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Yehuda Wolf, PA-C

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.

Emily Oslie, PA-C

Emily Oslie graduated with a B.S. in applied human biology from Seattle Pacific University where she played volleyball competitively. After graduation, she worked as a medical scribe in emergency departments and a primary care office in Seattle while applying to PA programs. Emily attended the Duke Physician Assistant Program where she served as class president and earned her PA certification and masters in health sciences. She is currently working at Duke Urgent Care in Durham, North Carolina.

Adam Rosh, MD

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Kristian Savic, Copy Editor

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.

Erica Parrish, Content Manager

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.
Yehuda Wolf, PA-C

I am still in shell shock. This project has been years in the making and the original reason why I ever reached out to Adam. I can’t believe we are finally done. First, I need to thank my most amazing wife and children for their unparalleled support and encouragement. Thank you for allowing me to do work after I finally got home from work. A special thank you to my mentors Dr. Hylton Lightman, MD and Dov Landa, PA-C whose mentorship and guidance continually make me a better provider to my patients. Thanks to my right-hand Emily and the rest of the team, Kristian and Erica. You are truly the ink to my pen, the paper to my pad and without you this project would be nothing but a sloppy mess in my mind. Thank you Adam for your constant support and encouragement. Always pushing us "one step further." Finally, I would like to thank the One Above for his Goodness and His Grace that He has shown me throughout my life. May this be the first of many more projects to come. In the words of the ancient physician Maimonides, "Never allow the thought to arise in me that I have attained to sufficient knowledge, but vouchsafe to me the strength, the leisure and the ambition ever to extend my knowledge. For art is great, but the mind of man is ever expanding."

Emily Osie, 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.
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, Aequanimitas

“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
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.

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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
Let’s get started...
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## I. Abdominal Pain

### A. Age Differences in Abdominal Pain

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<thead>
<tr>
<th></th>
<th>2-12 yrs old</th>
<th>13-49 yrs old</th>
<th>&gt; 50 yrs old</th>
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<tbody>
<tr>
<td>Nonspecific abdominal pain</td>
<td>++</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>Appendicitis</td>
<td>+</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>Cholecystitis</td>
<td>+</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Bowel obstruction</td>
<td>+</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Non-abdominal causes</td>
<td>+++</td>
<td>++</td>
<td>++</td>
</tr>
</tbody>
</table>

**Notes**
B. Rapid Onset of Abdominal Pain

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)
II. Acute/Chronic Cholecystitis

A. 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 Boa sign

Diagnostic Studies
- Initially: RUQ ultrasound
- Gold standard: HIDA scan

Management
- Cholecystectomy
**B. Acute Cholecystitis**

→ Caused by obstructing gallstone or bile stasis

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

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

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

**Management**
- Supportive care
- Cholecystectomy

**Murphy sign**
Palpation of gallbladder during inspiration
Patient stops breathing in and winces with a 'catch' in breath

---

**Notes**
C. Chronic Cholecystitis

• Porcelain gallbladder

**Porcelain (calcified) Gallbladder**

*Increased Risk for Gallbladder Carcinoma*

Notes
D. "Extended" Radical Cholecystectomy

The scope of wedge resection of the gallbladder fossa (about 2 cm in thickness or more)

↔ Lines of division of the extrahepatic bile duct

The extent of regional lymph node dissection, which achieves en bloc harvesting of both the first-echelon nodes (cystic duct and pericholedochal node groups) and the second-echelon nodes (posterosuperior pancreaticoduodenal, retroportal, right celiac, and hepatic artery node groups)

---

Notes
E. Cholecystostomy Tube

Complications
- Dislodged catheter
- Bleeding
- Recurrent cholecystitis

Acute cholecystitis

Yes

High-risk patient

No

Percutaneous cholecystostomy tube

Laparoscopic cholecystectomy

Laparoscopic cholecystostomy tube

No

Safe dissection

Yes

Proceed with laparoscopic cholecystectomy

Notes
III. Acute/Chronic 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 predicts the severity (Note: they are difficult to apply and have been found not to be reliable)

Comments
• Pancreatic pseudocyst is a complication in adults

Notes
### Acute Pancreatitis cont.

#### Ranson’s Criteria

<table>
<thead>
<tr>
<th>At Admission</th>
<th>Score</th>
<th>Associated Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 55 years</td>
<td>3-4</td>
<td>15%</td>
</tr>
<tr>
<td>WBCs &gt; 16,000/mm³</td>
<td>5-6</td>
<td>40%</td>
</tr>
<tr>
<td>Glucose &gt; 200 mg/dl</td>
<td>&gt; 7</td>
<td>100%</td>
</tr>
<tr>
<td>LDH &gt; 350 IU/L</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AST &gt; 250 SF units</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**48 hours After Admission**

- Hematocrit fall > 10%
- BUN rise > 5 mg/dl
- Calcium < 8 mg/dl
- PO₂ < 60 mm Hg
- Base deficit > 4 mEq/L
- Fluid sequestration > 6 L

#### Notes
B. Hemorrhagic Pancreatitis

Cullen Sign
ecchymosis around the Umbilicus

Grey-Turner Sign
echymosis of the Flank
C. Chronic Pancreatitis

Pathophysiology
- Most common cause is alcoholism

Presentation
- Abdominal pain radiating to back
- Pancreatic insufficiency: malabsorption, steatorrhea, glucose intolerance

Diagnostic Studies
- CT/AXR reveals calcifications

Progressive inflammatory changes that result in permanent structural damage

Impaired exocrine and endocrine function
IV. Anal Disease

FISSURES • ABSCESS • FISTULA

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

<table>
<thead>
<tr>
<th>Primary Anal Fissures</th>
<th>Secondary Anal Fissures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local trauma</td>
<td>Crohn disease</td>
</tr>
<tr>
<td>Constipation</td>
<td>Other granulomatous diseases</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>Malignancy</td>
</tr>
<tr>
<td>Vaginal delivery</td>
<td>Communicable disease</td>
</tr>
<tr>
<td>Anal sex</td>
<td></td>
</tr>
</tbody>
</table>

Management
- Topical nifedipine or nitroglycerin
- Topical analgesic
- Stool softener
- Sitz bath
- Fiber

Notes
B. 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)

Notes
C. Anorectal Fistula

Pathophysiology
• Communication between rectum and perianal skin

Presentation
• May produce anal discharge and pain if the tract becomes occluded

Anorectal Fistula Park Classification

- Levator ani muscle
- Puborectalis muscle
- Internal anal sphincter
- External anal sphincter

- Extrasphincteric fistula (Parks type 4)
- Superficial fistula (Parks type 2)
- Transsphincteric fistula (Parks type 2)
- Intersphincteric fistula (Parks type 1)
- Suprasphincteric fistula (Parks type 3)
V. 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

Notes
### B. Acute Appendicitis

- Noncompressible, dilated appendix (> 6 mm outer diameter)
- Aperistalsis
- Distinct appendiceal wall layers
- Target appearance (axial view)
- Appendicolith
- Periappendiceal fluid collection
- Echogenic (white) prominent pericecal fat

#### Notes
Acute Appendicitis cont.

**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

---

**McBurney Point**
- Umbilicus
- 2/3 of the way from the umbilicus to the ASIS

**Rovsing Sign**
- Palpate here (LLQ)
- Pain elicited in RLQ

**Psoas Sign**
- (Retrocecal appendicitis)
- Hip extension

**Obturator Sign**
- (Retrocecal or pelvic appendicitis)
- Flexion of knee
- Internal hip rotation

---

Notes
VI. Bariatric Surgery

A. Bariatric surgery

**Background**
- The only proven method to reduce and maintain weight loss
- **Last resort** due to associated risks
- **NIH guidelines** for bariatric surgery:
  - BMI > 40 (approx. 100 pounds above ideal body weight)
  - BMI > 35 with a medical problem related to morbid obesity
  - Individuals must have failed other non-surgical weight loss programs
  - They must be psychologically stable and able to follow post-op instructions
  - Obesity is not caused by a medical disease such as endocrine disorders

**Complications**
- **Early**
  - Anastomotic leak
  - DVT and PE
  - Splenic injury
- **Late**
  - Nutritional problems and malnutrition
  - Marginal ulcer and anastomotic strictures
  - Internal hernia
  - Cholelithiasis
  - Band slippage, band erosion
  - Esophageal dilatation
B. Types of Bariatric Procedures

**Restrictive Procedures**: less extensive but less weight loss and a decrease in morbidity

**Adjustable Gastric Banding (AGB)**
- Proximal gastric pouch is created using an inflatable band
- About **35-45%** of EBW is lost in the first few years.
- Less complications are associated but require frequent follow-up for band adjustment

**Sleeve Gastrectomy (SG)**
- 85% of the stomach is laparoscopically removed and the stomach takes the shape of a sleeve.

**Malabsorptive Procedures**: more weight loss but problems with malnutrition.

- Not very popular due to complications and malnutrition problems associated with it
- About **70-90%** of EBW is lost

**Biliopancreatic Diversion (BPD)**
- BPD is subtotal gastrectomy with a very distal Roux-en-Y reconstruction.

**Biliopancreatic Diversion with/without Duodenal Switch (BPD/DS)**
- BPD/DS is SG, duodenal transection with a very distal jejunileostomy.
Types of Bariatric Procedures cont.

Combination of Restrictive and Malabsorptive Procedures

**Roux-en-Y Gastric Bypass (RNYGB)**
- **Most commonly** used bariatric surgery for treatment of severe obesity in the US
- A proximal gastric pouch is created by transecting the stomach; a Roux-en-Y gastrojejunostomy is also created
- About 75-85% of excess body weight (EBW) is lost in the first 2 years
- Complications: **dumping syndrome** (rapid emptying of hyperosmolar chyme into the small bowel due to the destruction or bypass of the pyloric sphincter)

<table>
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<tr>
<th>Types of Bariatric Procedures</th>
<th>Restrictive</th>
<th>Malabsorptive</th>
<th>Combination of restrictive and malabsorptive</th>
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</thead>
<tbody>
<tr>
<td>Vertical banded gastroplasty</td>
<td>Laparoscopic adjustable gastric band</td>
<td>Jejunoleal bypass</td>
<td>Roux-en-Y gastric bypass</td>
</tr>
<tr>
<td>Sleeve gastrectomy</td>
<td>Biliopancreatic diversion</td>
<td>Biliopancreatic diversion with duodenal switch</td>
<td></td>
</tr>
</tbody>
</table>

Notes
### C. Post-Bariatric Surgery Micronutrient Supplementation

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<tr>
<th>Nutrient</th>
<th>Post-bariatric supplementation required to prevent deficiency (oral)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron</td>
<td>18 mg or 45 to 60 mg</td>
<td>• After all bariatric procedures&lt;br&gt;• Women of childbearing age and individuals with&lt;br&gt;history of iron deficiency: 45 to 60 mg</td>
</tr>
<tr>
<td>Vitamin B12</td>
<td>350 to 500 mcg</td>
<td>• After all bariatric procedures</td>
</tr>
<tr>
<td>Folate</td>
<td>400 to 1,000 mcg</td>
<td>• Women of childbearing age: 800 to 1000 mcg</td>
</tr>
<tr>
<td>Thiamin</td>
<td>12 to 100 mg</td>
<td>• After all bariatric procedures&lt;br&gt;• Recommend a B-complex vitamin of 50 mg or more in addition to a multivitamin</td>
</tr>
<tr>
<td>Calcium</td>
<td>1,200 to 2,400 mg (all sources)</td>
<td>• Supplement should include Vitamin D</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>3,000 IU (titrate to a serum 25(OH)D level of &gt; 30 ng/mL)</td>
<td>• Continue 3000 IU per day total, from all sources (eg, multivitamin, calcium supplement)</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>5,000 to 10,000 IU</td>
<td>• 5000 to 10,000 IU after gastric bypass or sleeve gastrectomy&lt;br&gt;• 10,000 IU after biliopancreatic diversion/ duodenal switch</td>
</tr>
<tr>
<td>Vitamins E/K</td>
<td>15mg/90 to 300 mcg</td>
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<tr>
<td>Zinc/copper</td>
<td>8 to 22 mg/1 to 2 mg</td>
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<tr>
<td>Selenium</td>
<td>Quantity contained in a “high potency” multivitamin</td>
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<tr>
<td>Magnesium</td>
<td>Quantity contained in a multivitamin that “contains magnesium”</td>
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</tr>
<tr>
<td>Additional B vitamins</td>
<td>100 to 200% of daily value (DV)</td>
<td></td>
</tr>
<tr>
<td>Trace minerals</td>
<td>Quantity contained in a multivitamin &quot;complete in minerals&quot;</td>
<td>• Molybdenum, manganese, chromium, etc</td>
</tr>
</tbody>
</table>

