical Exam
• **Frothy, green/yellow** cervical discharge, **"strawberry cervix"**

Diagnostic Studies
• Vaginal **pH > 5.0**
• Wet mount is diagnostic, revealing **flagellated**, motile, pear-shaped organisms

Management
• **Metronidazole**




**Treatment**
• Metronidazole or tinidazole

Notes


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## F. Bacterial Vaginosis

Pathophysiology
- **Most commonly** caused by ***Gardnerella vaginalis***

Presentation
- **Malodorous** vaginal discharge

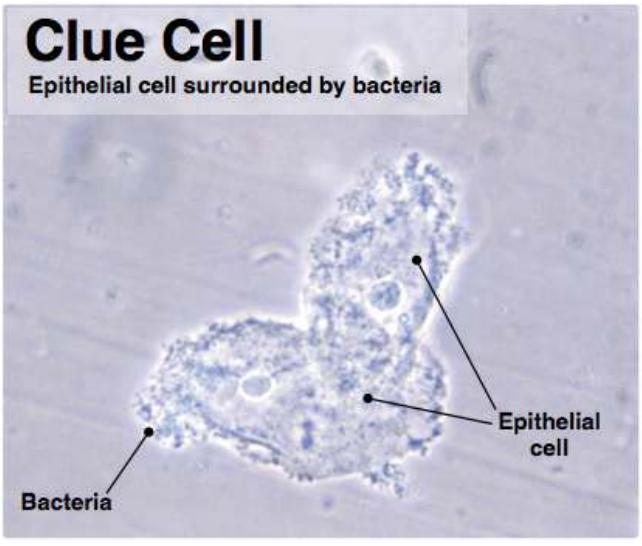
Physical Exam
- **Thin**, **gray** and **white discharge**

Diagnostic Studies
**Amsel Criteria** (3 of 4)
- **Clue cells**
- **A pH greater than 4.5**
- **Characteristic discharge appearance is thin, gray, and homogeneous**
- **Whiff test positive**

Management
- **Metronidazole**

***3 of 4 criteria must be met to diagnose bacterial vaginosis***

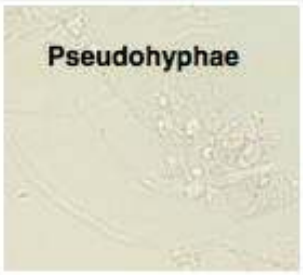
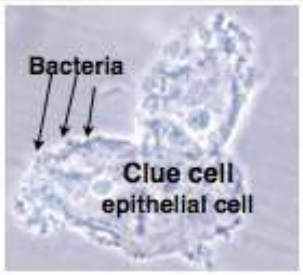
1. **A thin, white homogenous discharge**
2. **Presence of clue cells on microscopic exam**
3. **pH of vaginal fluid > 4.5**
4. **A fishy odor to vaginal discharge before or after addition of 10% KOH ("whiff test")**




Notes


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## G. Vaginal Infections

| | Candidiasis | Trichomoniasis | Bacterial vaginosis |
|---|---|---|---|
| Discharge | White | Green/yellow | Gray/white |
| Color |  |  |  |
| pH | < 4.5 | > 5 | > 4.5 |
| Amine odor with KOH | Negative | Positive | Positive |
| Wet mount | • WBC<br>• Spores<br>• **Pseudohyphae** | • WBC<br>• **Motile trichomonads** | • Few WBCs<br>• **Clue cells** |
| |  |  |  |

Notes


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CHAPTER 11

# Psychiatry/ Behavioral Medicine

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CHAPTER 11

# Psychiatry/Behavioral Medicine


478 **ANXIETY DISORDERS**

481 **BIPOLAR AND RELATED DISORDERS**

483 **DEPRESSIVE DISORDERS**

486 **NEUROCOGNITIVE DISORDERS**

490 **PANIC DISORDER**

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494 **SCHIZOPHRENIA SPECTRUM AND OTHER PSYCHOTIC DISORDERS**

497 **SPOUSE OR PARTNER NEGLECT/ VIOLENCE**

500 **SUBSTANCE USE DISORDERS**

501 **SUICIDE**



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# I. Anxiety Disorders

### A. General Anxiety Disorder

Presentation
• Persistent and excessive worry pertaining to **multiple events or domains** that continues for **six months or more**

Management
• SSRIs: **paroxetine** and **escitalopram**; SNRIs: **venlafaxine**
• Benzodiazepines (short-term use), BBs
• Buspirone is also effective (can take at least two weeks before it begins to help)
• Psychotherapy

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### B. Generalized Anxiety Disorder DSM-5 Criteria

| The diagnosis of generalized anxiety disorder is based on the presence of generalized, persistent, and excessive anxiety and a combination of various psychological and somatic complaints. DSM-5 diagnostic criteria for generalized anxiety disorder require the presence of the following: |
|---|
| A. Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least six months, about a number of events or activities (such as work or school performance). |
| B. The individual finds it difficult to control the worry. |
| C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms having been present for more days than not for the past six months):<br>1. Restlessness or feeling keyed up or on edge<br>2. Being easily fatigued<br>3. Difficulty concentrating or mind going blank<br>4. Irritability<br>5. Muscle tension<br>6. Sleep disturbance (difficulty falling or staying asleep, or restless, unsatisfying sleep) |
| D. The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning. |
| E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism). |
| F. The disturbance is not better explained by another mental disorder (e.g., anxiety or worry about having panic attacks in panic disorder, negative evaluation in social anxiety disorder [social phobia], contamination or other obsessions in obsessive-compulsive disorder, separation from attachment figures in separation anxiety disorder, reminders of traumatic events in post-traumatic stress disorder, gaining weight in anorexia nervosa, physical complaints in somatic symptom disorder, perceived appearance flaws in body dysmorphic disorder, having a serious illness in illness anxiety disorder, or the content of delusional beliefs in schizophrenia or delusional disorder). Because the majority of the anxiety symptoms are not specific to GAD, it is important to exclude the other anxiety disorders before making the diagnosis. |

Notes


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### C. Specific Phobias

Presentation
• Symptoms begin 10–15 minutes prior to **specific** stress event (i.e., flying, blood, social situations, spiders)

Management
• **Exposure therapy** (first line) teaches to relax and try to understand and overcome the fear
• Medications may be used (SSRIs) in combination with cognitive behavioral therapy (CBT)
• Benzodiazepines for **specific** and **predictable** triggers such as prior to flying
• Treat agoraphobia just as generalized anxiety disorder with SSRIs and CBT

### D. Specific Phobic Specifiers (DSM-5)

1. • Animal (e.g., spiders, insects, dogs)
2. • Natural environment (e.g., heights, storms, water)
3. • Blood-injection-injury (e.g., needles, invasive medical procedures)
4. • Situational (e.g., airplanes, elevators, enclosed places)
5. • Other (e.g., situations that may lead to choking or vomiting, or in children: loud sounds, costumed characters)

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# II. Bipolar and Related Disorders

## A. Bipolar

Presentation
- **Bipolar I**: A manic episode **with or without** a major depressive episode
- **Bipolar II**: **At least one hypomanic** episode (no mania) and **at least one** major depressive episode
- **Cyclothymic disorder**: A chronic mood disorder characterized by episodes of **depression** and **hypomania** for at least two years
  - This is a less intense but often longer lasting version of bipolar disorder
  - Cyclothymia has both high and low mood, but never as severe as either mania or major depression

Management
- Treatment depends on the manifestations and their severity
- Mood stabilizers: lithium, valproate, carbamazepine, lamotrigine
- Second-generation antipsychotics: aripiprazole, lurasidone, olanzapine, quetiapine, risperidone, ziprasidone

**Bipolar Disorder**



**DIG FAST** (Mania)

**D**istractibility
**I**mpulsivity - poor judgement, spending sprees, reckless driving
**G**randiosity - increased self esteem

**F**light of ideas - racing thoughts
**A**ctivities - psychomotor agitation
**S**leep - decreased need
**T**alkativeness - pressured speech

Notes


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### B. Comparison of DSM-5 Criteria for Bipolar I vs. Bipolar II Disorder

| Criteria | Bipolar I Disorder | Bipolar II Disorder |
|---|---|---|
| **Manic episodes** | **Yes** | **No** |
| **Hypomanic episodes** | Commonly occur, but not required | Yes |
| **Major depressive episodes** | Usually occur, but not required | Yes |
| **Mixed features** | May occur | May occur |
| **Anxious distress** | May occur | May occur |
| **Rapid cycling** | May occur | May occur |
| **Psychotic features** | May occur | May occur |
| **Catatonia** | May occur | May occur |

Notes


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# III. Depressive Disorders

### A. Major Depressive Disorder

Presentation
• Five or more SIG-E-CAPS for > two weeks nearly every day and at least one of the symptoms is **depressed mood or anhedonia**
  • SIG-E-CAPS: **S**adness, **I**nterest/anhedonia, **G**uilt, **E**nergy, **C**oncentration, **A**ppetite, **P**sychomotor activity, **S**uicidal

Management
• **SNRIs**, **SSRIs**
• Continue to increase dosage every three to four weeks until symptoms are in remission
• Full medication effect is complete in four to six weeks
• Augmentation with second medication may be necessary
• Follow-up within two to four weeks of starting medication and every two weeks until there is improvement, then monthly to monitor medication changes

**One or more** episodes of **depressed mood** or **anhedonia** for **at least 2 weeks**
**AND**
At least **5 of the SIG E CAPS** symptoms of depression

**S**leep - hypersomnia/insomnia
**I**nterest - loss of interest or pleasure in activities (anhedonia)
**G**uilt - a feeling of worthlessness or inappropriate guilt
**E**nergy - low energy
**C**oncentration - poor concentration
**A**ppetite - decreased or increased appetite
**P**sychomotor agitation or retardation
**S**uicidal ideation

Notes


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## DSM-5 Diagnostic Criteria for a Major Depressive Disorder

**A.** Five (or more) of the following symptoms have been present during the same two-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

*NOTE: Do not include symptoms that are clearly attributable to another medical condition.*

1) Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, hopeless) or observations made by others (e.g., appears tearful). (NOTE: In children and adolescents, can be irritable mood.)

2) Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation)

3) Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (NOTE: In children, consider failure to make expected weight gain.)

4) Insomnia or hypersomnia nearly every day

5) Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down)

6) Fatigue or loss of energy nearly every day

7) Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)

8) Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by their subjective account or as observed by others)

9) Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

**B.** The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

**C.** The episode is not attributable to the direct physiological effects of a substance or to another medical condition.

*NOTE: Criteria A through C represent a major depressive episode.*

NOTE: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgement based on the individual's history and the cultural norms for the expression of distress in the context of loss.

**D.** The occurrence of the major depressive episode is not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders.

**E.** There has never been a manic or hypomanic episode.

Specify:

With anxious distress
With mixed features
With melancholic features
With atypical features
With atypical features
With psychotic features
With catatonia
With peripartum onset
With seasonal pattern


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**B. Persistent Depressive Disorder (Dysthymia)**

• Mild chronic form of major depression lasting more than two years

| DSM-5 Diagnostic Criteria for Persistent Depressive Disorder (dysthymia) |
|---|
| **A.** Depressed mood for most of the day, for more days than not, as indicated either by subjective account or observation by others, for at least two years. |
| **B.** Presence, while depressed, of two (or more) of the following:<br>1) Poor appetite or overeating.<br>2) Insomnia or hypersomnia.<br>3) Low energy or fatigue.<br>4) Low self-esteem.<br>5) Poor concentration or difficulty making decisions.<br>6) Feelings of hopelessness. |
| **C.** During the two-year period (one year for children or adolescents) of the disturbance, the individual has never been without the symptoms in Criteria A and B for more than two months at a time. |
| **D.** Criteria for a major depressive disorder may be continuously present for two years. |
| **E.** There has never been a manic episode or a hypomanic episode, and criteria have never been met for cyclothymic disorder. |
| **F.** The disturbance is not better explained by a persistent schizoaffective disorder, schizophrenia, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder. |
| **G.** The symptoms are not attributable to the physiological effects of a substance (eg, a drug of abuse, a medication) or another medical condition (eg, hypothyroidism). |
| **H.** The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning. |
| **NOTE:** Because the criteria for a major depressive episode include four symptoms that are absent from the symptom list for persistent depressive disorder (dysthymia), a very limited number of individuals will have depressive symptoms that have persisted longer than two years but will not meet criteria for persistent depressive disorder. If full criteria for a major depressive episode have been met at some point during the current episode of illness, they should be given a diagnosis of major depressive disorder. Otherwise, a diagnosis of other specified depressive disorder or unspecified depressive disorder is warranted. |
| *From the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, (Copyright © 2013). American Psychiatric Association.* |

Notes


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# IV. Neurocognitive Disorders (Dementia)

### A. Delirium vs. Dementia

| Characteristic | Delirium | Dementia |
|---|---|---|
| **Onset** | Abrupt | Insidious |
| **Course** | Fluctuates | Slow decline |
| **Duration** | Hours to weeks | Months to years |
| **Attention** | Impaired | Intact early; impaired late |
| **Sleep-wake** | Disrupted | Usually normal |
| **Alertness** | Impaired | Normal |
| **Orientation** | Impaired | Intact early; impaired late |
| **Behavior** | Agitated, withdrawn or depressed | Intact early |
| **Speech** | Incoherent, rapid/slowed | Word-finding problems |
| **Thoughts** | Disorganized, delusions | Impoverished |
| **Perceptions** | Hallucinations/illusions | Intact early |