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**Notes**
**VII. Bowel Obstruction**

**A. 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

**Common radiologic findings**
- Dilated loops of bowel: above the point of obstruction
- Air-fluid levels - on upright or decubitus film
- “String of pearls” - small amount of gas caught between the valvulae conniventes in a fluid-filled mechanical SBO

**Mechanical Causes**
- Extramural
- Adhesion
- Hernia
- Neoplasm
- Abscess/phlegmon
- Mural
- Volvulus
- Neoplasm
- Crohn disease
- Radiation enteritis

**Notes**
B. 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

Rectum 9%
Cecum 6%
Ascending colon 5%
Hepatic flexure 3%
Transverse colon 9%
Splenic flexure 14%
Descending colon 16%
Sigmoid colon 38%
C. Hastra: Large Bowel

→ Small pouches caused by sacculation

Do not traverse circumference of intestine

Teniae coli
Run the length of large intestine

Hastra

Notes
D. Valvulae conniventes: Small Bowel

Valvulae conniventes (Plicae circulares)
- Mucosal folds of small intestines
- Begin in 2nd part of duodenum
- Disappears in distal ileum

Traverses circumference of intestine

Notes
**E. Sigmoid Volvulus**

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

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

**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)

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

Primary Biliary Cholangitis (formerly cirrhosis)

→ Slow progressive destruction of the small bile ducts

Notes

Gamma-glutamyltransferase (GGT)
Alkaline phosphatase
Alanine aminotransferase (ALT)
Aspartate aminotransferase (AST)
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
- Asymptomatic (50%)
- Pruritus
- Fatigue

Complications
- Cholestasis
- Liver failure

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

Notes
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

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
IX. Cholelithiasis/Choledocholithiasis

A. Cholelithiasis

Pathophysiology
- Gallstones most commonly comprised of cholesterol

Patient
- Four “Fs”: Female, Forty, Fat, Fertile

Presentation
- Slowly resolving RUQ pain, onset suddenly after eating fatty foods

Diagnostic Studies
- Ultrasound is diagnostic

Management
- Observation and supportive care
- Cholecystectomy if symptoms are persistent or if cholecystitis is present

Comments
- Choledocholithiasis occurs when a gallstone is lodged in the common bile duct, causing an outflow obstruction with resultant jaundice and liver damage

Risk Factors
- Increased age (40s)
- Female sex
- Multiparity
- Pregnancy
- Obesity
- Hypertriglyceridemia
- Profound weight loss
- Chronic intravascular hemolysis
B. Choledocholithiasis

→ Gallstones in the common bile duct

Cholelithiasis
Gallstones in gallbladder

Gallbladder
Cystic duct
Greater duodenal papilla
Pancreatic duct
Hepatic duct
Small bile duct
Gallstones in the Common bile duct
X. Colorectal Carcinoma

A. Polyps

Pathophysiology
- Common
- Main concern is malignant transformation, which occurs at different rates depending on the size and type of polyp
  - Distal colon: commonly benign
  - Proximal colon: more likely to be cancerous
- The larger the colonic polyp, the greater the risk of malignant transformation
- Villous adenomas have a 30–70% risk of malignant transformation
- The greater the number of concomitant colonic polyps, the greater the risk of malignant transformation

Comments
- Most common cause of painless rectal bleeding in the pediatric population
- Once identified, follow-up colonoscopy in three to five years

Notes
X. Colorectal Carcinoma

B. Familial Adenomatous Polyposis (FAP)

Pathophysiology
- Characterized by the development of hundreds to thousands of colonic adenomatous polyps.

Patient
- Mean age of polyp development is 15 years and cancer at 40 years.

Comments
- First-degree relatives of patients with FAP should undergo genetic screening after age 10 years of age.
- Family should undergo yearly sigmoidoscopy beginning at 12 years of age.

Colorectal Cancers

Sporadic cases

Familial Risk

Lynch syndrome (Hereditary Nonpolyposis Colorectal Cancer (HNPCC))

Familial Adenomatous Polyposis (FAP)

Hamartomatous Polyposis Syndrome (HPS)
### Coloectal Cancer Screening with Increased Risk Due to Family History

| Genetic syndromes (Familial adenomatous polyposis, Hereditary nonpolyposis colorectal cancer) | Early, intense screening, genetic counseling, genetic testing |
| Colorectal cancer or adenoma in one first degree relative or Colorectal cancer or adenomas in two or more first degree relatives | Colonoscopy beginning age 40 or 10 years earlier than age of youngest at diagnosis, whichever comes first |
| Colorectal cancer or adenoma in one first degree relative ≥ 60 years or Colorectal cancer in two or more second degree relatives | Average risk screening starting at age 40 |

---

**Notes**
Colorectal Carcinoma: Screening cont.

<table>
<thead>
<tr>
<th>Characteristics of Colorectal Cancer Screening Strategies</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Screening Method</strong></td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td><strong>Stool-Based Tests</strong></td>
</tr>
<tr>
<td>gFOBT</td>
</tr>
<tr>
<td>FIT</td>
</tr>
<tr>
<td>FIT-DNA</td>
</tr>
<tr>
<td><strong>Direct Visualization Tests</strong></td>
</tr>
<tr>
<td>Colonoscopy</td>
</tr>
<tr>
<td>CT colonography</td>
</tr>
<tr>
<td>Flexible sigmoidoscopy</td>
</tr>
<tr>
<td>Flexible sigmoidoscopy with FIT</td>
</tr>
</tbody>
</table>

**FIT**: Fecal immunochemical test  
**FIT-DNA**: Multitargeted stool DNA test  
**gFOBT**: Guaiac-based fecal occult blood test

Notes
## D. Colorectal Carcinoma: Treatment Options

<table>
<thead>
<tr>
<th>Stage</th>
<th>Surgery</th>
<th>Chemotherapy/Biologics</th>
<th>Radiation</th>
<th>Interventional Radiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Stage I</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Stage II</td>
<td>Yes</td>
<td>Yes, for rectal and high risk colon cancers. FOLFOX (5-FU/Leucovorin/Oxaliplatin) or CapeOx (Capecitabine/Oxaliplatin)</td>
<td>Yes, for rectal cancer. Given in tandem with 5-FU or Xeloda</td>
<td>No</td>
</tr>
<tr>
<td>Stage III</td>
<td>Yes</td>
<td>FOLFOX or CapeOx</td>
<td>Yes, for rectal cancer. Given in tandem with 5-FU or Xeloda</td>
<td>No</td>
</tr>
<tr>
<td>Stage IV</td>
<td>Yes, if the tumor is obstructive or blocking the bowel. No, if the tumor is not blocking the bowel</td>
<td>FOLFOX, FOLFIRI, FOLFIRINOX, irinotecan, Avastin, Erbitux, Vectibix, Zaltrap, Stivarga, Lonsurf, Cyramza</td>
<td>Yes, for rectal cancer and in certain other cases</td>
<td>Possibly. Options could be Radio Frequency Ablation (RFA), Stereotactic Body Radiation Therapy (SBRT), or chemoembolization</td>
</tr>
</tbody>
</table>

**Notes**
XI. Diarrhea/Constipation/Obstipation/Change in Bowel Habits

A. Food Poisoning

- *Bacillus cereus*: eating reheated rice; diarrhea
- *C. botulinum*: canned food, diarrhea
- *Clostridium perfringens*: reheated meat or canned foods within 24 hours; watery diarrhea and epigastric pain
- Ciguatera: sea bass, grouper, red snapper; diarrhea
- *E. coli O157:H7*: undercooked meat; diarrhea, HUS
- *Salmonella*: poultry, meat, eggs
- Scombroid: peppery tasting fish; diarrhea
- *Staphylococcus aureus*: eating meats, mayonnaise, custard 1–6 hours ago; nausea, vomiting, abdominal pain, and diarrhea
- *V. parahaemolyticus, V. vulnificus*: shellfish and seafood; diarrhea
- *Yersinia*: undercooked pork
### B. Invasive Gastroenteritis

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

**Notes**
### C. Non-Invasive Gastroenteritis

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

---

**Notes**
D. Traveler’s Diarrhea

Pathophysiology
• Most commonly caused by enterotoxigenic Escherichia coli (ETEC)

Patient
• History of recent travel

Presentation
• Abrupt onset of watery diarrhea, nausea, abdominal cramping

Management
• Rehydration and ciprofloxacin (azithromycin in pregnant women and children)

Traveler’s Diarrhea

→ Enterotoxigenic Escherichia coli (ETEC)
(Most common cause)

• Diarrhea
• Abdominal cramps
• Nausea
• Bloating
E. *Clostridioides Difficile* Colitis
(Pseudomembranous Colitis)

**Patient**
- History of recent antibiotic use, *clindamycin* is most common

**Presentation**
- Frequent watery stools, abdominal pain

**Diagnostic Studies**
- Nucleic acid amplification test (NAAT)

**Management**
- **Adults**
  - Nonsevere or severe: *oral vancomycin* or oral fidaxomicin
  - Fulminant: oral vancomycin with parenteral metronidazole
- **Children**
  - Nonsevere: oral vancomycin or oral metronidazole
  - Severe or fulminant: oral vancomycin

**Comments**
- In patients with at least two *Clostridioides* infection recurrences treated with appropriate antibiotic therapy, the guidelines recommend use of fecal microbiota transplantation
**Clostridioides Difficile Colitis cont.**

- Spore-forming
- Toxin-producing
- Gram-positive
- Anaerobic

**Clinical**

**Nonsevere disease** (WBV < 15,000 cells/mL & creatinine < 1.5 mg/dL)
- Watery diarrhea
- Abdominal pain and cramping
- Fever
- Nausea, anorexia
- Pseudomembranous colitis

**Severe and fulminant colitis** (WBV > 15,000 cells/mL or creatinine > 1.5 mg/dL)
- Hypotension, shock, ileus, or megacolon

**Diagnosis**

Laboratory stool test for C. difficile toxin or C. difficile toxin B gene
- Enzyme immunoassay (EIA)
- Glutamate dehydrogenase (GDH) antigen
- Toxins A and B

**Antimicrobial Agents that may Induce Clostridioides difficile Diarrhea and Colitis**

<table>
<thead>
<tr>
<th>Frequently associated</th>
<th>Occasionally associated</th>
<th>Rarely associated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluoroquinolones</td>
<td>Macrolides</td>
<td>Aminoglycosides</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>Trimethoprim-sulfamethoxazole</td>
<td>Tetracyclines</td>
</tr>
<tr>
<td>Cephalosporins (broad spectrum)</td>
<td></td>
<td>Metronidazole</td>
</tr>
<tr>
<td>Penicillins (broad spectrum)</td>
<td></td>
<td>Vancomycin</td>
</tr>
</tbody>
</table>