Notes


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**B. Types of Dementia**



## Types of Dementia

**Alzheimer disease**
2/3 of dementia cases.
Risk factors: advanced age, family history

**Vascular dementia**
1/4 of cases
Risk factors: HTN, dyslipidemia, DM, smoking, adv age

**Lewy body dementia**
Cognitive fluctuations, visual hallucinations, Parkinsonism

**Frontotemporal dementia**
Personality and social behavior changes, nonfluent speech

**Neurodegenerative conditions**
Huntington disease, metabolic abnormalities

Notes

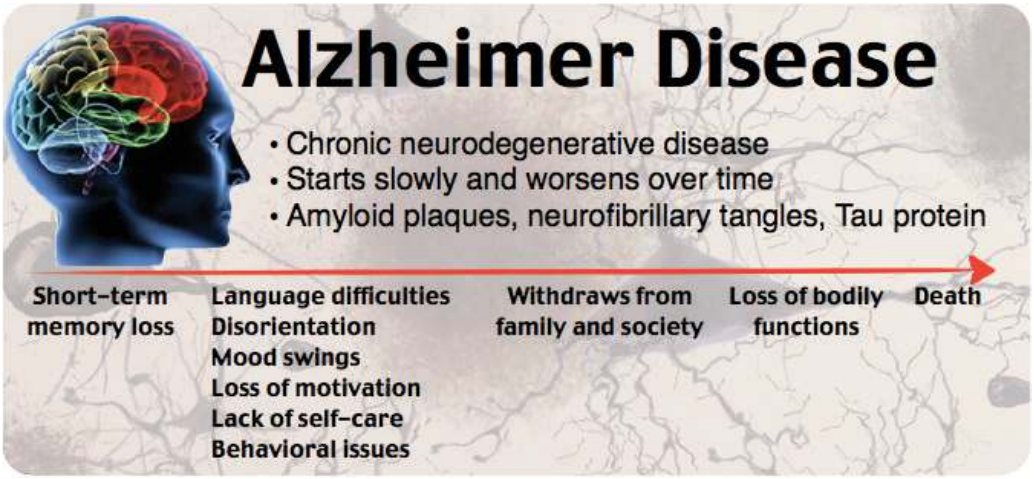

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### C. Alzheimer Disease

Presentation
• **Short-term memory loss**, confusion about the location of familiar places, difficulty completing daily tasks that would normally take less time to do, poor judgment which can lead to bad decision making, changes in mood or personality

Diagnostic Studies
• Definitive diagnosis is confirmed by histopathologic examination of the brain showing **amyloid plaques** and **neurofibrillary tangles**
• In practice, diagnosis is made with clinical criteria
• Mini-mental state exam monitors progression

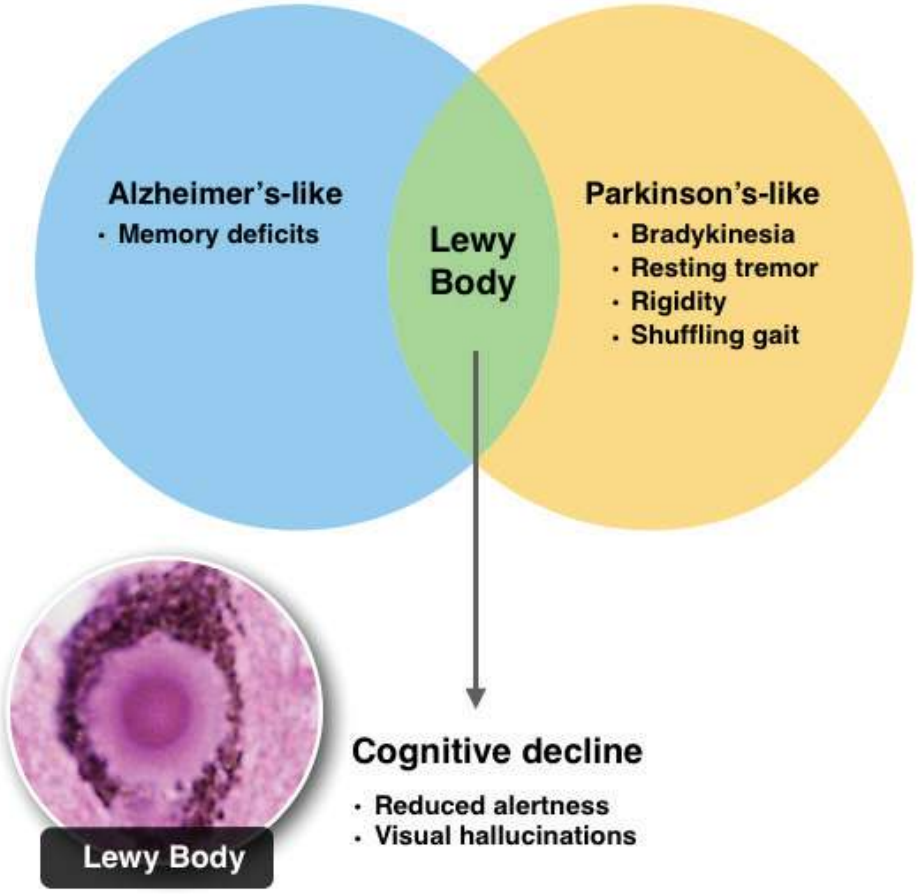
Management
• Cholinesterase inhibitors (donepezil), NMDA antagonists (memantine)
• No known cure; medications aim to slow progression




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**D. Lewy Body Dementia**




Notes


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# V. Panic Disorder

### A. Panic disorder

Presentation
• Recurrent, **unexpected** panic attacks with at least a month or more of **worry** or **avoidant behavior**
• Palpitations, chest pain, sweating, SOB
• Symptoms develop abruptly and reach a **peak within 10 minutes**
• Can occur with or without agoraphobia

Management
• **CBT** (cognitive behavioral therapy; relaxation, desensitization, examining behavior consequences)
• SSRIs: paroxetine, sertraline, fluoxetine
• Benzodiazepines for acute attacks (watch for abuse)

| DSM-5 Criteria for Panic Disorder |
|---|
| A. Recurrent unexpected panic attacks |
| B. At least one of the attacks has been followed by a month or more of one or both of the following:<br>(1) Persistent concern or worry about additional panic attacks or their consequences<br>(2) A significant maladaptive change in behavior related to the attacks |
| C. The disturbance is not attributable to the physiological effects of a substance or another medical condition |
| D. The disturbance is not better explained by another mental disorder such as:<br>• Feared social situations, as in social anxiety disorder<br>• Circumscribed phobic objects or situations, as in specific phobia<br>• Obsessions, as in obsessive-compulsive disorder<br>• Reminders of traumatic events, as in posttraumatic stress disorder<br>• Separation from attachment figures, as in separation anxiety disorder |

Notes


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**B. Dominant Symptoms of Panic Disorder**

1. Sudden onset of palpitations
2. Chest pain
3. Choking sensation
4. Dizziness
5. Feelings of unreality (depersonalization or derealization)
6. Secondary fear of dying, losing control, or going mad

Notes


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# VI. Post-Traumatic Stress Disorder

### A. Post-Traumatic Stress Disorder

Presentation
- **Recent traumatic event** which causes an acute stress reaction
- Once the symptoms persist past one month it is now considered post-traumatic stress disorder (PTSD)

Management
- **SSRIs** are first line
- **CBT**
- **Prazosin** for nightmares
- Benzodiazepines, if used, should not be continued for more than two weeks after a traumatic event

Notes


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**B. Posttraumatic Stress Disorder (PTSD) in Adults**

| Clinical | |
|---|---|
| **Re-experiencing trauma** | • Intrusive memories<br>• Dreams<br>• Flashbacks |
| **Avoidance** | • Social withdrawal<br>• Emotional detachment<br>• Sense of foreshortened future |
| **Hyperarousal** | • Insomnia<br>• Irritability<br>• Difficulty concentrating<br>• Exaggerated startle response |
| **Management** | |
| **Trauma-focused psychotherapy** | • Exposure<br>• trauma-focused cognitive-behavioral therapy<br>• Eye movement desensitization and reprocessing |
| **Serotonin reuptake inhibitor** | • SSRIs (e.g. paroxetine)<br>• SNRIs (e.g. venlafaxine) |
| **Second-generation antipsychotics** | • Quetiapine (monotherapy)<br>• Risperidone (augmentation with SRI) |
| **Alpha-adrenergic receptor blockers** | • Prazosin |

Notes


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# VII. Schizophrenia Spectrum and Other Psychotic Disorders

## A. Schizophrenia

Presentation

- **Two** of the following in a **one month** period and **continuous signs** for **more than six months**; hallucinations/delusions not needed for diagnosis
  - **Delusions**
  - **Hallucinations**: auditory (**most common**), tactile, olfactory, visual
  - **Disorganized speech** or **thought** processes, unable to stay on topic (loose associations) unable to provide answer related to questions (tangential response)
  - **Disorganized behavior**: unpredictable agitation, inappropriate sexual behavior, childlike silliness, catatonic motor behavior, lacking self-care or hygiene
  - **Negative symptoms**: blunted affect, poor posture, lack goal-directed activities or initiative
  - **Impairment**: inability to hold job or maintain relationships

Management

- **Hospitalize** if suicidal, unable to care for self, or pose a threat to self or others
- First line: **serotonin and dopamine antagonists** (SDAs); **atypical antipsychotics** (risperidone, olanzapine, aripiprazole, ziprasidone, quetiapine, asenapine, paliperidone) for negative symptoms and fewer side effects

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**Schizophrenia cont.**

| Phase | Signs and Symptoms |
|---|---|
| **Prodromal** | • Social isolation<br>• New interest in religion/philosophy<br>• Restlessness<br>• Difficulty concentrating |
| **Positive symptoms** | • Delusions<br>• Hallucinations<br>• Strange behaviors<br>• Incoherent thought process<br>• Grossly disorganized |
| **Negative symptoms** | • Flat affect<br>• Decreased fluency<br>• Decreased productivity of thought and speech<br>• Social withdrawal<br>• Decreased goal-directed behavior |
| **Physical manifestations** | • Neurological disturbances<br>• Catatonia<br>• Metabolic disturbances |
| **Residual** | • Occurs between episodes of psychosis<br>• Patient retains flat affect and continues to hear voices |

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### B. Schizoaffective Disorder

1. Meets diagnostic criteria for **schizophrenia**
2. Meets diagnostic criteria for a **mood disorder**
3. Requires at least one **two-week period** of illness during which he or she experiences **hallucinations or delusions** or both in the absence of any prominent mood episode

Notes


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# VIII. Spouse or Partner Neglect/Violence

## A. Domestic Violence

Pathophysiology
• Includes physical, sexual, and psychological abuse between intimate partners

Patient
• Victim is usually a **woman**, and more than one in three women have experienced domestic violence in their lifetime
• Women who are marginalized by their race, gender, sexuality, and class are more vulnerable to domestic violence

Presentation
• Physical injuries, psychological problems, social isolation, loss of a job, financial difficulties, and even death can result
• Domestic violence often begins or, if already present, increases during pregnancy and the postpartum period

Management
• Keeping safe, for example having a plan of escape, is the most important consideration

Comments
• Women are more likely to be assaulted or murdered when attempting to **report** the abuse or **leave** the abusive relationship; up to 75% of domestic assaults occur after separation

## B. Domestic Violence: Characteristic Injuries

• Bilateral injuries, especially to the extremities
• Injuries at multiple sites
• Fingernail scratches, cigarette burns, rope burns
• Abrasions, minor lacerations, welts
• Subconjunctival hemorrhage suggests vigorous struggle between victim and assailant

Notes


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**C. Sexual Assault**

Patient
• Male victims reported in 5% of cases (recent data suggests this can be as high as 22% due to significant lack of reporting)

Diagnostic Studies
• **Pregnancy testing** is indicated
• **Toluidine blue**: detects vulvar tears
• **Wood lamp**: detects semen stains

Management
• Consideration of **postexposure prophylaxis** for HIV and hepatitis B immunoglobulin only if the perpetrator is known to be infected with either of these
• Standard treatment covers **gonorrhea**, **chlamydia**, and **trichomoniasis**
• Standard vaccination is to **start hepatitis B series** if nonimmunized
• Consider pregnancy status and prevention

Comments
• Infections considered diagnostic for abuse in prepubescent population: syphilis, human immunodeficiency virus, gonorrhea, and chlamydia

Notes


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**D. Clinical Findings Associated with Sexual Abuse**

| | |
|---|---|
| **Genital-rectal complaints** | • Genital, anal, or urethral trauma*, bleeding, or itching<br>• Chronic genital or anal pain<br>• Genital infection or discharge<br>• Vaginal or rectal foreign body<br>• Vulvitis or vulvovaginitis<br>• Anal inflammation<br>• Sexually transmitted infection*<br>• Pregnancy*<br>• Dysuria<br>• Recurrent urinary tract infection |
| **Medical complaints** | • Anorexia<br>• Abdominal pain<br>• Headaches<br>• Enuresis, encopresis<br>• Chronic constipation<br>• Painful defecation<br>• Bruises to the soft or hard palate |
| **Behavioral changes** | • Clinging<br>• Temper tantrums, aggression, self injury<br>• Sleep disturbance, nightmares<br>• Appetite disturbance<br>• Neurotic or conduct disorders<br>• Excessive fears, phobias<br>• Depression, withdrawal, low self-esteem<br>• Social problems with peers<br>• School problems<br>• Substance abuse<br>• Suicidal ideation<br>• Explicit description of sexual contact*<br>• Promiscuity, prostitution, sexual perpetration on others*<br>• Inappropriate knowledge of adult sexual behavior*<br>• Excessive sexual curiosity, sexual acting out*<br>• Compulsive masturbation* |
| *Specific, significant indicators of possible sexual abuse | |

*Hymel, KP, Jenny, C. Child sexual abuse. Pediatr Rev 1996; 17:236 and Ludwig, S. Child abuse. In: Textbook of Pediatric Emergency Medicine, 4th ed, Fleisher, GR, Ludwig, S (Eds), Lippincott, Williams & Wilkins, Philadelphia 2000. p.1669.*


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# IX. Substance Use Disorders

### A. Substance Use Disorder

• Use of substances that leads to impairment or distress
• Not necessarily dependent

| DSM-5 Diagnostic Criteria for Substance Use Disorder |
|---|
| **A problematic pattern of use leading to clinically significant impairment or distress is manifested by two or more of the following within a 12-month period:** |
| • Often taken in larger amounts or over a longer period than was intended |
| • A persistent desire or unsuccessful efforts to cut down or control use |
| • A great deal of time is spent in activities necessary to obtain, use, or recover from the substance's effects |
| • Craving or a strong desire or urge to use the substance |
| • Recurrent use resulting in a failure to fulfill major role obligations at work, school, or home |
| • Continued use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by its effects |
| • Important social, occupational, or recreational activities are given up or reduced because of use |
| • Recurrent use in situations in which it is physically hazardous |
| • Continued use despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance |
| • Tolerance |
| • Withdrawal |
| ***Subtypes of the disorder include:*** |
| • Mild – Two to three criteria |
| • Moderate – Four to five criteria |
| • Severe – Six or more criteria |

Notes


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# X. Suicide

### A. Suicidal Behaviors

Patient
- In all age groups, **male deaths** by suicide outnumber female deaths 4 : 1
- **Women attempt** suicide two to three times more often than men; among girls aged 15–19 years old, there may be 100 attempts to every one attempt among boys of the same age

Comments
- Suicide is the **8th leading cause of death** in the United States and the 3rd leading cause of death in ages 15–24
- On average, primary care physicians encounter > six potentially suicidal people in their practice each year
- Most people who die by suicide were seen by a physician within one year before killing themselves, and about one-third had been under the care of a mental health care practitioner during the preceding year

Notes


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**B. Modified SADPERSONS Scale for Suicide Risk Assessment**

| Characteristic | Score |
|---|---|
| **S**ex (male) | 1 |
| **A**ge (< 19 or > 45) | 1 |
| **D**epression or hopelessness | 2 |
| **P**revious attempts/psychiatric care | 1 |
| **E**xcessive alcohol or drug use | 1 |
| **R**ational thinking loss | 2 |
| **S**eparated, divorced, or widowed | 1 |
| **O**rganized or serious attempt | 2 |
| **N**o social support | 1 |
| **S**tated future intent | 2 |

| Risk Assessment | |
|---|---|
| 0 - 4 | Low |
| 5 - 6 | Medium |
| 7 - 10 | High |

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CHAPTER 12

# Hematology

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CHAPTER 12

# Hematology


505 **ACUTE LEUKEMIA**

507 **ANEMIA**

515 **APLASTIC ANEMIA**

519 **CLOTTING FACTOR DISORDERS**

523 **HEMOLYTIC ANEMIA**

527 **HYPERCOAGULABLE STATES**

529 **LYMPHOMAS**

533 **POLYCYTHEMIA**

534 **SICKLE CELL DISEASE**

537 **THROMBOCYTOPENIA**



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# I. Acute Leukemia

## A. Acute Lymphocytic Leukemia (ALL)

Patient
• Usually a **child**, second peak adults > 60

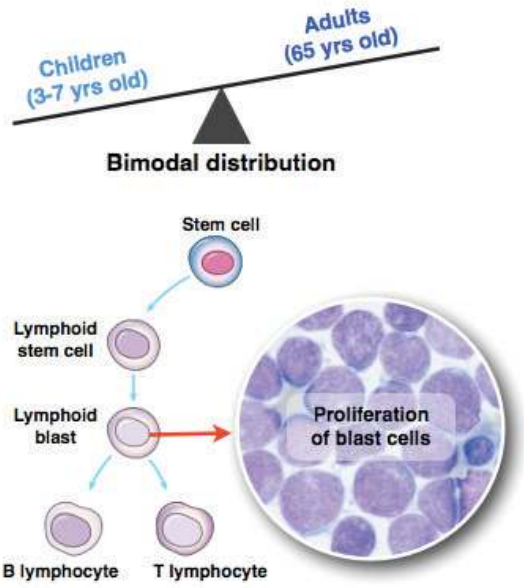
Presentation
• Limping, bone pain, fever, lymphadenopathy

Physical Exam
• Hepatosplenomegaly

Diagnostic Studies
• Anemia, thrombocytopenia and **lymphoblasts** on peripheral smear

Comments
• **Most common** acute leukemia in **children**

**Most common cancer in children**




**Clinical (More common in ALL than AML)**
• Bone pain
• Lymphadenopathy
• Hepatosplenomegaly
• CNS Involvement

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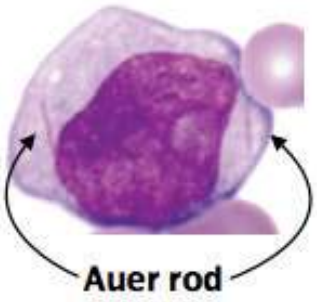
## B. Acute Myeloid Leukemia (AML)

Patient
• Adult

Presentation
• **Fever**, **fatigue**, **anemia**, easy bruising or bleeding, petechiae, bone and joint pain, and persistent or frequent infections

Physical Exam
• Hepatosplenomegaly

Diagnostic Studies
• Normocytic, normochromic anemia and **Auer rods**







**Clinical**
• Constitutional (fatigue, anorexia, weight loss, fever)
• Bone pain
• Lymphadenopathy

**Laboratory**
• Leukocytosis
• Anemia
• Thrombocytopenia

Notes


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# II. Anemia

### A. Causes of Anemia

| Low mean corpuscular volume (microcytic anemia: MCV < 80 fL) |
|---|
| • Iron-deficiency anemia<br>• Thalassemic disorders<br>• Anemia of inflammation/anemia of chronic disease (late; uncommon)<br>• Sideroblastic anemia (e.g., congenital, lead, alcohol, drugs; uncommon)<br>• Copper deficiency, zinc poisoning (rare)<br>• Hemolysis |
| **Normal mean corpuscular volume (normocytic anemia: MCV 80–100 fL)** |
| • Acute blood loss<br>• Iron-deficiency anemia (early)<br>• Anemia of inflammation/anemia of chronic disease (e.g., infection, inflammation, malignancy)<br>• Bone marrow suppression (may also be macrocytic)<br>• Chronic renal insufficiency<br>• Hypothyroidism<br>• Hypopituitarism<br>• Hemolysis |
| **Increased mean corpuscular volume (macrocytic anemia: MCV > 100 fL)** |
| • Excessive ethanol use<br>• Folate deficiency<br>• Vitamin B12 deficiency<br>• Myelodysplastic syndromes<br>• Acute myeloid leukemias<br>• Reticulocytosis<br>• Drug-induced anemia (e.g., hydroxyurea, AZT, chemotherapeutic agents)<br>• Liver disease<br>• Hypothyroidism |

Notes


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## B. Iron Deficiency Anemia

Presentation
• Weakness, dizziness, and fatigue

**Clinical**

| *Symptoms* | *Signs* |
|---|---|
| • Fatigue | • Pallor |
| • Weakness | • Dry or rough skin |
| • Headache | • Blue sclerae |
| • Irritability | • Atrophic glossitis |
| • Exercise intolerance | • Cheilosis |
| • Exertional dyspnea | • Koilonychia |
| • Pica and ice craving | • Esophageal web |

Physical Exam
• Pallor, tachycardia, atrophic **glossitis**, **koilonychia** (spoon-shaped nails)

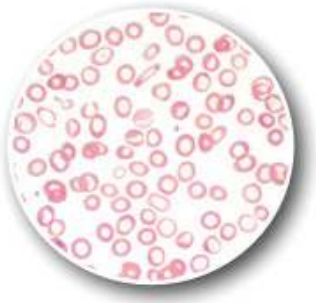
Diagnostic Studies
• **Microcytic**, **hypochromic red blood cells**
• **Decreased** serum **iron** level, **increased** total iron binding capacity (**TIBC**), and decreased serum **ferritin** levels

Comments
• **Most common** cause of anemia

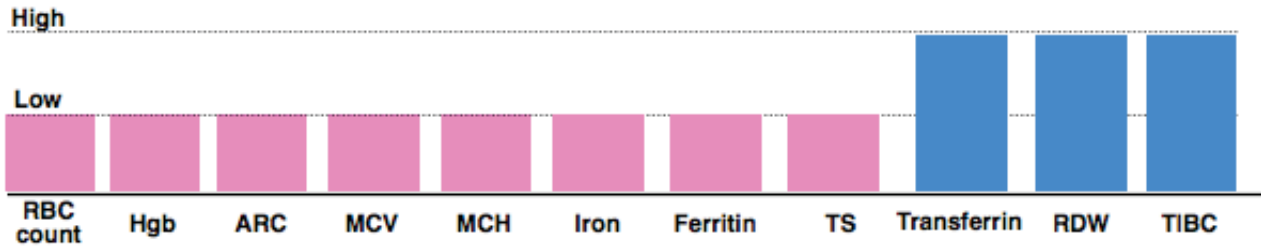

Koilonychia


Atrophic glossitis


Angular cheilosis


Hypochromic, microcytic

**Laboratory**




Hgb: Hemoglobin
ARC: Absolute reticulocyte count
MCV: Mean corpuscular volume
MCH: Mean corpuscular hemoglobin
TS: Transferrin saturation
RDW: Red cell distribution width
TIBC: Total iron binding capacity

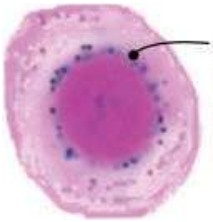
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### C. Sideroblastic Anemia

Pathophysiology
• **Most commonly** caused by an X-linked recessive, **mitochondrial defect** that prevents the incorporation of iron into hemoglobin

Patient
• **Older** than 65 years with a history of prolonged exposure to toxins (ethanol, lead, or isoniazid)



**Ringed sideroblast**
Granules of iron accumulate in the mitochondria surrounding the nucleus

Diagnostic Studies
• Mild anemia with **increased** serum **iron** and **ferritin**
• Total iron-binding capacity (**TIBC**) is normal or **decreased** and **transferrin** is **decreased**
• **Reticulocyte** count will be **low**
• Blood smear will show a **dimorphic** cell population
• Bone marrow exam will show **ringed sideroblasts** when stained with **Prussian blue**

Notes


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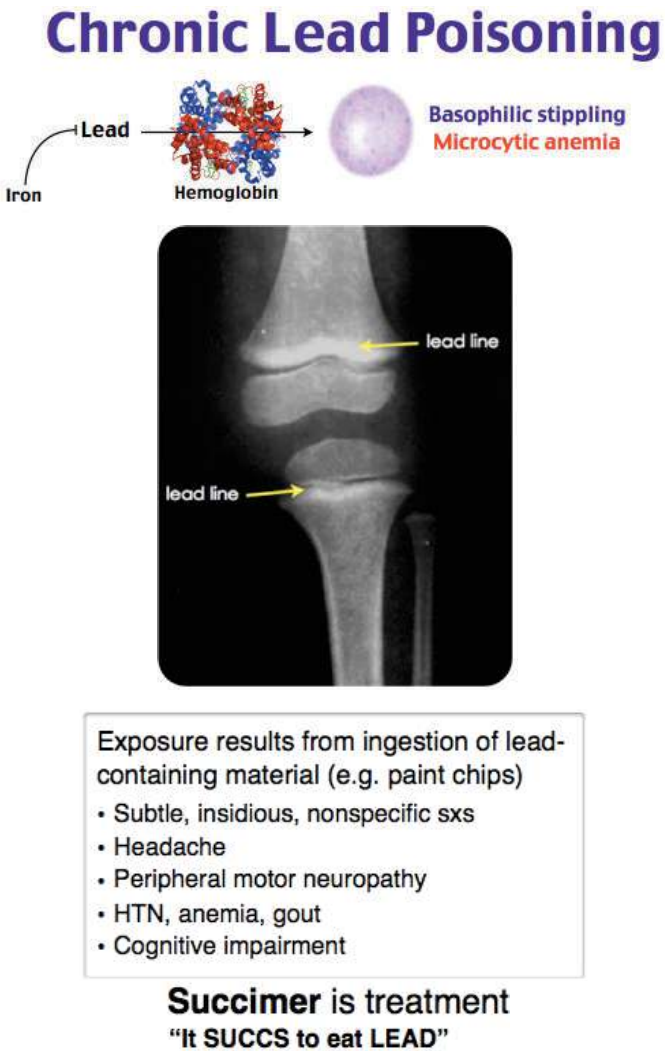
### D. Lead Poisoning