**Notes**
F. Treatment of Nonsevere *Clostridioides* Colitis – Associated Diarrhea in Adults

| Initial episode | Preferred: Vancomycin 125 mg PO QID for 10 days  
Alternative: Fidaxomicin 200 mg PO BID for 10 days  
Alternative: Metronidazole 500 mg PO TID for 10 days |
|-----------------|--------------------------------------------------|
| First recurrence| Preferred: Vancomycin 125 mg PO QID for 10 days  
Alternative: Fidaxomicin 200 mg PO BID for 10 days |
| Second recurrence| Preferred: Tapering and pulsed oral vancomycin with or without probiotics  
125 mg PO QID for 7 to 14 days  
125 mg PO BID for 7 days  
125 mg PO QD for 7 days  
125 mg PO QoD for 7 days  
125 mg PO every 3 days for 14 days  
Alternative: Fidaxomicin 200 mg PO BID for 10 days |
| Subsequent recurrence| Preferred: Fecal microbiota transplant  
Alternative: Vancomycin (tapering and pulsed) with probiotics  
Alternative: Fidaxomicin 200 mg PO BID for 10 days |

Notes
G. Treatment of *Clostridioides* Infection in Children

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Suggested treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First episode</strong></td>
<td></td>
</tr>
<tr>
<td>Mild or moderate</td>
<td>• Vancomycin 40 mg/kg per day orally divided in four doses for 10 days, <strong>or</strong></td>
</tr>
<tr>
<td></td>
<td>• Metronidazole 30 mg/kg per day orally divided in four doses for 10 days</td>
</tr>
<tr>
<td>Severe</td>
<td>• Vancomycin 40 mg/kg per day orally divided in four doses for 10 days</td>
</tr>
<tr>
<td>Fulminant</td>
<td>• Vancomycin 40 mg/kg per day orally divided in four doses until clinical improvement and then (if applicable) decrease the maximum dose to 125 mg to complete 10 days</td>
</tr>
<tr>
<td>Fulminant disease and ileus</td>
<td>• Metronidazole 30 mg/kg per day IV divided in four doses, <strong>plus</strong></td>
</tr>
<tr>
<td></td>
<td>• Vancomycin 10 mg/kg per dose in normal saline administered by retention enema four times per day; the volume of solution varies with age:</td>
</tr>
<tr>
<td></td>
<td>1 through 4 years: 50 mL</td>
</tr>
<tr>
<td></td>
<td>5 through 11 years: 75 mL</td>
</tr>
<tr>
<td></td>
<td>≥ 12 years: 100 mL</td>
</tr>
<tr>
<td><strong>Recurrent episodes</strong></td>
<td></td>
</tr>
<tr>
<td>First recurrent, mild or moderate</td>
<td>• Repeat regimen used for first episode</td>
</tr>
<tr>
<td>Subsequent recurrent, mild or moderate</td>
<td>• Pulsed-tapered vancomycin (maximum dose: 125 mg):</td>
</tr>
<tr>
<td></td>
<td>10 mg/kg orally four times daily for 10 to 14 days, followed by</td>
</tr>
<tr>
<td></td>
<td>10 mg/kg orally twice daily for 7 days, followed by</td>
</tr>
<tr>
<td></td>
<td>10 mg/kg orally once daily for 7 days, followed by</td>
</tr>
<tr>
<td></td>
<td>10 mg/kg orally every other day for 7 days, followed by</td>
</tr>
<tr>
<td></td>
<td>10 mg/kg orally every 3 days for 2 to 8 weeks</td>
</tr>
</tbody>
</table>

**Notes**
H. 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

**Clinical**
- Abdominal distension and pain
- Chronic constipation
- Fecal incontinence/overflow diarrhea

**Complications**
- Rectal necrosis and ulcers

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

**Fecal Impaction**
→ A solid, immobile bulk of feces in the colon as a result of chronic constipation
I. Common Laxatives

<table>
<thead>
<tr>
<th>Category</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bulk formers</td>
<td>Psyllium, methylcellulose</td>
</tr>
<tr>
<td>Softeners</td>
<td>Docusate</td>
</tr>
<tr>
<td>Stimulants</td>
<td>Senna, bisacodyl, castor oil</td>
</tr>
<tr>
<td>Saline preps</td>
<td>Magnesium hydroxide, citrate</td>
</tr>
<tr>
<td></td>
<td>Magnesium citrate, sulfate</td>
</tr>
<tr>
<td>Lubricants</td>
<td>Mineral oil</td>
</tr>
<tr>
<td>Osmotics</td>
<td>Lactulose, sorbitol, glycol</td>
</tr>
<tr>
<td>Prokinetics</td>
<td>Tegaserod, metoclopramide</td>
</tr>
</tbody>
</table>

Notes
XII. Diverticular Disease

A. Diverticulosis

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

**Presentation**
- **Painless hematochezia**

**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**

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

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

---

**Notes**

**Diverticulosis**

*Most common cause of significant lower GI bleeding*
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)
XIII. Esophageal Cancer

A. Esophageal Cancer

Pathophysiology
- **Squamous cell carcinoma** most commonly caused by smoking and alcohol
- **Adenocarcinoma** most commonly caused by Barrett esophagus

Patient
- Older man

Presentation
- Weight loss and dysphagia to solid foods

Diagnostic Studies
- Endoscopy with biopsy

Comments
- Most common location for cancer is **distal third**
- Most common 50-70 years of age
- Male > Female
- Solid food dysphagia
- Weight loss
- Dx: Endoscopy with biopsy
- Staging: CT chest and liver

Notes
A. Esophageal Strictures

Pathophysiology
- **Esophageal web**: thin membranes in the mid-upper esophagus
- **Plummer-Vinson**: esophageal webs + dysphagia + iron deficiency anemia
- **Schatzki ring** is a **diaphragm-like** mucosal ring that forms at the esophagogastric junction (the B ring); if the lumen of this ring becomes too small, symptoms occur

Presentation
- **Solid food dysphagia** in a patient with a history of GERD

Diagnostic Studies
- Diagnosed with **barium swallow**

Management
- Endoscopic dilation

Diagnosis
- Barium esophagram
- Endoscopy
- Esophageal manometry
- 24 hour pH monitoring

Management
- Stricture dilation
- Proton pump inhibitors
XV. Gastric Cancer

A. Gastric Carcinoma

Pathophysiology
- Adenocarcinoma is most common

Patient
- Man with a history of *H. pylori* infection

Presentation
- Loss of appetite, unintentional weight loss

Physical Exam
- Left supraclavicular node (Virchow node), left axillary node (Irish node), periumbilical node (Sister Mary Joseph node)

Clinical
- Weight loss and persistent abdominal pain (most common)
- Nausea, dysphagia
- Melena, early satiety
- Ulcer-type pain

Paraneoplastic syndromes
- Seborrheic keratoses (sign of Leser-Trélat)
- Acanthosis nigricans
- Microangiopathic hemolytic anemia
- Membranous nephropathy
- Hypercoagulable states (Trousseau syndrome)
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
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

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

*Risk varies depending on source

Notes
XVII. Hematemesis

Hematemesis:
Commonly caused by acute upper GI bleed and patients present with vomiting of blood or coffee-ground-like material.
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

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

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

Management
- Supportive
- Endoscopic therapy (active bleeding)
- Acid suppression (no active bleeding)
B. Esophageal Varices

Pathophysiology
- Most commonly caused by portal hypertension

Patient
- History of chronic liver disease, alcoholism

Diagnostic Studies
- Upper endoscopy will reveal dilated submucosal gastric veins

Management
- Acute: hemodynamic support, octreotide, vasopressin, Sengstaken-Blakemore tube
- Chronic: BBs, variceal ligation

Comments
- Associated with massive upper GI bleed

Acute Management of Bleeding
- Hemodynamic resuscitation
- Octreotide
- Banding, sclerotherapy
- Prophylactic antibiotics (e.g., ceftriaxone)

Chronic Management
- Beta-blockers
- Endoscopic variceal ligation

Cirrhosis → Portal hypertension → Varices

Notes
XVIII. Hemorrhoids

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

Notes
### B. Classification of Internal Hemorrhoids

<table>
<thead>
<tr>
<th>Degree of prolapse</th>
<th>Clinical description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>Hemorrhoids do not protrude through the anus</td>
</tr>
<tr>
<td>2nd</td>
<td>Hemorrhoids prolapse but reduce spontaneously</td>
</tr>
<tr>
<td>3rd</td>
<td>Hemorrhoids prolapse and require manual reduction</td>
</tr>
<tr>
<td>4th</td>
<td>Hemorrhoids cannot be reduced and may strangulate</td>
</tr>
</tbody>
</table>
C. Excision of Thrombosed External Hemorrhoid

- Anesthetize
- Make an elliptical incision of skin overlying clot
- Remove clot

Notes
XIX. Hepatic Carcinoma

A. Hepatocellular Carcinoma

Pathophysiology
- Most common primary liver cancer
- Most common cause is chronic HBV/HCV cirrhosis

Presentation
- Rapidly growing ascites

Diagnostic Studies
- Bloody ascitic fluid
- Elevated alpha-Fetoprotein (AFP)
XX. Hernias

INGUINAL • FEMORAL • INCISIONAL • HIATAL

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 (< one year old, > 40 years old)

Management

- Nonreducible: **emergent** surgical consultation

Comments

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

<table>
<thead>
<tr>
<th>Reducible</th>
<th>Soft, easy to place back into defect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incarcerated</td>
<td>Firm and painful, non-reducible by direct manual pressure</td>
</tr>
<tr>
<td>Strangulated</td>
<td>Painful due to impaired blood flow, ischemia, and necrosis, overlying skin changes</td>
</tr>
</tbody>
</table>

Notes
B. Inguinal Triangle

→ Hesselbach Triangle

Notes
## Hernias

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

### Notes
C. Hiatal Hernia

Pathophysiology
• Type I: sliding
• Type II-IV: paraesophageal

Presentation
• May have reflux symptoms

Diagnostic Studies
• Endoscopy or barium swallow

Management
• Manage reflux, consider surgery if symptoms are not well controlled
D. Paraesophageal Hernia

- True hernia
- Hernia sac
- Upward dislocation of gastric fundus through defect in phrenoesophageal membrane

Clinical
- Asymptomatic or vague, intermittent symptoms
- Epigastric or substernal pain or postprandial fullness, nausea, retching
- GERD symptoms are less prevalent as compared to sliding (type 1) hernia

Diagnosis
- Upper endoscopy
- Barium swallow (most sensitive)

Management
- Asymptomatic (conservative vs prophylactic surgery)
- Symptomatic (surgical management)
### XXI. Inflammatory Bowel Disease

#### 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

**Rectum always involved** (but not ankylosing spondylitis)

**Colectomy eliminates cancer risk**

**Extends proximally in continuous fashion**

**Notes**
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
C. Crohn Disease vs. Ulcerative Colitis

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

Notes
XXII. Jaundice

A. Classification of Jaundice According to Mechanism

<table>
<thead>
<tr>
<th>Unconjugated hyperbilirubinemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased bilirubin production*</td>
</tr>
<tr>
<td>• Extravascular hemolysis</td>
</tr>
<tr>
<td>• Extravasation of blood into tissues</td>
</tr>
<tr>
<td>• Intravascular hemolysis</td>
</tr>
<tr>
<td>• Dyserythropoiesis</td>
</tr>
<tr>
<td>• Wilson disease</td>
</tr>
<tr>
<td>Impaired hepatic bilirubin uptake</td>
</tr>
<tr>
<td>• Heart failure</td>
</tr>
<tr>
<td>• Portosystemic shunts</td>
</tr>
<tr>
<td>• Some patients with Gilbert syndrome</td>
</tr>
<tr>
<td>• Drugs (e.g. rifampin, probenecid)</td>
</tr>
<tr>
<td>Impaired bilirubin conjugation</td>
</tr>
<tr>
<td>• Crigler-Najjar syndrome types I and II</td>
</tr>
<tr>
<td>• Gilbert syndrome</td>
</tr>
<tr>
<td>• Neonates</td>
</tr>
<tr>
<td>• Hyperthyroidism</td>
</tr>
<tr>
<td>• Ethinyl estradiol</td>
</tr>
<tr>
<td>• Liver disease (chronic hepatitis, advanced cirrhosis)</td>
</tr>
</tbody>
</table>

Notes
Classification of Jaundice According to Mechanism cont.