Patient
• Child

Presentation
• **Headache**, **joint pain**, and **constipation**

Diagnostic Studies
• X-ray will show hyperdense lines at metaphyses (**"lead lines"**)
• Labs will show **microcytic**, **hypochromic** anemia and **basophilic stippling** on peripheral smear

Management
• Oral **succimer** or IV **EDTA** (calcium disodium edetate, given after chelating agent)




Notes


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**Lead Poisoning cont.**

# Lead toxicity

**Most** is due to **chronic low-level exposure**.
**Acute toxicity** from **single expose is rare**.



**Adult**

Exposure is primarily **occupational** (e.g. construction, mining, welding, moonshine)





**Child**

Exposure results from **accidental** ingestion of lead-containing material (e.g. paint chips)

**Acute**

- Nausea, vomiting, abdominal pain
- Ataxia
- Encephalopathy, Seizures

**Chronic**

- Subtle, insidious, nonspecific symptoms
- Headache
- Peripheral motor neuropathy
- HTN, anemia, gout
- Cognitive impairment

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**E. Megaloblastic Anemia**

Pathophysiology
• **Most commonly** caused by **vitamin B12** (**cobalamin**) or **folate deficiency**

Patient
• Vegan or alcoholic

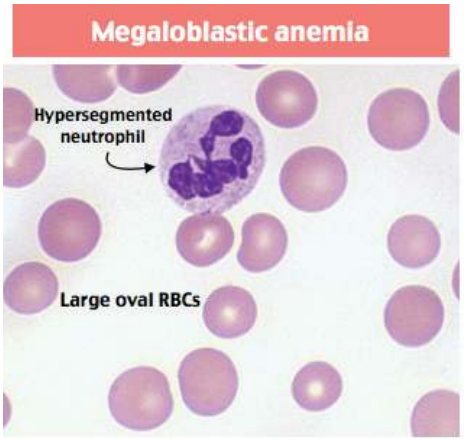
Presentation
• Fatigue, weakness

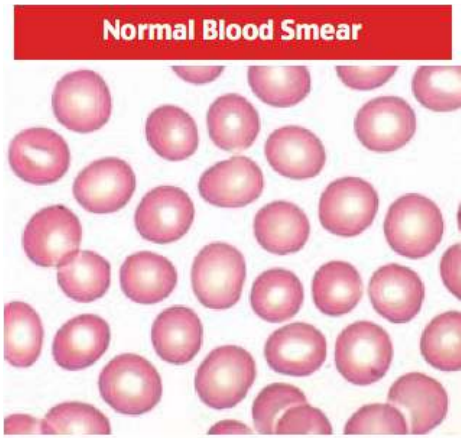
Physical Exam
• Pallor, glossitis

Diagnostic Studies
• **MCV > 100** and **hypersegmented neutrophils**

Management
• Supplement deficient vitamins, usually IM loading then PO

Comments
• **Only vitamin B12 deficiency** results in **neurological symptoms**

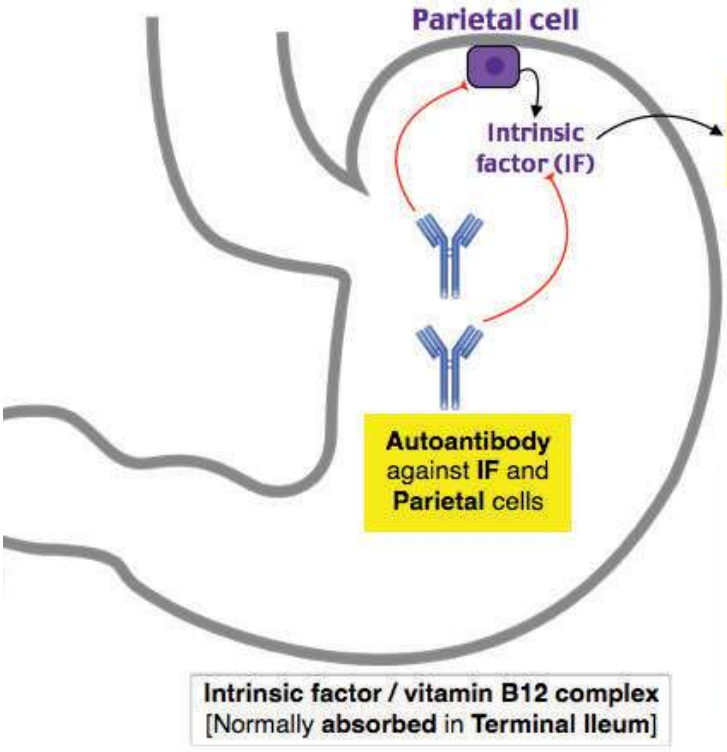



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## F. Vitamin B12 Deficiency




**Intrinsic Factor (IF)** required for **Vitamin B12 absorption**

- **Chronic atrophic gastritis**
- **Vitamin B12 deficiency**
- Increased risk of **gastric cancer**
- Increased risk of **carcinoid tumor**
- **Anemia** (pernicious)

**Clinical**
- Symmetric peripheral neuropathy
- Ataxia
- Personality changes
- Dementia
- Glossitis, vaginal atrophy, malabsportion

Notes


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## G. Anemia of Chronic Disease

Presentation
• Fatigue

Diagnostic Studies
• **Normocytic anemia**, **low** serum **iron**, **high** or normal **ferritin**

| | Iron Deficiency Anemia | Anemia of Chronic Disease | Thalassemia |
|---|---|---|---|
| **MCV** | Low/normal | Low/normal | Low/normal |
| **RDW** | High | Normal | Normal |
| **Iron** | Low | Low | Normal/high |
| **TIBC** | High | Normal/low | Normal |
| **Ferritin** | Low | Normal/high | Normal |
| **Transferrin saturation** | Low | Low/Normal | Normal |
| **Transferrin** | High | Normal | Normal |

MCV = mean corpuscular volume
RDW = red cell distribution width
TIBC = total iron binding capacity

Notes


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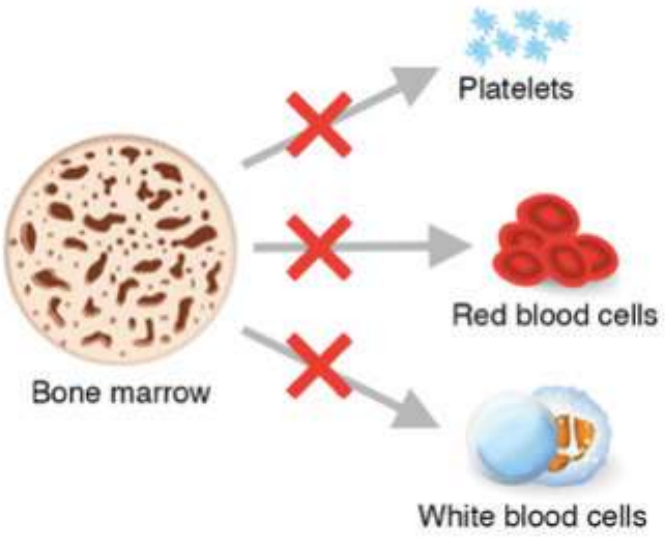
# III. Aplastic Anemia

### A. Aplastic Anemia

Patient
• History of recurrent infections

Presentation
• Mucosal **hemorrhage**, prolonged menstrual bleeding, petechiae, ecchymoses, and fatigue

Diagnostic Studies
• **Peripheral pancytopenia** and **bone marrow hypoplasia** or **aplasia**
• Diagnosis is made by bone marrow aspiration and biopsy

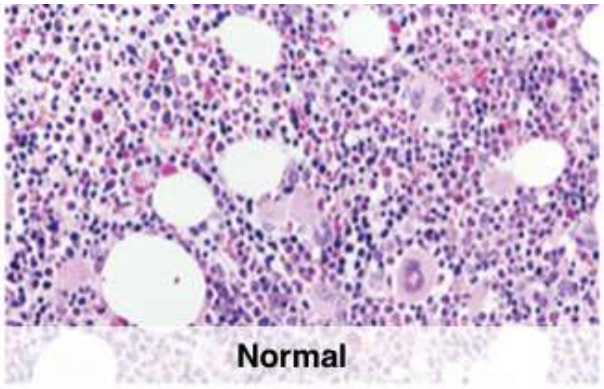


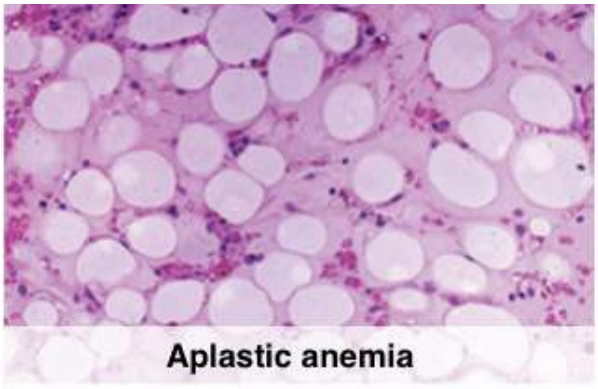

**Pancytopenia + bone marrow hypoplasia/aplasia**

**Loss of hematopoietic stem cells**

**Clinical**
• Recurrent infections (neutropenia)
• Mucosal hemorrhage (thrombocytopenia)
• Fatigue (anemia)

**Etiology**
• Autoimmune mechanisms
• Direct injury to hematopoietic stem cells
• Viral infection
• Clonal and genetic disorders


Normal

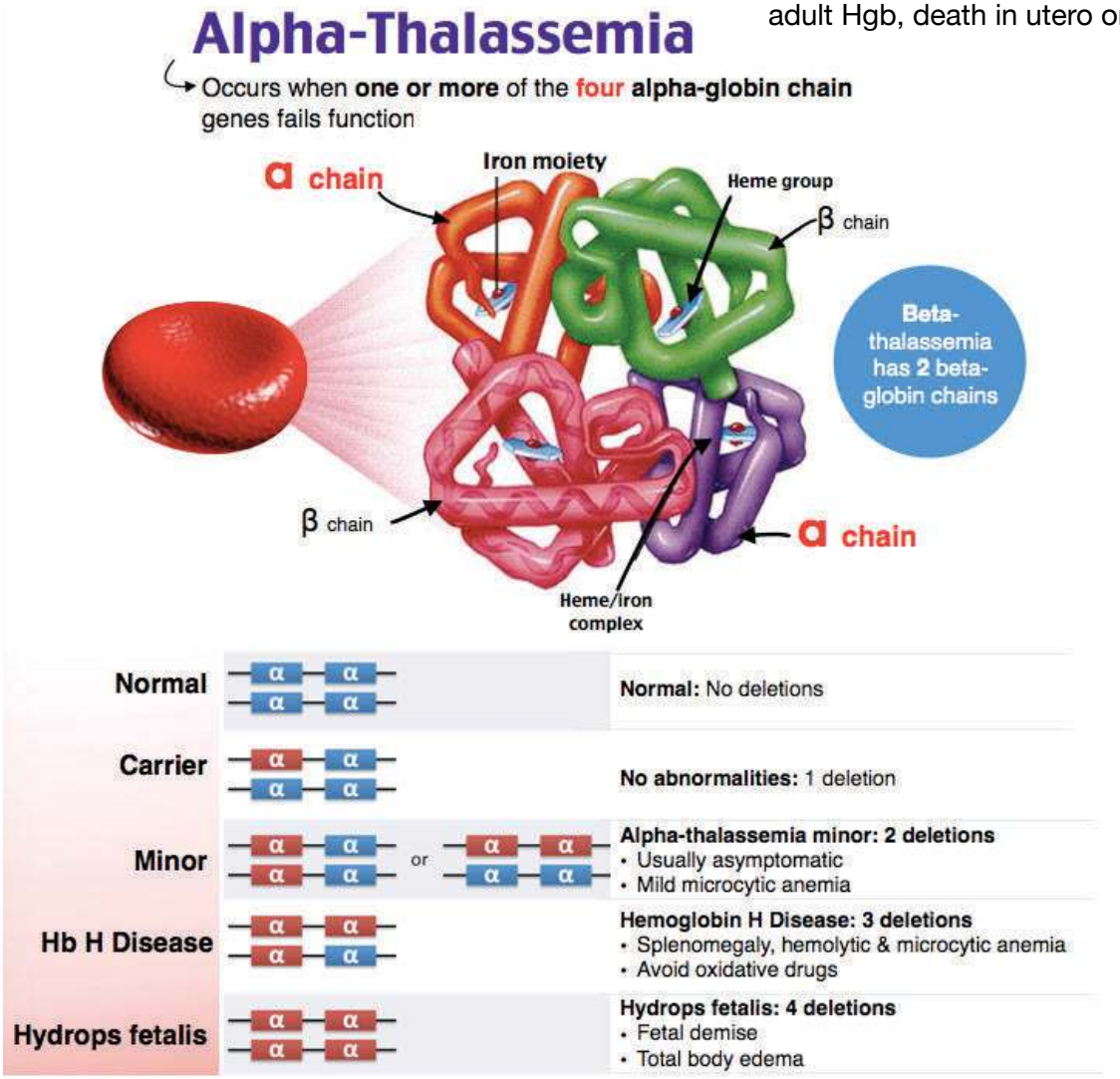

Aplastic anemia

Notes


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### B. Alpha Thalassemia

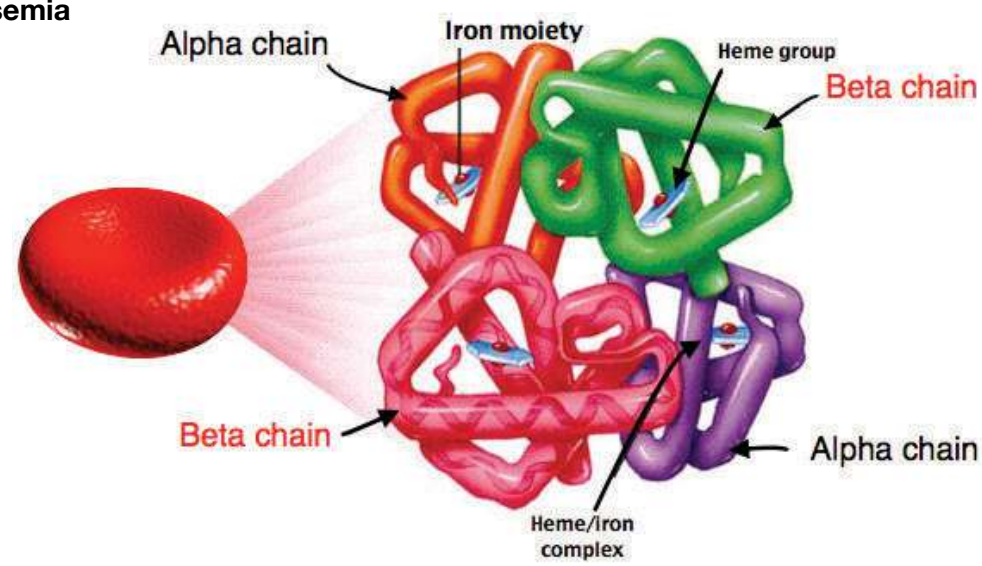
• 1 gene inactive: **silent carrier**; asymptomatic
• 2 genes inactive: **alpha thalassemia trait**; mild anemia, very low MCV, asymptomatic
• 3 genes inactive: **hemoglobin H disease**; marked microcytic, hypochromic anemia, splenomegaly, Hgb H 4–10%
• 4 genes inactive: **hydrops fetalis**; no fetal or adult Hgb, death in utero or neonatally




Notes


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### C. Beta Thalassemia




More common in people from **Asia**, **Mediterranean countries**, **Middle East**, **Africa**, and **Asia**

**MAJOR (Cooley Anemia)**

- Mutation in two beta chains
- Profound microcytic hypochromic anemia
- Requires lifelong blood transfusions
- Complications (splenomegaly, cholelithiasis, hemosiderosis)

**INTERMEDIA**

- Based on clinical observation
- Requires occasional transfusions

**MINOR**

- Mutation in one beta chain (Carrier state)
- Mild laboratory anemia
- Asymptomatic

Degree of severity

Notes


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### D. Beta Thalassemia Minor

Diagnostic Studies
• Microcytic and hypochromic cells, **increased Hgb A2**, **target cells**

Management
• Genetic counseling, avoid iron

### E. Beta Thalassemia Major (Cooley anemia)

Patient
• History of **failure to thrive**

Physical Exam
• Hepatosplenomegaly

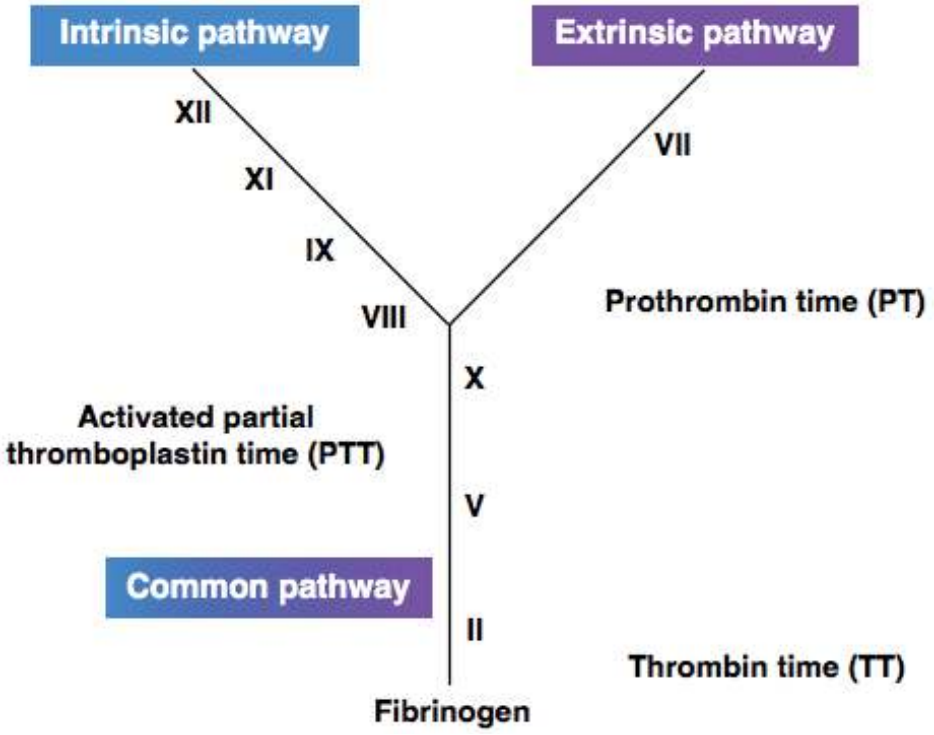
Diagnostic Studies
• Severe anemia (Hgb about 6 g/dL), **decreased MCV**, **increased Hgb F**

Management
• Transfusions, splenectomy, iron chelation

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# IV. Clotting Factor Disorders




Notes


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**A. Hemophilia A**

Pathophysiology
• **Most commonly** caused by X-linked recessive **factor VIII deficiency**

Patient
• Child

Presentation
• Easy bruising or **hemarthroses**

Diagnostic Studies
• **Increased** partial thromboplastin time (**PTT**)



**Hemophilia A** *("A"ight)*
- X-linked recessive
- Decreased synthesis of Factor VIII (8)
- Treat with recombinant Factor VIII

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**B. Hemophilia B (Christmas Disease)**

Pathophysiology
• **Most commonly** caused by X-linked recessive **factor IX deficiency**

Patient
• History of **minor trauma** causing **large amounts of bleeding** or **hemarthroses**

Presentation
• **Spontaneous bleeding episodes** involving skin, mucous membranes, and joints

Diagnostic Studies
• **Prolonged** activated partial thromboplastin time (**aPTT**) and **normal** prothrombin time (**PT**)

Management
• Exogenous **factor IX** concentrate



**Hemophilia B**
- X-linked recessive
- Decreased synthesis of Factor IX (9)
- Treat with recombinant Factor IX
- Christmas disease

Notes


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### C. Von Willebrand Disease

Pathophysiology
• **Autosomal dominant**

Patient
• History of a **parent** with similar symptoms

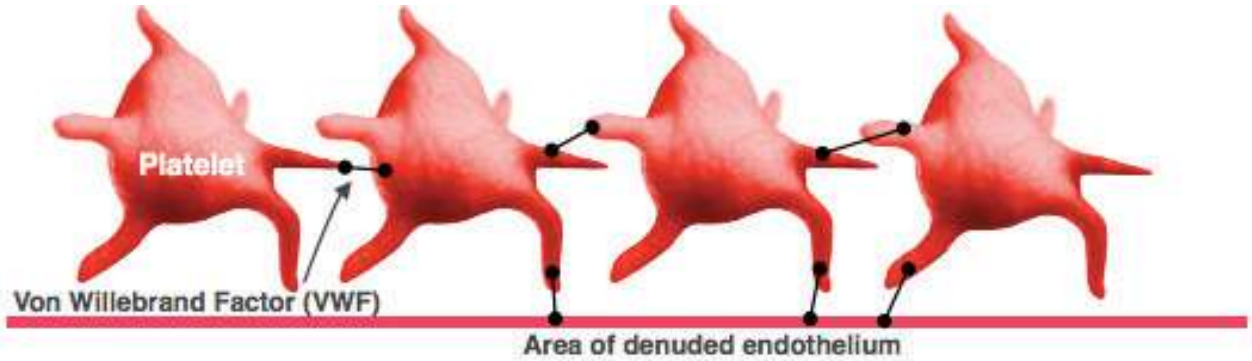
Presentation
• **Mucosal hemorrhage** or **bleeding** that is **difficult to control**

**Most common inherited bleeding disorder**

Diagnostic Studies
• **Decreased** factor VIII activity, **prolonged bleeding time**, normal PT/INR, normal aPTT (unless very low VIII activity)

Management
• Desmopressin (**DDAVP**)

Comments
• **Most common inherited** bleeding disorder




**Von Willebrand Factor (VWF)**
• Important role in primary hemostasis
• Binds platelets
• Binds endothelial components
• Forms adhesive bridge b/w platelets and vascular subendothelial structures
• Contributes to fibrin clot formation (carries factor VIII)

**Treatment**
• Desmopressin (DDAVP)

**Clinical**
• Easy bruising
• Skin bleeding
• Prolonged bleeding from mucosal surfaces (oropharyngeal, GI, uterine)

**Laboratory**
• Normal platelet count (mild thrombocytopenia in type 2B
• Normal PT/INR
• Normal aPTT (prolonged if very low factor VIII)

Notes


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# V. Hemolytic Anemia

### A. Hemolytic Anemia

| | Extravascular | Intravascular |
|---|---|---|
| **Etiologies** | • Warm autoimmune hemolytic anemia<br>• Hypersplenism<br>• Delayed hemolytic transfusion reaction<br>• Hemoglobinopathies | • Cold autoimmune hemolytic anemia<br>• Acute hemolytic transfusion reaction<br>• Microangiopathic hemolysis<br>• G6PD deficiency<br>• Paroxysmal nocturnal hemoglobinuria<br>• Hemoglobinopathies |
| **Site of RBC destruction** | Macrophage (spleen, liver) | Blood vessels |
| **Haptoglobin** | Low | Very Low |
| **LDH** | High | Very high |
| **Urine hemosiderin** | Negative | Positive |
| **Urine hemoglobin** | Negative | Positive (in severe cases) |
| **Peripheral smear** | Spherocytes | Schistocytes |

Notes


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## B. Transient Aplastic Crisis

Pathophysiology
• **Most commonly** caused by recent **parvovirus B19** infection

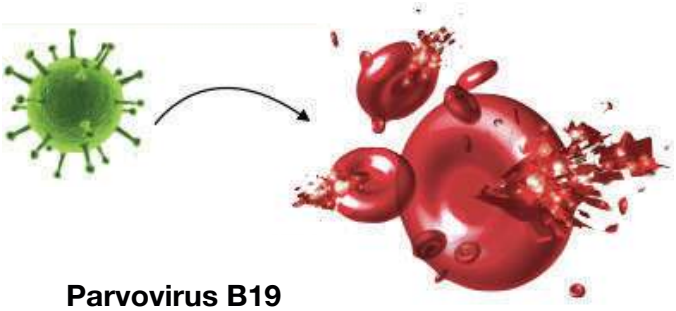
Patient
• History of sickle cell anemia, iron deficiency anemia, or hereditary spherocytosis

Presentation
• Symptoms of **anemia**: **pallor**, **fatigue**, **lethargy**, and shortness of breath

Diagnostic Studies
• **Undetectable** reticulocyte count and a **drop** in hemoglobin concentration **> 30%**

**Transient Aplastic Crisis** – Temporary **suspension of erythropoiesis** leads to **severe anemia**

**At-risk populations**
• Sickle cell disease
• Hereditary spherocytosis
• Iron-deficiency anemia



**Parvovirus B19**

1. Invades RBC precursors
2. Multiplies and destroys
3. Causes a drop in hemoglobin (undetectable reticulocyte count)

**Clinical**
• Pallor
• Weakness
• Lethargy
• Typically no rash

Notes


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### C. Hereditary Spherocytosis

Pathophysiology
• **Most commonly** caused by an **autosomal dominant** genetic disease