### Conjugated hyperbilirubinemia

**Defect of canalicular anion transport**
- Dubin-Johnson syndrome

**Defect of sinusoidal reuptake of conjugated bilirubin**
- Rotor syndrome

**Extrahepatic cholestasis (biliary obstruction)**
- Choledochocholithiasis
- Intrinsic and extrinsic tumors (e.g. cholangiocarcinoma)
- Primary sclerosing cholangitis
- AIDS cholangiopathy
- Acute and chronic pancreatitis
- Strictures after invasive procedures
- Parasitic infections (e.g. *Ascaris lumbricoides*)

**Intrahepatic cholestasis**
- Viral hepatitis
- Alcoholic hepatitis
- Nonalcoholic steatohepatitis
- Chronic hepatitis
- Primary biliary cholangitis
- Drugs (e.g. alkylated steroids, chlorpromazine)
- Sepsis and hypoperfusion states
- Infiltrative diseases (e.g. amyloidosis, lymphoma, sarcoidosis, TB)
- Total parenteral nutrition
- Pregnancy
- End-stage liver disease

*Serum bilirubin < 4 mg/dL in absence of underlying liver disease

---

Notes
XXIII. Melena/Hematochezia

A. Melena

- Black tarry stool
- Most common cause is upper GI bleed

B. Hematochezia

- Bright red blood per rectum (BRBPR)
C. Lower GI Bleeding

**Upper GI Bleeding**

- Angiodysplasia
- Hemorrhoids

**Lower GI Bleeding**

- Diverticula
- Ischemic colitis
- Ligament of Treitz
- Colitis (Noninfectious (UC, Crohn), Infectious)
- Polyps
- Carcinoma
- Hemorrhoids

Notes
XXIV. Pancreatic Carcinoma

A. Pancreatic Cancer

Pathophysiology
- Most common type is adenocarcinoma

Patient
- History of smoking

Presentation
- Painless jaundice and weight loss

Physical Exam
- Palpable, nontender gallbladder (Courvoisier sign), migratory thrombophlebitis (Trousseau syndrome)

Diagnostic Studies
- CA 19-9 serum marker

Clinical Presentation
- Vague, diffuse pain in the epigastrium and LUQ (if tail is involved)
- Diarrhea and weight loss
- Depression
- Painless jaundice
- Palpable gallbladder (Courvoisier sign)

Notes
XXV. Pancreatic Pseudocyst

A. Pancreatic Pseudocyst

Collection of fluid is rich in pancreatic enzymes, blood, and necrotic tissue

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

<table>
<thead>
<tr>
<th>Early (&lt; 4 weeks)</th>
<th>Late (&gt; 4 weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection is rare</td>
<td>Drainage is reasonable if symptomatic</td>
</tr>
<tr>
<td>Drainage not recommended</td>
<td>• Acute peripancreatic collection</td>
</tr>
<tr>
<td></td>
<td>• Acute necrotic collection</td>
</tr>
<tr>
<td></td>
<td>• Pseudocyst</td>
</tr>
<tr>
<td></td>
<td>• Walled-off necrosis</td>
</tr>
</tbody>
</table>

Notes
XXVI. Peptic Ulcer Disease

A. Peptic Ulcer Disease

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

Presentation
• Gnaing epigastric pain
  • Duodenal ulcer: pain is alleviated by ingesting food ("DUDe 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
• Awakens patient at night
• Pain immediately after meals

Notes
B. Perforated Peptic Ulcer

- Abdominal pain that radiates to back
- Referred pain to shoulder
- Pneumoperitoneum
- Surgical consultation
- Exploratory laparotomy

Notes
XXVII. Pyloric Stenosis

A. Pyloric Stenosis

Patient
- Infant 2–6 weeks old

Presentation
- Non-bilious projectile vomiting after feeding and early satiety

Physical Exam
- RUQ olive-like mass (hypertrophied pylorus)

Diagnostic Studies
- Hypochloremic hypokalemic metabolic alkalosis
- Ultrasound (target sign) or upper GI series (string sign)

Management
- Surgical intervention

Clinical
- Projectile, non-bilious vomiting
- Immediate postprandial vomiting
- Infant is hungry between feedings (hungry vomiter)

Management
- Supportive, treat electrolyte imbalances
- Pyloromyotomy

Progressive non-bilious vomiting
- More common 2 weeks - 2 months of life

Hypertrophied pylorus
- Olive-shaped mass in RUQ

Hypochloremic hypokalemic metabolic alkalosis

Diagnosis
- Ultrasound

Notes
XXVIII. Small Bowel Carcinoma

A. Small Bowel Carcinoma

Pathophysiology
• Rare, delay in diagnosis is common
• Adenocarcinomas represent 25–40 percent of small bowel cancers
• Most common location is the duodenum

Presentation
• Most common presenting symptom is abdominal pain that is typically intermittent and crampy in nature

<table>
<thead>
<tr>
<th>Risk Factors for Small Bowel Cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hereditary cancer syndromes and adenocarcinoma</td>
</tr>
<tr>
<td>Chronic inflammation (Crohn’s disease)</td>
</tr>
<tr>
<td>Dietary factors (alcohol, refined sugar, red meat, salt-cured and smoked foods)</td>
</tr>
<tr>
<td>Neuroendocrine tumors</td>
</tr>
</tbody>
</table>
XXIX. Toxic Megacolon

A. Toxic Megacolon

Pathophysiology

• Most commonly caused by inflammatory bowel disease

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, emergent surgical consultation

Clinical Diagnosis

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

Inflammatory bowel disease or infectious colitis characterized by total or segmental nonobstructive colonic dilatation + Systemic toxicity

Notes
## ACID/BASE DISORDERS
*See Chapter Urology/Renal*

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<td><strong>CARDIAC DISEASE HISTORY</strong></td>
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<td></td>
<td>Myocardial Infarction</td>
</tr>
<tr>
<td></td>
<td>Unstable Angina</td>
</tr>
<tr>
<td></td>
<td>Valvular Disease</td>
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<tr>
<td></td>
<td>Hypertension</td>
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<td></td>
<td>Arrhythmias</td>
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<td></td>
<td>Heart Failure</td>
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</table>

## FLUID/VOLUME DISORDERS
Volume Overload/Depletion

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<td>113</td>
<td><strong>METABOLIC DISEASE HISTORY</strong></td>
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<td></td>
<td>Diabetes</td>
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<td></td>
<td>Adrenal Insufficiency</td>
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</table>

## DEEP VENOUS THROMBOSIS

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<td><strong>ELECTROLYTE DISORDERS</strong></td>
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<td></td>
<td><em>See Chapter Urology/Renal</em></td>
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</tbody>
</table>

## METABOLIC DISEASE HISTORY
Diabetes
Adrenal Insufficiency

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<td>114</td>
<td><strong>PULMONARY DISEASE HISTORY</strong></td>
</tr>
<tr>
<td></td>
<td>Asthma</td>
</tr>
<tr>
<td></td>
<td>Chronic Obstructive Pulmonary Disease</td>
</tr>
</tbody>
</table>

## HEMATOLOGIC DISEASE HISTORY
Clotting Disorders
*See Chapter Hematology*
Anticoagulant Use

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<td><strong>RISK ASSESSMENT</strong></td>
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## SUBSTANCE USE DISORDER

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<th>Topic</th>
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<tr>
<td>131</td>
<td><strong>TOBACCO USE/DEPENDENCE</strong></td>
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## POSTOPERATIVE FEVER

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<td><strong>POSTOPERATIVE FEVER</strong></td>
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</table>

## WOUNDS/INFECTIONS

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<th>Page</th>
<th>Topic</th>
</tr>
</thead>
<tbody>
<tr>
<td>139</td>
<td><strong>WOUNDS/INFECTIONS</strong></td>
</tr>
</tbody>
</table>
I. Cardiac Disease History

MYOCARDIAL INFARCTION • UNSTABLE ANGINA • VALVULAR DISEASE
HYPERTENSION • ARRHYTHMIAS • HEART FAILURE

1. MI, UNSTABLE 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

Notes
### B. 12-Lead ECG in Perioperative Evaluation

| Preoperative resting 12-lead electrocardiogram is reasonable for patients with: | Class IIa  
Level of evidence: B |
|---|---|
| - Known coronary heart disease  
- Significant arrhythmia  
- Peripheral arterial disease  
- Cerebrovascular disease  
- Other significant structural heart disease, except for those undergoing low-risk surgery | |

| Preoperative resting 12-lead ECG may be considered for: | Class IIb  
Level of evidence: B |
|---|---|
| - Asymptomatic patients without known coronary heart disease  
- Except for those undergoing low-risk surgery | |

---

**Notes**
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

<table>
<thead>
<tr>
<th>Location</th>
<th>Leads</th>
<th>Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior and Septal</td>
<td>V1–V4</td>
<td>Left anterior descending (LAD)</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, aVF</td>
<td>Right coronary artery (RCA) (70%+) or Left circumflex (LCx)</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL, V5–V6</td>
<td>Left circumflex (LCx), Diagonal of Left anterior descending</td>
</tr>
</tbody>
</table>

**Notes**
D. 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)

### Notes

<table>
<thead>
<tr>
<th>Test</th>
<th>Time Detectable from Onset of Ischemia</th>
<th>Peak</th>
<th>Return to Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin T &amp; I</td>
<td>3-12 hrs</td>
<td>24-48 hrs</td>
<td>5-14 days</td>
</tr>
<tr>
<td>Creatine kinase (CK)</td>
<td>3-12 hrs</td>
<td>24 hrs</td>
<td>48-72 hrs</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>1-2 hrs</td>
<td>8-10 hrs</td>
<td>1-2 days</td>
</tr>
</tbody>
</table>
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)

Notes
2. VALVULAR DISEASE

A. Cardiac Murmurs

<table>
<thead>
<tr>
<th>Valve</th>
<th>Side Best Auscultated</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic valve</td>
<td>Right</td>
<td>2nd intercostal space, just lateral to the sternum</td>
</tr>
<tr>
<td>Pulmonic valve</td>
<td>Left</td>
<td>2nd intercostal space, just lateral to the sternum</td>
</tr>
<tr>
<td>Tricuspid valve</td>
<td>Left</td>
<td>4th-5th intercostal space, over left sternal border</td>
</tr>
<tr>
<td>Mitral valve</td>
<td>Left</td>
<td>5th intercostal space, midclavicular line (Apex)</td>
</tr>
</tbody>
</table>

Notes
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 **radiating to the carotids** which **decreases** with Valsalva
- Paradoxically **split S2**
- **S4 gallop**

Management
- Aortic valve replacement

---

Chest Pain

Dyspnea Syncope

- Crescendo-decrescendo
- Systolic ejection murmur
- Delayed/diminished carotid pulses
- Paradoxically split S2
- Narrow pulse pressure

---

Notes
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
Aortic Insufficiency (Aortic Regurgitation) cont.

### Aortic Regurgitation

<table>
<thead>
<tr>
<th></th>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causes</strong></td>
<td>Endocarditis</td>
<td>Rheumatic heart disease</td>
</tr>
<tr>
<td></td>
<td>Aortic dissection</td>
<td>Congenital (bicuspid) valve</td>
</tr>
<tr>
<td></td>
<td>Trauma</td>
<td>Prior endocarditis</td>
</tr>
<tr>
<td><strong>Clinical</strong></td>
<td>Severe dyspnea</td>
<td>Exertional dyspnea</td>
</tr>
<tr>
<td></td>
<td>Pulmonary edema</td>
<td>Fatigue</td>
</tr>
<tr>
<td></td>
<td>Acute heart failure</td>
<td>Heart failure</td>
</tr>
<tr>
<td><strong>Pulse pressure</strong></td>
<td>Normal</td>
<td>Wide</td>
</tr>
<tr>
<td><strong>Murmur/Heart sound</strong></td>
<td>Short, diastolic murmur, S3</td>
<td>High-pitched decrescendo diastolic blowing murmur best heard along left sternal border</td>
</tr>
</tbody>
</table>

**Notes**
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
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
## 3. HYPERTENSION

### A. JNC 8 Treatment for HTN

- **BP goals:**
  - Age < 60 or diabetic: **140/90** mm Hg
  - Age > 60: **150/90** mm Hg

- **Treatment:**
  - First line is always lifestyle modifications
  - Nonblack: Thiazide, ACE inhibitor, or ARB or CCB alone or in combination
  - Black: **Thiazide** or **CCB** alone or in combination
  - CKD (with or without diabetes): all races **ACE inhibitor** or **ARB** alone or in combination with other drug classes

### JNC 8 Recommendations

<table>
<thead>
<tr>
<th>Patient subgroup</th>
<th>Target SBP (mm Hg)</th>
<th>Target DBP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 60 years</td>
<td>&lt; 150</td>
<td>&lt; 90</td>
</tr>
<tr>
<td>&lt; 60 years</td>
<td>&lt; 140</td>
<td>&lt; 90</td>
</tr>
<tr>
<td>&gt; 18 years w/chronic kidney disease</td>
<td>&lt; 140</td>
<td>&lt; 90</td>
</tr>
<tr>
<td>&gt; 18 years w/diabetes</td>
<td>&lt; 140</td>
<td>&lt; 90</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Population</th>
<th>Initial therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Non-African-American</td>
<td>- Thiazides</td>
</tr>
<tr>
<td></td>
<td>- Calcium channel blockers</td>
</tr>
<tr>
<td></td>
<td>- ACE inhibitors</td>
</tr>
<tr>
<td></td>
<td>- ARBs</td>
</tr>
<tr>
<td>General African-American</td>
<td>- Thiazides</td>
</tr>
<tr>
<td></td>
<td>- Calcium channel blockers</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>- Treatment should include ACE inhibitor or ARB</td>
</tr>
</tbody>
</table>

- **Up-litrate** or add therapy after 1 month if BP goal not achieved
- **Do NOT** use ACE inhibitors and ARBs together
- **If > 3 drugs needed,** refer to hypertension specialist

---

**Notes**
### B. Treatment of Hypertensive Emergencies

<table>
<thead>
<tr>
<th>Drug</th>
<th>Onset of action</th>
<th>Adverse effects</th>
<th>Special indications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vasodilators</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Sodium nitroprusside  | Immediate       | Nausea, vomiting, muscle twitching, thiocyanate and cyanide poisoning            | • May cause reflex tachycardia  
• Combine with beta-blocker  
• Caution with azotemia  
• 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  
• 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**
### C. Types of Hypertension

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Primary (Essential)</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common</td>
<td>More common</td>
<td>Less common</td>
</tr>
<tr>
<td>Onset</td>
<td>Gradual</td>
<td>Acute</td>
</tr>
<tr>
<td>Age</td>
<td>≥ 40 years old</td>
<td>1st–2nd decade &amp; 5th–6th decade</td>
</tr>
<tr>
<td>Clinical</td>
<td>Sxs occur years after onset</td>
<td>Symptoms occur at the start of hypertension</td>
</tr>
<tr>
<td>Family History</td>
<td>Strong</td>
<td>May or may not be present</td>
</tr>
</tbody>
</table>

**Etiology**
- Idiopathic
- Chronic kidney disease
- Medications
- Renovascular disease (renal artery stenosis)
- Drugs (EtOH, cocaine)
- Hyperaldosteronism
- Polycythemia vera
- Hyper/hypothyroidism
- Coarctation of the aorta
- Cushing disease
- Sleep apnea
- Pheochromocytoma
- Hyperparathyroidism

**Prognosis**
- Lifelong
- May or may not resolve

---

**Notes**
4. 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
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)
  - PR 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

---

1. Progressive prolongation of PR interval
2. Dropped QRS complex

---

**Notes**
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

**Mobitz II**

- **Fixed PR interval**

  ![Image of ECG showing fixed PR interval and dropped QRS complexes](image)

  - 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**
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
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

Notes

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

A. Acute Decompensated Heart Failure

Presentation
- Exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea

Physical Exam
- Pitting edema, S₃ 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)
### Acute Decompensated Heart Failure cont.

**Decreased Hypoperfusion**
- Cold sweaty extremities
- Oliguria
- Mental confusion
- Dizziness
- Diminished pulse pressure

**Increased Congestion**
- Pulmonary edema
- Peripheral edema
- JVD
- Hepatomegaly
- Acites

<table>
<thead>
<tr>
<th>No hypoperfusion</th>
<th>Warm - Dry</th>
<th>Warm - Wet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoperfusion</td>
<td>Cold - Dry</td>
<td>Cold - Wet</td>
</tr>
</tbody>
</table>


### 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

<table>
<thead>
<tr>
<th>Class</th>
<th>Functional state</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No limitation</td>
<td>Asymptomatic during usual daily activities</td>
</tr>
<tr>
<td>II</td>
<td>Slight limitation</td>
<td>Mild symptoms (dyspnea, fatigue, or chest pain) with ordinary activities</td>
</tr>
<tr>
<td>III</td>
<td>Moderate limitation</td>
<td>Symptoms noted with minimal activity</td>
</tr>
<tr>
<td>IV</td>
<td>Severe limitation</td>
<td>Symptoms at rest. Any activity results in limitations.</td>
</tr>
</tbody>
</table>
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
II. Deep Venous Thrombosis

A. Deep Vein Thrombosis

Pathophysiology
- Most commonly caused by stasis, hypercoagulable state, trauma (Virchow triad)

Patient
- History of smoking, long-distance travel, surgery, oral contraceptive use

Presentation
- Unilateral leg edema, leg pain, tenderness, and warmth

Physical Exam
- Asymmetrical swelling
- Positive Homan sign

Diagnostic Studies
- Initially: duplex ultrasound
- Gold standard: venography

Management
- IV heparin, then switch to warfarin
- Low molecular weight heparin
- Direct oral anticoagulants

Comments
- Wells Criteria used for risk stratification

Notes
## B. Risk Factors for Deep Vein Thrombosis in Ambulatory Patients

<table>
<thead>
<tr>
<th>Category</th>
<th>Risk Factors</th>
</tr>
</thead>
</table>
| **Acquired (persistent)** | Advanced age  
                          | Malignancy  
                          | Antiphospholipid antibodies  
                          | Prior history of DVT/PE |
| **Acquired (transient)** | Recent surgery or major trauma  
                          | Pregnancy  
                          | Oral contraceptives/hormone replacement therapy  
                          | Prolonged immobilization |
| **Inherited**    | Antithrombin III deficiency  
                          | Proteins C and S  
                          | Factor V Leiden  
                          | Prothrombin gene mutation |
C. Virchow Triad

- Circulatory stasis
- Thombosis
- Endothelial injury
- Hypercoagulable state

Notes
D. Pretest Probability of DVT (Modified Wells score)

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active cancer (treatment ongoing or within the previous 6 months or palliative)</td>
<td>1</td>
</tr>
<tr>
<td>Paralysis, paresis, or recent plaster immobilization of the lower extremities</td>
<td>1</td>
</tr>
<tr>
<td>Recently bedridden for more than 3 days or major surgery, within 4 weeks</td>
<td>1</td>
</tr>
<tr>
<td>Localized tenderness along the distribution of the deep venous system</td>
<td>1</td>
</tr>
<tr>
<td>Entire leg swollen</td>
<td>1</td>
</tr>
<tr>
<td>Calf swelling by more than 3 cm when compared to the asymptomatic leg (measured below tibial tuberosity)</td>
<td>1</td>
</tr>
<tr>
<td>Pitting edema (greater in symptomatic leg)</td>
<td>1</td>
</tr>
<tr>
<td>Collateral superficial veins (nonvaricose)</td>
<td>1</td>
</tr>
<tr>
<td>Alternative diagnosis as likely or more likely than that of DVT</td>
<td>-2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Score</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>High probability</td>
<td>50 to 75%</td>
</tr>
<tr>
<td>Moderate probability</td>
<td>17%</td>
</tr>
<tr>
<td>Low probability</td>
<td>3%</td>
</tr>
</tbody>
</table>

**Modified Wells Criteria**

<table>
<thead>
<tr>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previously documented DVT (added for modified score)</td>
</tr>
</tbody>
</table>

**Modified Wells Score**

<table>
<thead>
<tr>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>DVT likely</td>
</tr>
<tr>
<td>DVT unlikely</td>
</tr>
</tbody>
</table>


- Low Pretest Probability
  - D-dimer
    - Positive → Ultrasound
    - Normal → No further testing

- Mod Pretest Probability
  - D-dimer
    - Positive → Ultrasound
    - Normal → No further testing

- High Pretest Probability
  - Ultrasound

High sensitivity D-dimer Whole leg ultrasound
## III. Hematologic Disease History

### ANTICOAGULANT USE

#### A. Anticoagulation Management

<table>
<thead>
<tr>
<th>Bridging protocol for warfarin</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>5 days before surgery</strong></td>
<td>• Stop warfarin; start LMWH or UFH</td>
</tr>
</tbody>
</table>
| **1 day before surgery**       | • Check INR. If INR > 1.5, give 1-2 mg of oral vitamin K  
|                               | • Recheck INR |
| **Surgery day**                | • Start LMWH or UFH 12-24 hrs after surgery, if bleeding risk is low |
| **1 day after surgery**        | • Start warfarin |
| **5 days after surgery**       | • Stop LMWH or UFH once INR is > 2  
|                               | • Continue warfarin |

<table>
<thead>
<tr>
<th>Protocol for direct oral anticoagulant</th>
<th></th>
</tr>
</thead>
</table>
| **1-2 days before surgery**           | • Stop agent  
|                                       | • Dabigatran 2 days prior  
|                                       | • Apixaban and rivaroxaban 1 day prior |
| **1 day after surgery**               | • Start agent |

### Protocol for antiplatelet agents

| **7 days before surgery**            | • Stop aspirin or clopidogrel |
| **1 day after surgery**              | • Start agent 12-24 hours after surgery |

LMWH: Low molecular weight heparin  
UFH: Unfractionated heparin

---

Notes
IV. Fluid/Volume Disorders

VOLUME OVERLOAD/DEPLETION

A. Volume Overload/Depletion

Calculation of Maintenance Fluids

- Daily maintenance for sensible and insensible loss in adult = 1,500 to 2,500 mL depending on age, gender, weight, BSA
- Multiply patient weight (kg) × 30 = fluid over 24 hours
- Increased requirements for fever, hyperventilation, and increased catabolism IV fluid replacement for short period
- Indications for urinary catheter placement:
  - Anticipating long procedure
  - Performing urologic or low pelvic surgery
  - Need to monitor fluid balance

<table>
<thead>
<tr>
<th>Holliday-Segar 4-2-1 Maintenance Fluid Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>First 10 kg</td>
</tr>
<tr>
<td>Second 10 kg</td>
</tr>
<tr>
<td>Additional kgs</td>
</tr>
</tbody>
</table>
V. Metabolic Disease History

DIABETES • ADRENAL INSUFFICIENCY

1. DIABETES

A. Diabetes Insipidus (DI)

Presentation
- Polyuria, polydipsia

Diagnostic Studies
- Increase in plasma osmolality and decrease in urine osmolality

Central DI

Pathophysiology
- Most commonly caused by decreased ADH production

Diagnostic Studies
- Vasopressin challenge test: > 50% increase in urine osmolality and decrease urine volume

Management
- Intranasal DDAVP

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
Diabetes Insipidus (DI) cont.