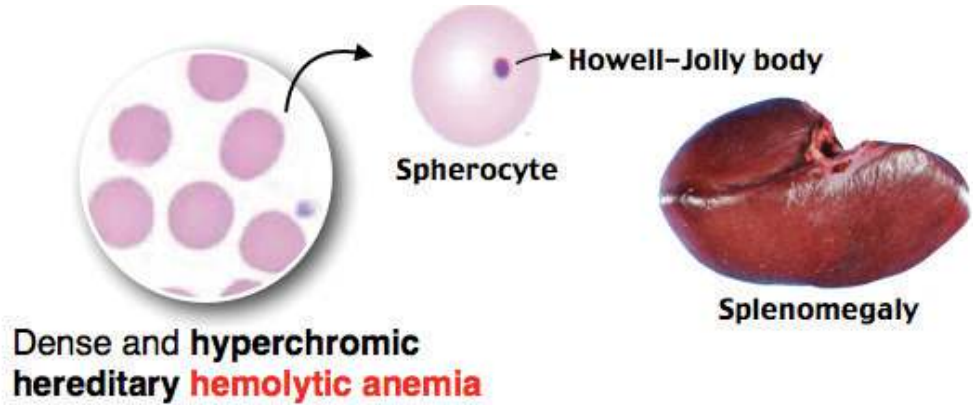
Patient
• Positive family history

Presentation
• Symptoms of **anemia**: **pallor**, **fatigue**, **lethargy**, and shortness of breath

Diagnostic Studies
• MCV low or normal, **increased** **MCHC**, **elevated** reticulocyte count, **elevated** indirect bilirubin
• **Only disorder** that will cause an **increase** in mean corpuscular hemoglobin concentration (**MCHC**)
• Peripheral smear will show **spherocytes** and **Howell-Jolly bodies**

Management
• Daily **folic acid**

**Hereditary Spherocytosis**
**Autosomal dominant**




Notes


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## D. G6PD Deficiency

X-linked

Coombs test NEGATIVE (Nonimmune)

Pathophysiology
• **X-linked recessive**

Patient
• History of taking **antimalarials**, **sulfonamides**, **nitrofurantoin**, **fava beans**

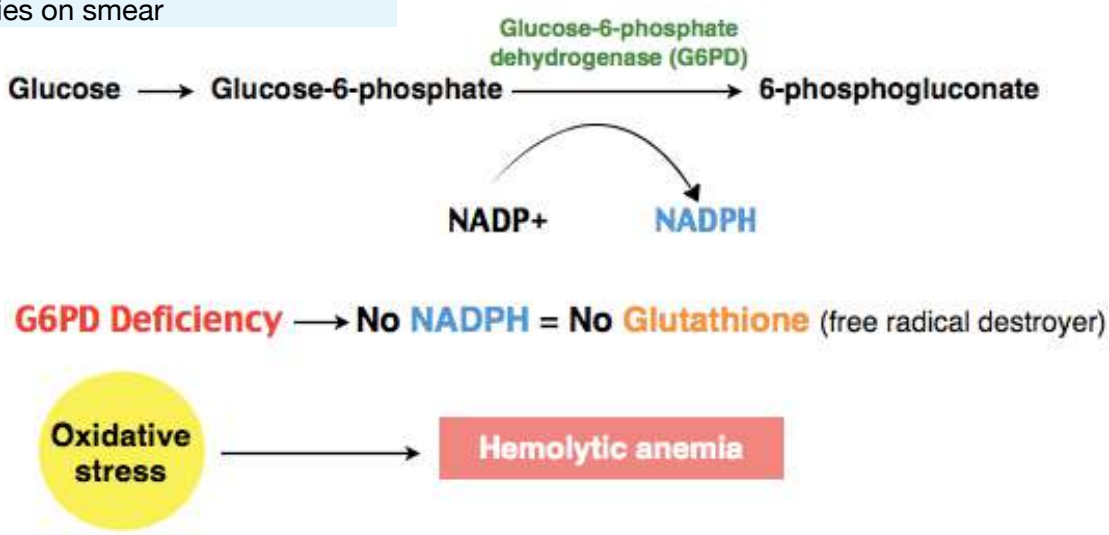
Diagnostic Studies
• **Heinz bodies** on peripheral smear



• Causes a nonimmune hemolytic anemia (infection, medications, Fava beans)
• **Protects against malaria**
• Heinz bodies on smear

**G6PD Deficiency**
**Glucose-6-phosphate dehydrogenase**

**Heinz Body**
• Inclusion within RBC
• Composed of denatured hemoglobin
• Formed as a result of oxidative damage
• Macrophages in the spleen remove the denatured hemoglobin
• Results in bite cells




Notes


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# VI. Hypercoagulable States

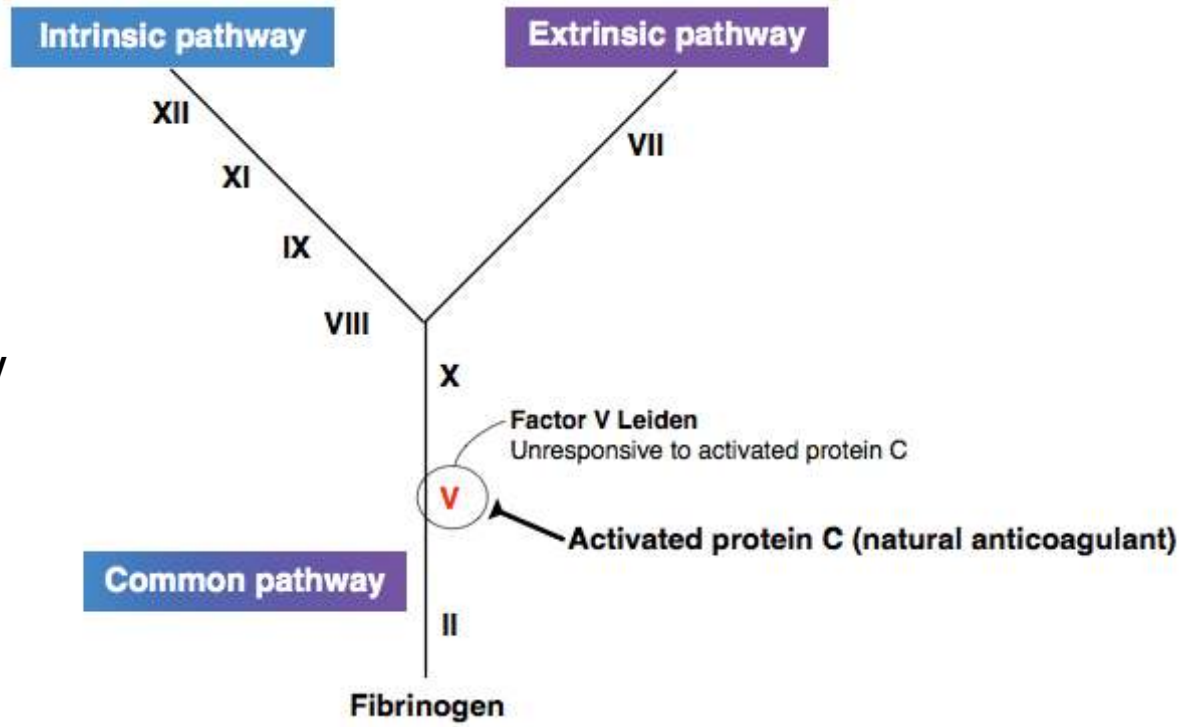
### A. Factor V Leiden

Pathophysiology
• **Most commonly** caused by an **autosomal dominant** genetic disorder

Comments
• **Most common** inherited hypercoagulability disorder

**Factor V Leiden**
• Results from a single point mutation in the factor V gene
• Mutation renders factor V insensitive to the actions of activated protein C (aPC)
• Individual becomes hypercoagulable (increased risk for venous thromboembolism)

**Factor V Leiden**
**Defective factor V**




Notes


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## B. Antiphospholipid Antibody Syndrome

Pathophysiology
- **Autoimmune** disorder
- Arterial, venous, or small vessel thromboembolic events

Patient
- History of lupus or other rheumatic diseases

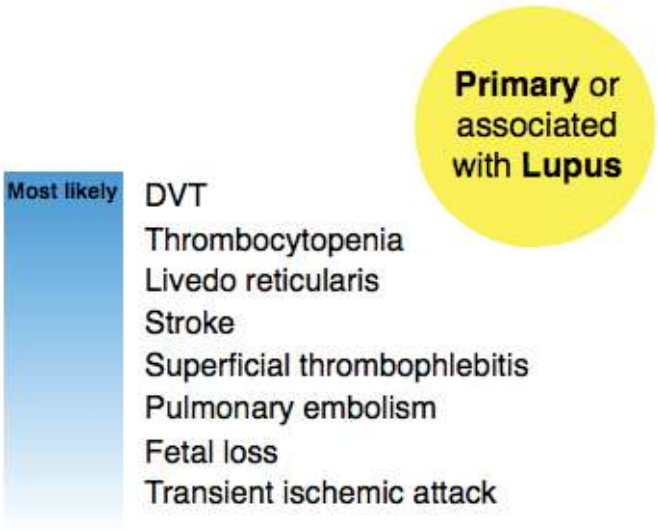
Presentation
- **Repeated spontaneous abortions**

Diagnostic Studies
- **Thrombocytopenia**

Management
- Treatment is anticoagulation

### Antiphospholipid Antibody Syndrome

**An autoimmune multisystem disorder characterized by arterial, venous, or small vessel thromboembolic events**




**Diagnosis**
- Anticardiolipin antibodies
- Anti-beta 2-glycoprotein I antibodies
- Lupus anticoagulant

Notes


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# VII. Lymphomas

### A. Burkitt Lymphoma

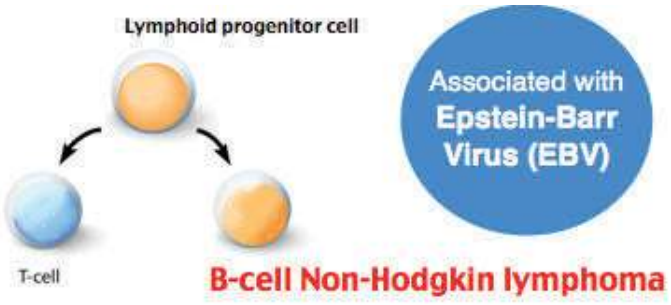
Pathophysiology
• B cell non-Hodgkin lymphoma

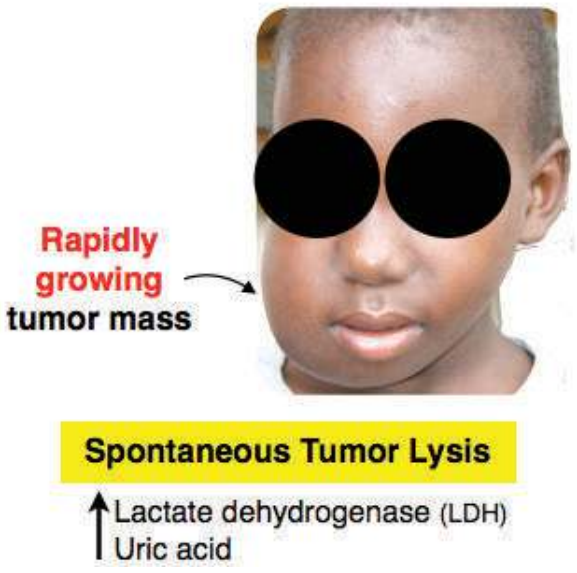
Patient
• History of **EBV**

Presentation
• Enlarging mass over jaw

Diagnostic Studies
• **"Starry sky"** pattern seen on pathology

Management
• Chemotherapy; monitor for tumor lysis syndrome



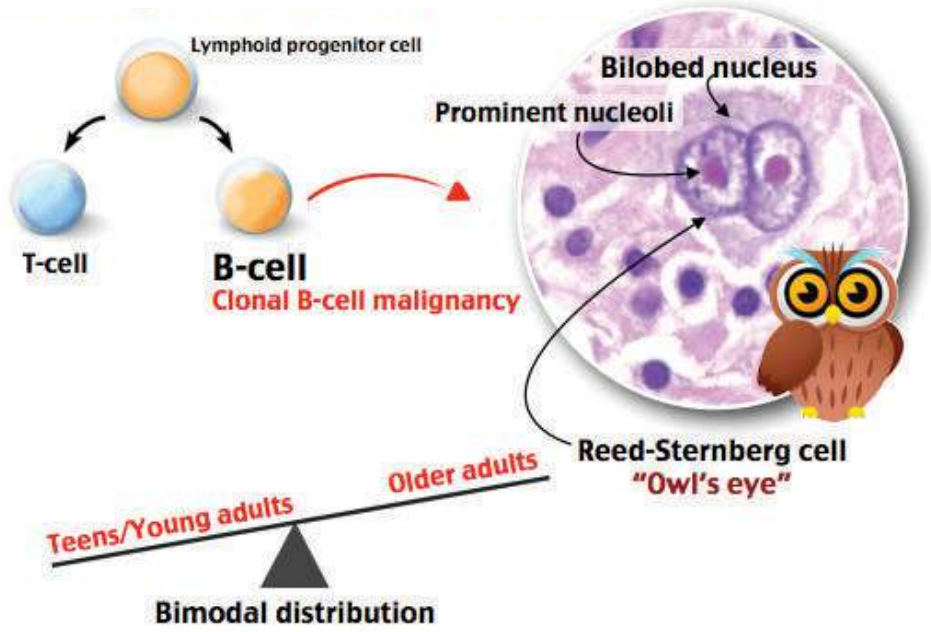

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## B. Hodgkin Lymphoma

Presentation
• **Supraclavicular** and **painless** lymphadenopathy, pruritus, fever, night sweats, unintentional weight loss, and frequent infections