**Central**
- Decreased secretion of ADH
- Pituitary gland
- Antidiuretic Hormone (ADH)

**Sodium**
- Urine: Dilute
- Serum: Concentrated

**Causes**
- Idiopathic
- Head trauma
- Pituitary tumor
- Neurosurgery

**Nephrogenic**
- Kidney Resistant to ADH
- Antidiuretic Hormone (ADH)

**Causes**
- Lithium toxicity
- Renal Disease
- Hypokalemia
- Pregnancy
- Medications

---

Notes
B. 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)

Notes
Autoimmune destruction of beta cells

↓ insulin = ↑ glucose

Notes
C. 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 $\geq 200 \text{ mg/dL}$
    • Fasting plasma glucose of $\geq 126 \text{ mg/dL}$ on **two** separate occasions
    • Glycated hemoglobin (A1C) of $\geq 6.5%$
    • Plasma glucose of $> 200 \text{ mg/dL}$ **two hours** after a **75 g glucose load** during an oral glucose tolerance test

Management
• Lifestyle modifications, then medication
  (metformin is first line)
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)
E. General Goals of Perioperative Diabetes Management

<table>
<thead>
<tr>
<th>Avoidance of hypoglycemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevention of ketoacidosis and hyperosmolar states</td>
</tr>
<tr>
<td>Maintenance of fluid and electrolyte balance</td>
</tr>
<tr>
<td>Avoidance of marked hyperglycemia</td>
</tr>
</tbody>
</table>

Notes
### F. Perioperative Characteristics of Oral Hypoglycemics

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sulfonylureas</strong></td>
<td>Increase the risk of hypoglycemia</td>
</tr>
<tr>
<td><strong>Metformin</strong></td>
<td>Contraindicated in conditions that increase the risk of renal hypoperfusion, lactate accumulation, and tissue hypoxia</td>
</tr>
<tr>
<td><strong>Thiazolidinediones</strong></td>
<td>May worsen fluid retention and peripheral edema and could precipitate congestive heart failure</td>
</tr>
<tr>
<td><strong>Sodium-glucose cotransporter 2 (SGLT2)</strong> inhibitors</td>
<td>Increase the risk of hypovolemia. There have also been reports of acute kidney injury and euglycemic diabetic ketoacidosis in patients with type 2 diabetes taking SGLT2 inhibitors</td>
</tr>
<tr>
<td><strong>Dipeptidyl peptidase-4 (DPP-4) inhibitors and GLP-1 analogs</strong></td>
<td>Could alter gastrointestinal motility and worsen the postoperative state</td>
</tr>
</tbody>
</table>

---

**Notes**
G. Diabetic Ketoacidosis

Patient
• Diabetic with recent Infection, Ischemia (cardiac, mesenteric), Infarction, Ignorance (poor control), Intoxication (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
Diabetic Ketoacidosis cont.

**DKA Diagnosis**
- Blood glucose > 250 mg/dL
- Moderate ketonuria or ketonemia
- Arterial pH < 7.3
- Serum bicarb < 15 mEq/L

**HHS Diagnosis**
- Blood glucose > 600 mg/dL
- Min/No ketonuria or ketonemia
- Arterial pH > 7.3
- Serum bicarb > 20 mEq/L
- Altered level of consciousness
- Serum osmolality > 320 mOsm/kg

**DKA**
- Glucose > 250 mg/dL
  - Assess K+
  - Infuse 0.14 u/kg/hr IV insulin
  - Glucose fails by ≥ 10% in 1st hour
  - Check glucose hourly
  - DKA

**HHS**
- Glucose > 600 mg/dL
  - Glucose fails by < 10% in 1st hour
  - Bolus 0.14 u/kg body weight/hr insulin and continue insulin infusion
  - DKA

- Lower insulin infusion to 0.02 - 0.05 u/kg/hr
- Change IV fluid to 5% dextrose with 0.45% NaCl at 150-250 mL/hr
- Keep glucose between 150-250 mg/dL until DKA resolved

- Lower insulin infusion to 0.02 - 0.05 u/kg/hr
- Change IV fluid to 5% dextrose with 0.45% NaCl at 150-250 mL/hr
- Keep glucose between 200-300 mg/dL until HHS resolved

**DKA Resolution**
- Blood glucose < 200 mg/dL PLUS any two:
  - Arterial pH > 7.3
  - Serum bicarb > 15 mEq/L
  - Anion gap < 12 mEq/L

**HHS Resolution**
- Normal serum osmolality
- Normal vital signs
- Baseline mental status

---

2. 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

Adrenal Crisis

Adrenal Gland

\[
\text{Cortisol} \downarrow \text{Aldosterone} = \downarrow \text{Sodium}\ 
\uparrow \text{Glucose} \uparrow \text{Potassium}
\]
VI. Pulmonary Disease History

ASTHMA • CHRONIC OBSTRUCTIVE PULMONARY DISEASE

1. 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
- O2: 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$_2$O
  - Permissive hypercapnia to avoid breath stacking

Notes
## B. Pharmacologic Management of Acute Asthma Exacerbations

<table>
<thead>
<tr>
<th><strong>Beta-2 agonist bronchodilator</strong> (nebulized or metered-dose inhaler)</th>
<th><strong>Systemic glucocorticoids</strong> (for those with a poor response to treatment after one hour or with initial therapy for patients on chronic oral glucocorticoids)</th>
</tr>
</thead>
</table>
| • Albuterol by MDI 4 to 8 puffs every 20 minutes up to 1 hour, then every 1 to 4 hours, prn  
• Albuterol by nebulizer 0.063 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  
• Albuterol by continuous nebulization, administering 10 to 15 mg per hr | • For patients who can be managed at home: prednisone 40 to 60 mg per day in a single or divided dose  
• 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 |
| **Ipratropium** |  |
| • By nebulizer, 500 mcg every 20 minutes for 3 doses, then prn. Can be given simultaneously with beta-2 agonist.  
• By MDI, 4 to 8 inhalations every 20 minutes for 3 doses, then prn |  |

**For patients not responding to above therapies, consider adjunct therapies**  
• Intravenous magnesium sulfate 2 g infused over 20 minutes, in absence of renal insufficiency  
• Subcutaneous terbutaline 0.25 mg every 20 minutes for up to 3 doses
2. CHRONIC OBSTRUCTIVE PULMONARY DISEASE

A. Emphysema

Pathophysiology
- Caused by abnormal and permanent enlargement of airspaces via **airspace wall destruction**

Patient
- Older with a history of smoking

Presentation
- Dyspnea

Physical Exam
- **Pink skin, pursed-lip breathing, barrel chest, decreased breath sounds, and hyperresonance to percussion** ("pink puffer")

Diagnostic Studies
- **Decreased FEV₁** and **increased TLC**

Comments
- **Emphysema in a young nonsmoker**: think **alpha-1 antitrypsin deficiency**
B. Chronic Bronchitis

Presentation
• Chronic productive cough for at least three months in at least two successive years
• Overweight and cyanotic (“blue bloater”)

Physical Exam
• Decreased breath sounds, increased resonance on percussion of the lung fields, and use of accessory muscles to breathe

Diagnostic Studies
• FEV₁/FVC ratio is less than 0.7 and FEV₁ is less than 80% of predicted

Management
• Smoking cessation, lifestyle changes with pulmonary rehabilitation, inhaled bronchodilators, and inhaled steroids
Notes
C. Chronic Bronchitis vs. Emphysema

Blue Bloater  
Chronic Bronchitis

Symptoms
- Chronic, productive cough
- Purulent sputum
- Hemoptysis
- Mild dyspnea initially
- Cyanosis (due to hypoxemia)
- Peripheral edema (due to cor pulmonale)
- Crackles, wheezes
- Prolonged expiration
- Obese

Complications
- Secondary polycythemia vera due to hypoxemia
- Pulmonary hypertension due to reactive vasoconstriction from hypoxemia
- Cor pulmonale from chronic pulmonary hypertension

Pink Puffer  
Emphysema

Symptoms
- Dyspnea
- Minimal cough
- Increased minute ventilation
- Pink skin, pursed-lip breathing
- Accessory muscle use
- Cachexia
- Hyperinflation, barrel chest
- Decreased breath sounds
- Tachypnea

Complications
- Pneumothorax due to bullae
- Weight loss due to work of breathing
VII. Risk Assessment

A. Assessing the Risk of Postoperative Pulmonary Complications

- **History and physical exam seeking known risk factors for pulmonary complications**

  - **Resective lung surgery**
  - **Specific periop eval**

  - **Pos**
    - COPD
    - Unexplained dyspnea or exercise intolerance
    - Smoking history within past 2 months
    - Poor general health status (ASA class > 2)
    - Heart failure
    - Obstructive sleep apnea
    - Pulmonary hypertension
    - Abnormal chest exam
    - Upper abdominal surgery
    - Abdominal aortic aneurysm, or thoracic surgery
    - Surgery expected to last > 3 hours
    - Emergency Surgery

    - **Chest X-ray**
    - **PFTs** if uncharacterized dyspnea or if uncertain of airflow obstruction in patient with COPD or asthma

  - **Neg**
    - Low risk
    - Proceed to surgery without further evaluation

- **Abnormal test or multiple risk factors**
  - **High risk**
    - Reconsider indications for surgery
    - Perioperative treatment to reduce risk
    - Consider shorter procedure
    - Consider epidural or spinal anesthesia or regional block

- **Normal test**
  - **Moderate risk**
    - Perioperative treatment to reduce risk
### B. Preoperative Frailty Assessment

<table>
<thead>
<tr>
<th>Frailty criteria</th>
<th>Assessment</th>
<th>Score</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Shrinkage</strong></td>
<td>Ask the patient: Have you unintentionally lost ≥ 10 lbs in the past year? <strong>Yes / No</strong></td>
<td>If <strong>Yes</strong>, add 1 point</td>
<td></td>
</tr>
</tbody>
</table>
| **Weakness (grip strength)** | 1. Ask the patient to hold dynamometer in dominant hand with arms parallel to their body without squeezing arms against their body.  
2. Adjust the handle to ensure that the middle phalanx rests on the inner handle.  
3. Ask the patient to squeeze the handle and record.  
4. Perform three trials, and obtain the average value. Record results below:  
   - Trial 1: _______ kg force  
   - Trial 2: _______ kg force  
   - Trial 3: _______ kg force  
   - **Average:** _______ kg force | Compare patient’s average with the lowest 20th percentile by gender and BMI shown below:  
|             | **Men** | **Women** | **Men** | **Women** |        |        |
| BMI        | Kg force | BMI        | Kg force | **value** |        |        |
| ≤ 24       | ≤ 20     | ≤ 23       | ≤ 17     |            |        |        |
| 24.1–26    | ≤ 30     | 23.1–26    | ≤ 17.3   |            |        |        |
| 26.1–28    | ≤ 31     | 26.1–29    | ≤ 18     |            |        |        |
| > 28       | ≤ 32     | > 29       | ≤ 21     |            |        |        |
| **Exhaustion**       | Ask the patient the following two questions:  
1. How often in the last week did you feel that everything you did was an effort?  
2. How often in the last week did you feel that you could not get going? **_______** | **Add 1 point if the average** falls within or below the above values |        |
| **Low physical activity** | Ask the patient the following four questions:  
1. Can you get out of bed or chair yourself? **Yes / No**  
2. Can you dress and bathe yourself? **Yes / No**  
3. Can you make your own meals? **Yes / No**  
4. Can you do your own shopping? **Yes / No** | **Add 1 point for a score of 2 or 3 for EITHER question** |        |
| **Slowness**         | 1. Ask the patient to stand up and walk toward the tape on the ground.  
2. Using a stopwatch, record the time it takes for the patient to walk 15 feet. Record results below:  
   - Trial: _______ seconds | **Add 1 point if the trial time falls higher than the above values** |        |

#### Frailty Score

Total the number of points for each criterion (the total should be 0-5) to determine the frailty score.