Diagnostic Studies
• Lymph node biopsy will reveal **Reed-Sternberg cells**




**Clinical**
• Non-tender enlarged cervical lymphadenopathy
• Mediastinal mass on chest X-ray
• Hepatosplenomegaly
• Pruritus

**B symptoms**
• Fever
• Night sweats
• Weight loss

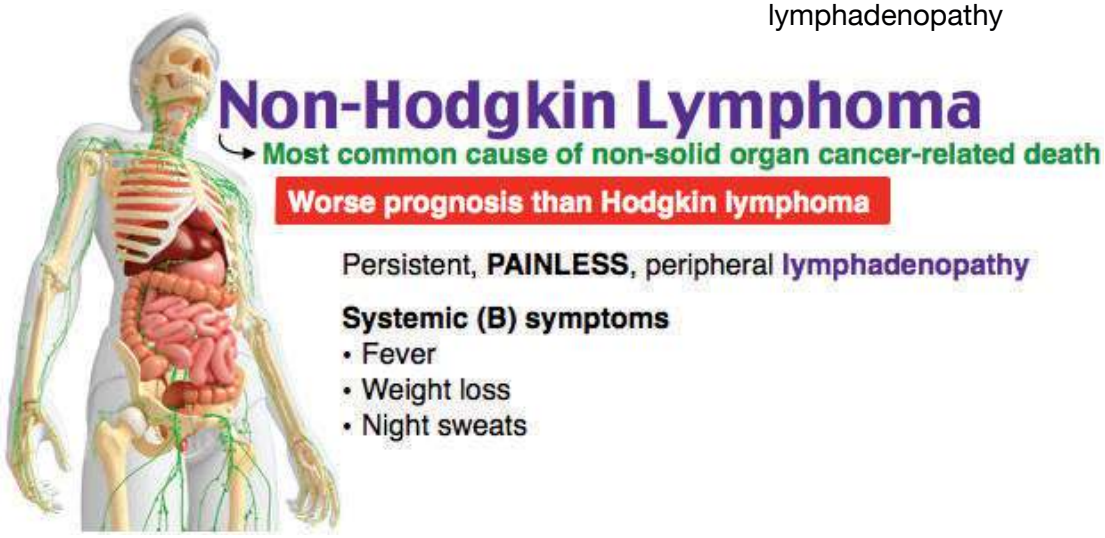
Notes


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### C. Non-Hodgkin Lymphoma

Patient
• Immunocompromised (**HIV**)

Presentation
• Systemic B symptoms (fever, night sweats, weight loss) and **painless** peripheral lymphadenopathy




Notes


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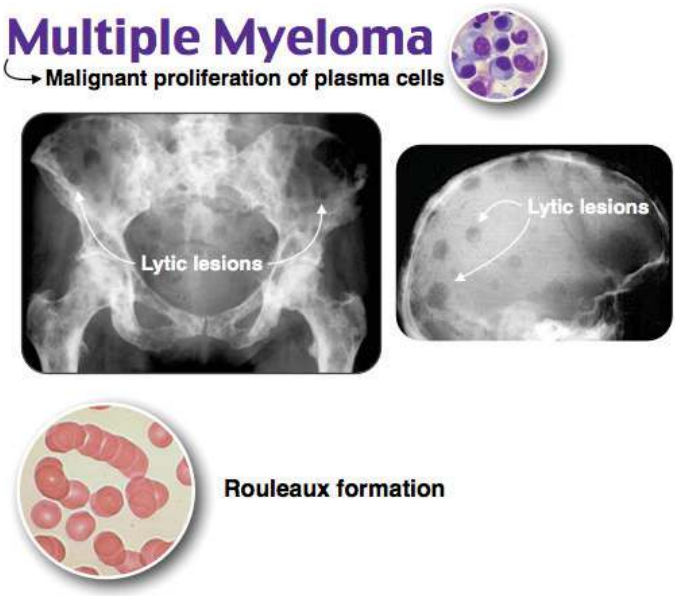
## D. Multiple Myeloma

Patient
• Elderly

Presentation
• Back pain

Physical Exam
• **CRABB**: **hyperC**alcemia, **R**enal insufficiency, **A**nemia, lytic **B**one lesions/**B**ack pain

Diagnostic Studies
• **Rouleaux** formation on peripheral blood smear
• Serum protein electrophoresis: **M spike**
• Urine protein electrophoresis: **Bence-Jones proteins**
• X-ray will show **lytic lesions**




**Clinical**
• Bone pain (most common...back and ribs)
• Spinal cord compression
• Hypogammaglobulinemia (leading to sepsis)
• Renal failure (most commonly due to hypercalcemia)
• Anemia/Bleeding
• Hyperviscosity syndrome

Notes


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# VIII. Polycythemia

### A. Polycythemia Vera

Pathophysiology
• **Most commonly** caused by mutation of the Janus kinase 2 gene (***JAK2***)

Presentation
• Headache, dizziness, **pruritus after showering**

Physical Exam
• Hypertension, **splenomegaly**

Diagnostic Studies
• **Increased RBC mass**, **overproduction of all cell lines**, **Increased Hgb**

Management
• Phlebotomy, hydroxyurea, aspirin

**Clinical**
• Fatigue
• Pruritus
• Facial plethora (ruddy cyanosis)
• Erythromelalgia (burning pain and erythema, pallor or cyanosis)
• Engorged retinal veins
• Thrombosis
• Gouty arthritis

**Polycythemia Vera**
**Chronic Myeloproliferative Neoplasm (*JAK2* mutation)**






Hemoglobin > 18.5 g/dL in men
Hemoglobin > 16.5 g/dL in women



**Aquagenic Pruritus**
**Pruritus** following a **warm bath** or **shower**

**Treatment**
• Hydroxyurea
• Phlebotomy
• Aspirin

Notes


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# IX. Sickle Cell Disease

### A. Sickle Cell Disease

Pathophysiology
- **Decreased** oxygen, dehydration, and acidosis leads to **sickling**
- **Sickling** leads to **vaso-occlusive ischemia**
- Newborns: initially asymptomatic (due to **increased HbF**)
- Dysfunctional spleen **increases** infection risk
  - ***Salmonella*** osteomyelitis: **most common** cause in sickle cell patients
  - ***S. pneumoniae*** sepsis: **most common** cause of death in children

Presentation
- **Most common** presentation in infants: **dactylitis**
- Aseptic necrosis of the femoral head
- **Acute chest syndrome**:
  - **most common cause of death in adults**
  - fever, CP
  - Chest X-ray: pulmonary infiltrate
- **Splenic sequestration crisis**: rapid splenic sequestration of RBCs leads to splenomegaly and severe anemia

Management
- Opioid protocol for acute pain crisis
- **Hydroxyurea** to reduce frequency of crises

Notes


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### B. Common Complications in Sickle Cell Disease

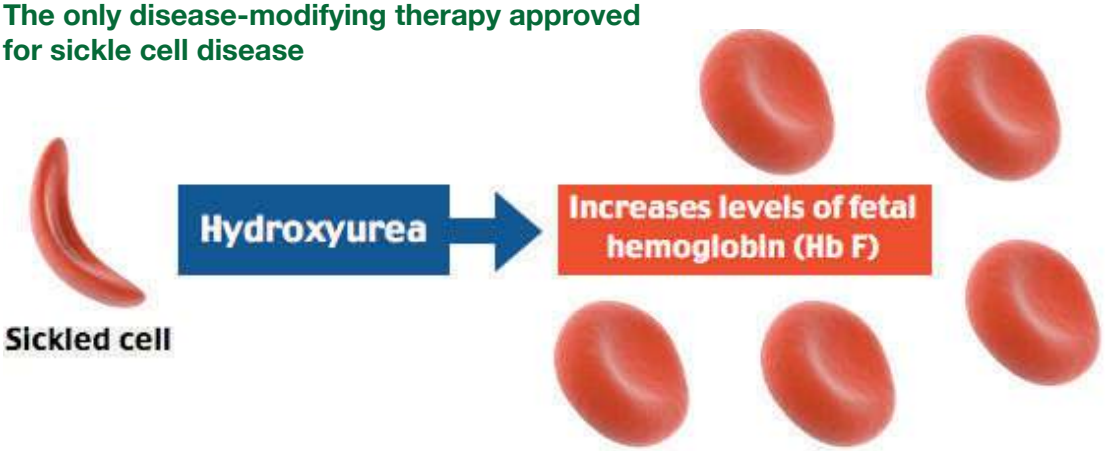
| Complication | Clinical | Diagnosis | Management |
|---|---|---|---|
| **Vaso-occlusive pain crisis** | • Pain in ribs, back, limbs<br>• Pain may last 5 to 7 days | • Clinical<br>• CBC/Retic cnt to eval for aplastic crisis | • Supportive<br>• Hydroxyurea |
| **Acute chest syndrome** | • Fever, cough, chest pain<br>• Hypoxia, lung consolidation | • CXR: New pulmonary infiltrate | • Support<br>• Antibiotics<br>• Exchange transfusion |
| **Splenic sequestration crisis** | • Abdominal pain<br>• Pallor, shock<br>• Splenomegaly | • Very low hemoglobin<br>• Reticulocytosis | • Supportive care<br>• PRBC transfusion<br>• Splenectomy |
| **Aplastic crisis** | • Pallor, shock<br>• Lethargy | • Very low hemoglobin<br>• No reticulocytosis | • Supportive<br>• PRBC transfusion |
| **Stroke** | • Aphasia, hemiparesis<br>• Vision change | • CT/MRI | • Supportive<br>• Exchange transfusion |
| **Leg ulcers** | • Over malleoli | • Clinical | • Local wound care |
| **Sepsis (Encapsulated organisms)** | • Meningitis<br>• Osteomyelitis<br>• Pneumonia | • BCX<br>• CXR<br>• UCX | • Supportive<br>• Antibiotics |
| **Cholelithiasis (Biliary stone from increased hemolysis)** | • RUQ pain | • Clinical<br>• Ultrasound | • Supportive<br>• Elective cholecystectomy |
| **Osteonecrosis** | • Hip pain<br>• Shoulder pain | • X-ray | • Supportive<br>• Arthroplasty |
| **Priapism** | • Painful erection | • Clinical | • Terbutaline<br>• Exchange transfusion<br>• Urology consultation |

Notes


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### C. Hydroxyurea

**The only disease-modifying therapy approved for sickle cell disease**




- Reduces the frequency of painful crises
- Reduces the need for blood transfusions

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# X. Thrombocytopenia

## A. Primary Immune Thrombocytopenia (Formerly ITP)

Pathophysiology
• **Most commonly** caused by **antiplatelet antibodies**

Patient
• Child, **2–6 years old**
• History of recent viral infection

Presentation
• Red spots on skin or easy bleeding

Physical Exam
• **Petechiae**, **purpura**, and **gingival bleeding**

Diagnostic Studies
• Platelets **< 50,000/µL**

Management
• Observation, steroids, IVIG

**Children**
**(2–6 yrs old)**
• Most often **acute**
• Often follows a **viral infection**

**Adults**
**(20–50 yrs old)**
• Most often **chronic**

**Primary Immune Thrombocytopenia**
**Formerly Idiopathic thrombocytopenic purpura**



**Clinical**
• Petechiae
• Gingival bleeding
• Epistaxis
• Menorrhagia
• GI bleeding
• Intracranial hemorrhage

**Management**
• Supportive (children)
• Platelets (only for severe bleeding or extremely low platelet count)
• Corticosteroids
• IVIG or anti-D immunoglobin
• Splenectomy

Notes


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### B. Thrombotic Thrombocytopenic Purpura (TTP)

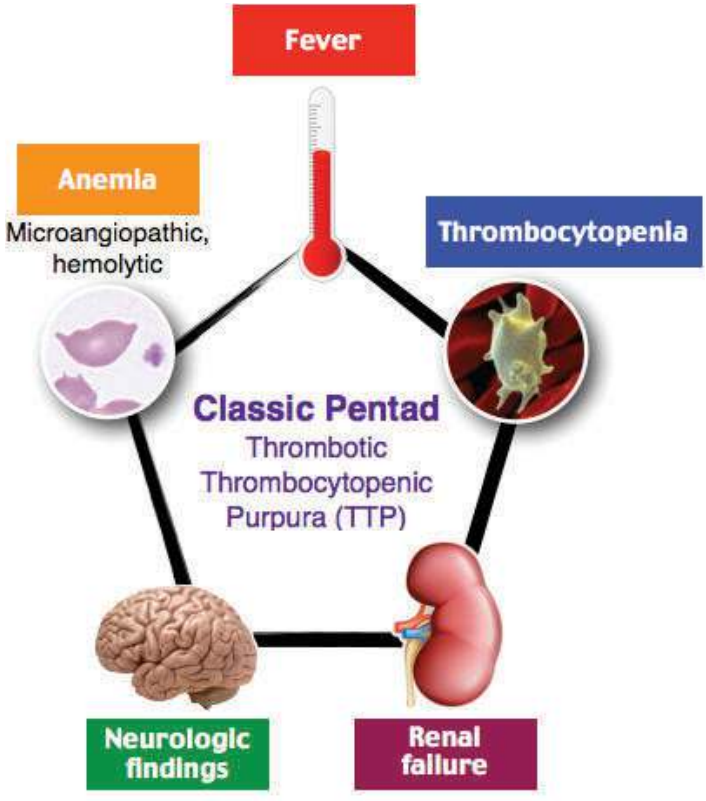
Pathophysiology
• **Most commonly** caused by severely decreased protease **ADAMTS13** activity