- **0-1:** Not frail
- **2-3:** Intermediate (pre-frail)
- **4-5:** Frail

**If the patient is in the intermediate frail or frail categories, please notify the surgeon.**
### C. Low, Intermediate, and High Intrinsic Cardiac Risk Operations

<table>
<thead>
<tr>
<th>Description</th>
<th>Odds ratio (95% CI)</th>
<th>Estimated cardiac risk of hypothetical patient (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Low intrinsic cardiac risk</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partial mastectomy (lumpectomy)</td>
<td>0.22 (0.15–0.31)</td>
<td>0.05</td>
</tr>
<tr>
<td>Arthroscopic rotator cuff repair</td>
<td>0.32 (0.19–0.54)</td>
<td>0.07</td>
</tr>
<tr>
<td>Simple mastectomy (complete breast)</td>
<td>0.37 (0.26–0.50)</td>
<td>0.08</td>
</tr>
<tr>
<td>Laparoscopic appendectomy</td>
<td>0.45 (0.33–0.62)</td>
<td>0.10</td>
</tr>
<tr>
<td>Laparoscopic cholecystectomy</td>
<td>0.62 (0.53–0.72)</td>
<td>0.14</td>
</tr>
<tr>
<td><strong>Intermediate intrinsic cardiac risk</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transurethral resection of bladder tumor, large</td>
<td>0.85 (0.61–1.20)</td>
<td>0.19</td>
</tr>
<tr>
<td>Laparoscopic prostatectomy</td>
<td>0.88 (0.69–1.12)</td>
<td>0.19</td>
</tr>
<tr>
<td>Open appendectomy</td>
<td>0.95 (0.51–1.75)</td>
<td>0.21</td>
</tr>
<tr>
<td>Total hip arthroplasty</td>
<td>0.95 (0.83–1.08)</td>
<td>0.21</td>
</tr>
<tr>
<td>Laparoscopic radical hysterectomy with bilateral salpingo-ovohorectomy</td>
<td>1.05 (0.57–1.94)</td>
<td>0.23</td>
</tr>
<tr>
<td><strong>High intrinsic cardiac risk</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laparoscopic total abdominal colectomy with ileostomy</td>
<td>1.50 (0.92–2.44)</td>
<td>0.33</td>
</tr>
<tr>
<td>Breast reconstruction with free flap</td>
<td>1.52 (0.81–2.86)</td>
<td>0.33</td>
</tr>
<tr>
<td>Open cholecystectomy</td>
<td>1.55 (1.25–1.92)</td>
<td>0.34</td>
</tr>
<tr>
<td>Open ventral hernia repair, incarcerated or strangulated, recurrent</td>
<td>1.78 (1.29–2.44)</td>
<td>0.39</td>
</tr>
<tr>
<td>Whipple procedure, pylorus-sparing</td>
<td>4.70 (4.00–5.53)</td>
<td>1.02</td>
</tr>
</tbody>
</table>

### Notes
VIII. Substance Use Disorder

A. Substance Abuse

Surgical Challenges
- Venous access, arterial injury, deep venous thrombosis (DVT), abscess formation and gas gangrene, tissue compression, crush injury and ischemia

Diagnostic Studies
- Obtain serum or urine tests

Comments
- Postoperatively be wary of the potential for hemodynamic compromise, poor wound healing, altered consciousness and difficulty with pain management
- Information regarding a patient’s drug habit may be very important for anesthesia in planning their anaesthetic care

Notes
IX. Tobacco Use/Dependence

A. Tobacco Use/Dependence

- Smoking within one year of surgery has been associated with increased postoperative complications including deleterious effects on wound healing which are thought to be related to the nicotine content of conventional tobacco
### B. Helping Patients Quit:

<table>
<thead>
<tr>
<th>Medications Used for Smoking Cessation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
</tr>
<tr>
<td>------</td>
</tr>
<tr>
<td><strong>First line</strong></td>
</tr>
<tr>
<td>Nicotine replacement therapy (NRT)</td>
</tr>
<tr>
<td>[Gum and patches]</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Bupropion (sustained release)</td>
</tr>
<tr>
<td>Varenicline</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Second line</strong></td>
</tr>
<tr>
<td>Nortriptyline</td>
</tr>
<tr>
<td>Combination therapies (Bupropion &amp; NRT)</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

**Notes**
X. Postoperative Fever

A. Postoperative Fever

• **Post-Operative Day (POD) 1-2: Wind**
  • Lungs, i.e. pneumonia, aspiration, and pulmonary embolism; **atelectasis** has been commonly cited as a cause of post-operative fever

• **POD 3-5: Water**
  • Urinary tract infection, possibly catheter-associated

• **POD 5-7: Wound**
  • Infection of the surgical incision(s), either superficial or deep

• **POD 5-7: (W)abscess**
  • Infection of an organ or space

• **POD ≥6: Walking**
  • Deep vein thrombosis or pulmonary embolism

• **POD Anytime: Wonder drugs** or “What did we do?”
  • Drug fever or reaction to blood products

• **POD Anytime: Wing/Waterway**
  • Bloodstream infection, phlebitis, or cellulitis related to intravenous lines, either central or peripheral
B. Causes of Postoperative Fever

**Infectious**
- Surgical site infection
- Pneumonia (ventilator-associated, aspiration)
- UTI (indwelling catheter)
- Intravascular catheter-associated infection
- Antibiotic-associated diarrhea
- Sinusitis
- Otitis media
- Parotitis
- Intra-abdominal abscess
- Meningitis
- Acute pyelonephritis
- Endocarditis
- Foreign body infection
- Osteomyelitis

**Noninfectious**

**Surgical site inflammation without infection**
- Hematoma/seroma
- Suture reaction

**Thrombosis**
- Deep vein thrombosis
- Pulmonary embolism

**Inflammatory**
- Gout/pseudogout
- Pancreatitis

**Vascular**
- Cerebral infarction/hemorrhage
- Subarachnoid hemorrhage
- Myocardial infarction
- Bowel ischemia/infarction

**Other**
- Medications
- Drug/alcohol withdrawal
- Transfusion reactions
- Transplant rejection
- Hyperthyroidism
- Hypoadrenalism
XI. Wounds/Infections

A. Wound Healing

- Major event: Clot formation, Hemostasis
- Cellular response: Lymphocytes, Macrophages, Neutrophils
- Vascular response: Vasoconstriction, Vasodilation
- Phase: Inflammatory, Proliferation, Remodeling

Timeline:
- Injury
- 3 days
- 7 days
- 3 weeks
- 1 year

Images showing stages of wound healing:
- Bleeding
- Inflammatory
- Proliferative
- Remodeling

Notes
## B. Classification of Surgical Wounds

<table>
<thead>
<tr>
<th>Type of wound</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class I</strong> Clean</td>
<td>- No hollow viscus entered</td>
</tr>
<tr>
<td></td>
<td>- Primary wound closure</td>
</tr>
<tr>
<td></td>
<td>- No inflammation</td>
</tr>
<tr>
<td></td>
<td>- No brakes in septic technique</td>
</tr>
<tr>
<td></td>
<td>- Elective procedure</td>
</tr>
<tr>
<td><strong>Class II</strong> Clean contaminated</td>
<td>- Hollow viscus entered but controlled</td>
</tr>
<tr>
<td></td>
<td>- No inflammation</td>
</tr>
<tr>
<td></td>
<td>- Primary wound closure</td>
</tr>
<tr>
<td></td>
<td>- Minor break in aseptic technique</td>
</tr>
<tr>
<td></td>
<td>- Mechanical drain used</td>
</tr>
<tr>
<td></td>
<td>- Bowel preparation preop</td>
</tr>
<tr>
<td><strong>Class III</strong> Contaminated</td>
<td>- Uncontrolled spillage from viscus</td>
</tr>
<tr>
<td></td>
<td>- Inflammation apparent</td>
</tr>
<tr>
<td></td>
<td>- Major break in aseptic technique</td>
</tr>
<tr>
<td><strong>Class IV</strong> Dirty</td>
<td>- Untreated, uncontrolled spillage from viscus</td>
</tr>
<tr>
<td></td>
<td>- Pus in operative wound</td>
</tr>
<tr>
<td></td>
<td>- Open suppurative wound, severe inflammation</td>
</tr>
</tbody>
</table>

Notes
### C. Recommended Antibiotic Prophylactic Regimens by Procedure

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Antibiotic</th>
<th>Dose (single dose within 1 hour before procedure)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hysterecomy (including supracervical) Vaginal</td>
<td>Cefazolin</td>
<td>2–3 g IV for patients weighing &gt; 120 kg</td>
</tr>
<tr>
<td>Abdominal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Laparoscopic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Robotic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uterine evacuation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suction D&amp;C</td>
<td>Doxycycline</td>
<td>200 mg</td>
</tr>
<tr>
<td>D&amp;E</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colporrhaphy</td>
<td>Cefazolin</td>
<td>2–3 g IV for patients weighing &gt; 120 kg</td>
</tr>
<tr>
<td>Vaginal sling placement</td>
<td>Cefazolin</td>
<td>2–3 g IV for patients weighing &gt; 120 kg</td>
</tr>
<tr>
<td>Laparotomy without entry into bowel or vagina</td>
<td>Consider cefazolin</td>
<td>2–3 g IV for patients weighing &gt; 120 kg</td>
</tr>
<tr>
<td>Cervical tissue excision procedures (LEEP, biopsy, endocervical curettage)</td>
<td>Not recommended</td>
<td></td>
</tr>
<tr>
<td>Cystoscopy</td>
<td>Not recommended</td>
<td></td>
</tr>
<tr>
<td>Endometrial biopsy</td>
<td>Not recommended</td>
<td></td>
</tr>
<tr>
<td>Laparoscopic procedures without entry into bowel or vagina</td>
<td>Not recommended</td>
<td></td>
</tr>
<tr>
<td>Hysterosalphingogram</td>
<td>Not recommended</td>
<td></td>
</tr>
<tr>
<td>Chromotubation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saline infusion sonography</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hysteroscopy</td>
<td>Not recommended</td>
<td></td>
</tr>
<tr>
<td>Operative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnostic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intrauterine device insertion</td>
<td>Not recommended</td>
<td></td>
</tr>
<tr>
<td>Oocyte retrieval</td>
<td>Not recommended</td>
<td></td>
</tr>
<tr>
<td>D&amp;C for nonpregnancy indication</td>
<td>Not recommended</td>
<td></td>
</tr>
<tr>
<td>Urodynamics</td>
<td>Not recommended</td>
<td></td>
</tr>
</tbody>
</table>
CHAPTER 3
Cardiovascular

144 AORTIC ANEURYSM/DISSECTION
149 ARTERIAL EMBOLISM/THROMBOSIS
152 ARTERIAL/VENOUS ULCER DISEASE
155 CHEST PAIN
History of Angina
159 CLAUDICATION

160 PERIPHERAL ARTERIAL DISEASE
162 DYSPNEA ON EXERTION
164 SYNCOPE
165 VARICOSE VEINS
I. Aortic Aneurysm/Dissection

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
- Angiography is the **gold standard**
- Aortic diameter > 3.0 cm is considered aneurysmal

**Management**
- If rupture is suspected - directly to OR
- < 4 cm: screen every 12 months
- > 4 cm: screen every six months
  - Rapidly expanding: > 0.5 cm in six months
  - or >1 cm per year: surgical repair
- > 5.5 cm: surgical intervention

**Comments**
- The USPSTF recommends one-time screening via ultrasonography in men aged 65–75 who have ever smoked
Abdominal Aortic Aneurysm (AAA) cont.