Patient
• Woman

Presentation
• Fever, **confusion**, difficulty speaking, **headache**, seizure, nausea, **vomiting**, diarrhea, renal failure

Diagnostic Studies
• **Elevated LDH**, **elevated indirect bilirubin**, normal coagulation studies, and thrombocytopenia
• **Microangiopathic hemolytic anemia** and **thrombocytopenia**

Management
• Plasma exchange with intravenous corticosteroids




Notes

**Causes**
Pregnancy, AIDS, Lupus, Scleroderma, Sjögren syndrome, Cyclosporin, quinidine, tacrolimus

**Management**
• Treat prior to definitive diagnosis
• Plasmapheresis
• Hematology consultation


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### C. Heparin-Induced Thrombocytopenia (HIT)

Patient
• History of recently starting **heparin**

Diagnostic Studies
• **50% decrease in platelets**

Management
• Stop heparin, use direct thrombin inhibitor

Comments
• Type 1: Platelet recovery with or without heparin cessation
• Type 2: Autoimmune, venous and arterial thrombosis

Notes


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### D. Pretest Probability Scoring for Heparin-Induced Thrombocytopenia (The 4T Score)

| Category | 2 Points | 1 Point | 0 Points |
|---|---|---|---|
| **Thrombocytopenia** | • Platelet count fall > 50% **AND**<br>• Platelet nadir ≥ 20,000 | • Platelet count fall 30%–50% **OR**<br>• Platelet nadir 10–19,000 | • Platelet count fall < 30% **OR**<br>• Platelet nadir < 10,000 |
| **Timing of platelet count fall** | • Clear onset between days 5 and 10 **OR**<br>• Platelet fall ≤ 1 day (prior heparin exposure w/in 30 days) | • Consistent with days 5–10 fall, but not clear or onset after day 10 **OR**<br>• Fall ≤ 1 day (prior heparin exposure 30–100 days ago) | • Platelet count fall < 4 days without recent heparin exposure |
| **Thrombosis or other sequelae** | New thrombosis or skin necrosis at heparin injection sites or acute systemic reaction after intravenous heparin bolus | Progressive or recurrent thrombosis or nonnecrotizing (erythematous) skin lesions or suspected thrombosis | None |
| **Other causes for thrombocytopenia** | None apparent | Possible | Definite |
| **Total Points** | **Probability of Heparin-Induced Thrombocytopenia** | | |
| **≤ 3** | Low probability | | |
| **4–5** | Intermediate probability | | |
| **≥ 6** | High probability | | |

Adapted from: Lo GK, Juhl D, Warkentin TE, Sigouin CS, Eichler P, Greinacher A. Evaluation of pretest clinical score (4 T's) for the diagnosis of heparin-induced thrombocytopenia in two clinical settings. *J Thromb Haemost* 2006; 4: 759–65.

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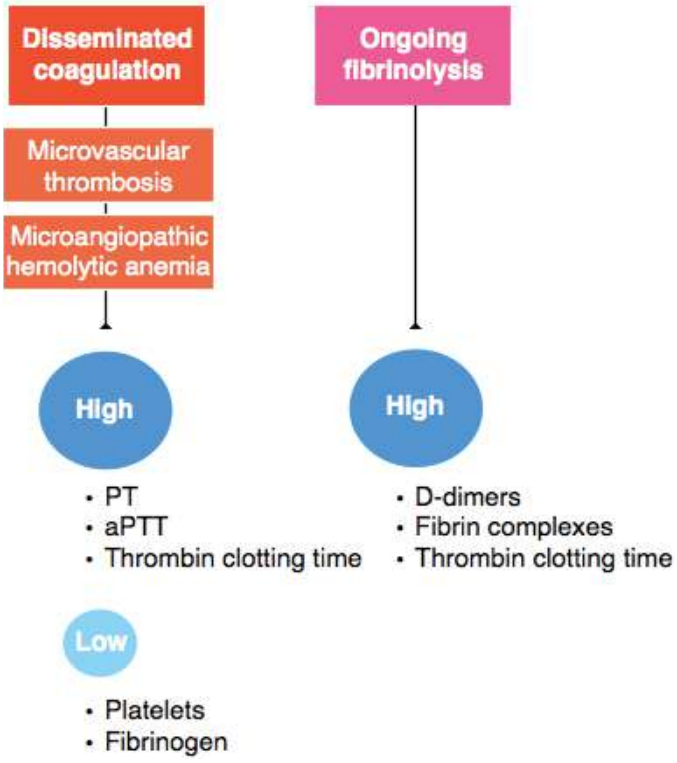
### E. Disseminated Intravascular Coagulation (DIC)

Pathophysiology
• **Most commonly** caused by **inappropriate activation of the coagulation system** by a massive release of tissue factor into the circulation

Patient
• History of sepsis, trauma, or obstetric complications

Diagnostic Studies
• Thrombocytopenia, **decreased fibrinogen**, **increased fibrin split products**, and **increased PT and PTT**

Management
• Treatment of the underlying condition
• If bleeding complications are present, replete clotting factors and platelets with fresh frozen plasma and platelets




Notes


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# Abbreviations

| | |
|---|---|
| **BB** | Beta Blocker |
| **CCB** | Calcium Channel Blocker |
| **HTN** | Hypertension |
| **BP** | Blood Pressure |
| **HR** | Heart Rate |
| **ACE** | Angiotensin-Converting Enzyme |
| **ACEI** | Angiotensin-Converting Enzyme Inhibitor |
| **ARB** | Angiotensin II Receptor Blocker |
| **CKD** | Chronic Kidney Disease |
| **MI** | Myocardial Infarction |
| **HDL** | High-Density Lipoprotein |
| **LDL** | Low-Density Lipoprotein |
| **ECG** | Electrocardiogram |
| **JVD** | Jugular Venous Distension |
| **HTN** | Hypertension |
| **CHF** | Congestive Heart Failure |
| **EF** | Ejection Fraction |
| **LLSB** | Lower Left Sternal Border |
| **RUQ** | Right Upper Quadrant |
| **CT** | Computerized Tomography |
| **AAA** | Abdominal Aortic Aneurysm |
| **CTA** | Computerized Tomography Angiography |
| **MRA** | Magnetic Resonance Angiography |
| **MRI** | Magnetic Resonance Imaging |
| **AVM** | Arteriovenous Malformation |
| **ESR** | Erythrocyte Sedimentation Rate |
| **ASAP** | As Soon As Possible |
| **CRP** | C-Reactive Protein |
| **DVT** | Deep Vein Thrombosis |
| **NSAID** | Non-Steroidal Antiinflammatory |
| **HPV** | Human Papillomavirus |
| **STI** | Sexually Transmitted Infection |
| **HSV** | Herpes Simplex Virus |
| **PCR** | Polymerase Chain Reaction |
| **HIV** | Human Immunodeficiency Virus |
| **AIDS** | Acquired Immunodeficiency Syndrome |
| **HHV** | Human Herpesvirus |
| **CD4** | Cluster Of Differentiation 4 |
| **KOH** | Potassium Hydroxide |
| **URI** | Upper Respiratory Infection |
| **CN** | Cranial Nerve |
| **TM** | Tympanic Membrane |
| **HCTZ** | Hydrochlorothiazide |
| **Ig** | Immunoglobulin |
| **IM** | Intramuscular |
| **I&D** | Incision And Drainage |
| **PCN** | Penicillin |
| **HIDA** | Hepatic Iminodiacetic Acid |
| **AMS** | Altered Mental Status |
| **ASCA** | Antibodies Against The Yeast *Saccharomyces Cerevisiae* |

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# Abbreviations

| | |
|---|---|
| **ANCA** | Anti-Neutrophil Cytoplasmic Antibodies |
| **p-ANCA** | Perinuclear Anti-Neutrophil Cytoplasmic Antibodies |
| **GI** | Gastrointestinal |
| **IBS** | Irritable Bowel Syndrome |
| **A-Fib** | Atrial Fibrillation |
| **CAD** | Coronary Artery Disease |
| **FAP** | Familial Adenomatous Polyposis |
| **CMV** | Cytomegalovirus |
| **EGD** | Esophagogastroduodenoscopy |
| **GERD** | Gastroesophageal Reflux Disease |
| **LES** | Lower Esophageal Sphincter |
| **PPI** | Proton Pump Inhibitor |
| **HAV** | Hepatitis A Virus |
| **HBV** | Hepatitis B Virus |
| **IVDA** | Intravenous Drug Abuse |
| **HEV** | Hepatitis E Virus |
| **AST** | Aspartate Aminotransferase |
| **ALT** | Alanine Aminotransferase |
| **IEA** | Inferior Epigastric Artery |
| **HUS** | Hemolytic Uremic Syndrome |
| **IV** | Intravenous |
| **IVF** | Intravenous Fluid |
| **CA** | Carbohydrate Antigen |
| **NGT** | Nasogastric Tube |
| **VCUG** | Voiding Cystourethrogram |
| **UA** | Urinalysis |
| **TMP-SMX** | Trimethoprim-Sulfamethoxazole |
| **CVA** | Costovertebral Angle |
| **CVA** | Cerebrovascular Accident |
| **UVJ** | Ureterovesicular Junction |
| **LDH** | Lactate Dehydrogenase |
| **PT** | Prothrombin Time |
| **PTT** | Partial Thromboplastin Time |
| **DIC** | Disseminated Intravascular Coagulation |
| **TIBC** | Total Iron Binding Capacity |
| **EDTA** | Ethylenediaminetetraacetic Acid |
| **G6PD** | Glucose-6-Phophatase |
| **RBC** | Red Blood Cell |
| **Hgb** | Hemoglobin |
| **HbF** | Fetal Hemoglobin |
| **HSCT** | Hematopoietic Stem Cell Transplantation |
| **GBM** | Glomerular Basement Membrane |
| **WBC** | White Blood Cell |
| **ESRD** | End-Stage Renal Disease |
| **ABG** | Arterial Blood Gas |
| **ADH** | Antidiuretic Hormone |
| **DI** | Diabetes Insipidus |
| **CSF** | Cerebrospinal Fluid |
| **TB** | Tuberculosis |

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# Abbreviations

| | |
|---|---|
| **AFB** | Acid-Fast Bacilli |
| **PPD** | Purified Protein Derivative |
| **VDRL** | Venereal Disease Research Laboratory |
| **RPR** | Rapid Plasma Reagin |
| **DNA** | Deoxyribonucleic Acid |
| **PIP** | Proximal Interphalangeal Joints |
| **DIP** | Distal Interphalangeal Joints |
| **PMN** | Polumorphonuclear Neutrophils |
| **AP** | Anteroposterior |
| **MTP** | Metatarsophalangeal Joint |
| **RF** | Rheumatoid Factor |
| **CK** | Creatine Kinase |
| **EMG** | Electromyography |
| **MCP** | Metacarpophalangeal Joint |
| **CCP** | Cyclic Citrullinated Peptide |
| **DMARD** | Disease-Modifying Antirheumatic Drugs |
| **HLA** | Human Leukocyte Antigen |
| **ROM** | Range Of Motion |
| **SI** | Sacroiliac |
| **TCA** | Tricyclic Antidepressant |
| **ICP** | Intracranial Pressure |
| **NMDA** | N-Methyl-D-Aspartate |
| **EEG** | Electroencephalogram |
| **TLC** | Total Lung Capacity |
| **FEV** | Forced Expiratory Volume |
| **FVC** | Forced Vital Capacity |
| **LFT** | Liver Function Tests |
| **PA** | Pulmonary Arterial |
| **PID** | Pelvic Inflammatory Disease |
| **IUD** | Intrauterine Device |
| **IVF** | In Vitro Fertilization |
| **FHR** | Fetal Heart Rate |
| **TSH** | Thyroid Stimulating Hormone |
| **FSH** | Follicle Stimulating Hormone |
| **LH** | Luteinizing Hormone |
| **OCP** | Oral Contraceptive Pill |
| **USPSTF** | U.S. Preventive Services Task Force |
| **TAH** | Total Abdominal Hysterectomy |
| **BSO** | Bilateral Salpingo Oophorectomy |
| **LMP** | Last Menstrual Period |
| **PAPP-A** | Pregancy-Associated Plasma Protein A |
| **NST** | Nonstress Test |
| **SOB** | Shortness of Breath |
| **CBT** | Cognitive Behavioral Therapy |

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