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

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

Notes
B. USPSTF Abdominal Aortic Aneurysm Screening

<table>
<thead>
<tr>
<th>Population</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men ages 65 to 75 with a history of smoking</td>
<td>One-time screening by ultrasound</td>
</tr>
<tr>
<td>Men ages 65 to 75 who have never smoked</td>
<td>Selective screening based on risk factors, patient and family history, and patient preference</td>
</tr>
<tr>
<td>Women ages 65 to 75 with a history of smoking</td>
<td>No recommendation for or against screening</td>
</tr>
<tr>
<td>Women ages 65 to 75 who have never smoked</td>
<td>Recommends against screening</td>
</tr>
</tbody>
</table>

Notes
C. Aortic Dissection

Patient
- Older, history of HTN, smoking, Marfan syndrome, bicuspid aortic valve

Presentation
- Sudden “ripping” or “tearing” chest pain radiating to back

Physical Exam
- Asymmetric pulses and BP, aortic regurgitation

Diagnostic Studies
- Chest X-ray will show a widened mediastinum
- CT (if stable) or transesophageal echocardiogram (unstable) establishes diagnosis

Management
- Focused on reducing BP and HR (beta-blockers followed by sodium nitroprusside)
- Consider surgical correction depending on location of dissection

Management
Anti-impulse therapy
- Reduced BP to lowest tolerable level (SBPs 100-120 mm Hg)
- Reduce HR < 60 bpm
- Intravenous beta-blockers (esmolol, labetalol, propranolol)
- Nitroprusside (only after HR is controlled)
- Pain control

Stanford Type A
- Involves Ascending aorta
- Surgical emergency

Stanford Type B
- Involves only descending aorta

Notes
Aortic Dissection cont.

Tissue layers

Aortic dissection

- Intima
- Media
- Adventitia
- Internal tear

Notes
II. Arterial Embolism/Thrombosis

A. 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)

Management
• IV heparin → emergent surgical embolectomy or bypass

Comments
• Most common site is femoral artery bifurcation

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
B. Superficial Thrombophlebitis

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

Physical Exam
• Induration, erythema, 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
C. Superficial Phlebitis

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

**Diagnosis**
- Clinical
- Ultrasonography

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

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial phlebitis</td>
<td>The presence of pain and inflammation involving a vein in the absence of thrombus</td>
</tr>
<tr>
<td>Superficial thrombophlebitis</td>
<td>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</td>
</tr>
<tr>
<td>Superficial vein thrombosis</td>
<td>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 &quot;superficial vein thrombosis&quot;</td>
</tr>
</tbody>
</table>
III. Arterial/Venous Ulcer Disease

A. Venous ulcers

Pathophysiology
• **Chronic defects** of the skin that fail to heal spontaneously and persist for longer than 4 weeks

Patient
• Older with a history of smoking, **chronic venous insufficiency**, or DVT

Physical Exam
• **Most commonly** located in the lower leg just above the ankle
• Relatively painless, surrounded by brown-stained skin and/or dry, itchy, and reddened skin

Management
• First-line treatment of ulcers includes **below-knee compression therapy**, leg elevation and exercises
• Surgical debridement
B. Venous Stasis Ulcer

**Clinical**
- Location between the knee and ankle
- Medial and lateral malleoli are the most common sites
- Surrounding dermatitis
- Pain is not severe
- Hyperpigmentation, lipodermatosclerosis, and stasis dermatitis

**Management**
- Local wound care
- Compression therapy
- Skin grafting (if unhealed after 12 months of therapy)

---

Notes
C. Arterial Ulcers

Pathophysiology
- Occlusive atherosclerotic disease, m/c site of occlusion SFA

Symptoms
- Intermittent claudication, rest pain often prominent at night (suggests severe ischemia)

Clinical Features
- Diminished or absent pulses, atrophy, shiny skin, decreased hair growth, thick toenails, decreased skin temperature, ischemic ulcers (on the toes, lateral malleolus)

Treatment
- For intermittent claudication conservative management (smoking cessation, antithrombotic therapy, exercise therapy, control of HTN, hyperlipidemia, diabetes, cilostazol)
- If rest pain, ischemic ulcerations present, surgery indicated
IV. Chest Pain

HISTORY OF ANGINA

A. Anginal Chest Pain

Notes
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
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.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td></td>
</tr>
<tr>
<td>Highly suspicious</td>
<td>2</td>
</tr>
<tr>
<td>Moderately suspicious</td>
<td>1</td>
</tr>
<tr>
<td>Slightly suspicious</td>
<td>0</td>
</tr>
<tr>
<td>ECG</td>
<td></td>
</tr>
<tr>
<td>Significant ST depression</td>
<td>2</td>
</tr>
<tr>
<td>Nonspecific repolarization disturbance</td>
<td>1</td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
</tr>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>&gt; 65 years</td>
<td>2</td>
</tr>
<tr>
<td>45 to 65 years</td>
<td>1</td>
</tr>
<tr>
<td>&lt; 45 years</td>
<td>0</td>
</tr>
<tr>
<td>Risk factors</td>
<td></td>
</tr>
<tr>
<td>&gt; 3 risk factors or history of atherosclerotic disease</td>
<td>2</td>
</tr>
<tr>
<td>1 or 2 risk factors</td>
<td>1</td>
</tr>
<tr>
<td>No risk factors known</td>
<td>0</td>
</tr>
<tr>
<td>Troponin</td>
<td></td>
</tr>
<tr>
<td>Use local assays and corresponding cutoffs</td>
<td></td>
</tr>
<tr>
<td>&gt; 2x normal limit</td>
<td>2</td>
</tr>
<tr>
<td>1 to 2x normal limit</td>
<td>1</td>
</tr>
<tr>
<td>≤ normal limit</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Heart Score</th>
<th>Initial troponin</th>
<th>Risk</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 3 points</td>
<td>Low</td>
<td></td>
<td>Repeat troponin at 3 hours and if negative, discharge home with outpatient follow-up.</td>
</tr>
<tr>
<td>1 to 2 points</td>
<td>High</td>
<td></td>
<td>Cardiology consultation and admission recommended. Further testing indicated.</td>
</tr>
<tr>
<td>≥ 4 points</td>
<td>High</td>
<td></td>
<td>Admit to hospital or observation. Further testing indicated.</td>
</tr>
<tr>
<td>1 to 2 points</td>
<td>High</td>
<td></td>
<td>Cardiology consultation and admission recommended. Further testing indicated.</td>
</tr>
</tbody>
</table>
### D. Causes of Nontraumatic Chest Pain in Children and Adolescents

<table>
<thead>
<tr>
<th>Life-threatening (1 to 6%)</th>
<th>Cardiac conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td>• Aortic stenosis</td>
</tr>
<tr>
<td></td>
<td>• Coarctation of the aorta</td>
</tr>
<tr>
<td></td>
<td>• Coronary artery abnormalities</td>
</tr>
<tr>
<td></td>
<td>• Variant angina after recreational drug use</td>
</tr>
<tr>
<td></td>
<td>• Classic angina</td>
</tr>
<tr>
<td></td>
<td>• Pericarditis</td>
</tr>
<tr>
<td></td>
<td>• Myocarditis</td>
</tr>
<tr>
<td></td>
<td>• Dilated cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td>• Tachyarrhythmias</td>
</tr>
<tr>
<td></td>
<td>• Aortic aneurysm or dissection</td>
</tr>
<tr>
<td></td>
<td>• Ruptured sinus of Valsalva aneurysm</td>
</tr>
<tr>
<td></td>
<td>• Airway foreign body</td>
</tr>
<tr>
<td></td>
<td>• Spontaneous pneumothorax</td>
</tr>
<tr>
<td></td>
<td>• Pulmonary embolism</td>
</tr>
<tr>
<td></td>
<td>• Pulmonary hypertension</td>
</tr>
<tr>
<td></td>
<td>• Sickle cell disease with acute chest syndrome</td>
</tr>
<tr>
<td></td>
<td>• Tumor Nontraumatic esophageal rupture (Boerhaave syndrome)</td>
</tr>
<tr>
<td></td>
<td>• Spinal cord compression</td>
</tr>
<tr>
<td>Common conditions (94 to 98%)</td>
<td>Musculoskeletal conditions</td>
</tr>
<tr>
<td></td>
<td>• Muscle strain</td>
</tr>
<tr>
<td></td>
<td>• Costochondritis</td>
</tr>
<tr>
<td></td>
<td>• Slipping rib syndrome</td>
</tr>
<tr>
<td></td>
<td>• Precordial catch (Tetralogy of Fallot)</td>
</tr>
<tr>
<td></td>
<td>• Fibromyalgia</td>
</tr>
<tr>
<td></td>
<td>• Pectus excavatum or carinatum</td>
</tr>
<tr>
<td></td>
<td>• Anxiety</td>
</tr>
<tr>
<td></td>
<td>• Panic disorder with or without hyperventilation syndrome</td>
</tr>
<tr>
<td></td>
<td>• Depression</td>
</tr>
<tr>
<td></td>
<td>• Hypochondriasis</td>
</tr>
<tr>
<td></td>
<td>• Somatization</td>
</tr>
<tr>
<td>Respiratory</td>
<td>• Pneumonia (can be life-threatening)</td>
</tr>
<tr>
<td></td>
<td>• Asthma (can be life-threatening)</td>
</tr>
<tr>
<td></td>
<td>• Chronic cough with muscle strain or, if severe, fractured rib</td>
</tr>
<tr>
<td></td>
<td>• Spontaneous pneumomediastinum</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>• Gastroesophageal reflux disease</td>
</tr>
<tr>
<td></td>
<td>• Medication-induced (&quot;pill&quot;) esophagitis</td>
</tr>
<tr>
<td></td>
<td>• Esophageal foreign body</td>
</tr>
<tr>
<td></td>
<td>• Esophageal spasm and achalasia</td>
</tr>
<tr>
<td></td>
<td>• Gastritis</td>
</tr>
<tr>
<td></td>
<td>• Peptic ulcer</td>
</tr>
<tr>
<td></td>
<td>• Irritable bowel disease</td>
</tr>
<tr>
<td></td>
<td>• Cholecystitis</td>
</tr>
<tr>
<td></td>
<td>• Pancreatitis</td>
</tr>
<tr>
<td>Breast</td>
<td>• Male adolescents: Gynecomastia</td>
</tr>
<tr>
<td></td>
<td>• Female adolescents: Pregnancy, thelarche, mastitis, or fibrocystic disease</td>
</tr>
<tr>
<td>Other conditions</td>
<td>• Tietze syndrome</td>
</tr>
<tr>
<td></td>
<td>• Pleurodynia</td>
</tr>
<tr>
<td></td>
<td>• Herpes zoster</td>
</tr>
</tbody>
</table>
V. Claudication

A. Claudication

Pathophysiology
• Reduced arterial blood supply cannot meet the metabolic demand of the muscles utilized during walking
• Symptom of peripheral arterial disease (PAD)

Presentation
• Pain in the leg with walking, relieved within a few minutes of rest
• Reproducible at the same walking distance each time
• Buttock and hip: Aortoiliac disease
• Thigh: Aortoiliac or common femoral artery
• Upper two-thirds of the calf: Superficial femoral artery
• Lower one-third of the calf: Popliteal artery
• Foot claudication: Tibial or peroneal artery
VI. Peripheral Arterial Disease

A. Peripheral Artery Disease

Pathophysiology
- Most commonly caused by atherosclerotic disease
- Most common site of occlusion is superficial femoral artery

Risk Factors
- Smoking, diabetes, hyperlipidemia, hypertension, and coronary artery disease

Presentation
- Pain in affected extremity related to the activity (intermittent claudication), rest pain, especially worse at night and awakens pt from sleep

Physical Exam
- Cool extremities with absent or diminished pulses, muscular atrophy, loss of hair, thick toenails, ulcers (lateral malleolus)

Diagnostic Studies
- Ankle-brachial index initial diagnostic test
Peripheral Artery Disease cont.

- Peripheral vascular ulcer
- Decreased pulses

- Cool, shiny extremity with decreased hair
- Rest pain is late finding

Ankle-brachial index (ABI)
- < 0.9 indicates > 50% stenosis
- < 0.4 indicates ischemia
VII. Dyspnea on Exertion

A. Dyspnea on Exertion

• Difficulty breathing when engaged in a simple activity like walking up stairs or going a short distance
### B. Physical Examination Signs of Dyspnea

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

### Reflex (neurally mediated)

<table>
<thead>
<tr>
<th>Vasovagal</th>
<th>Situational</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mediated by orthostatic or emotional stress</td>
<td>Cough, sneeze, micturition, postexercise, postprandial</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Carotid sinus</th>
<th>Unknown</th>
</tr>
</thead>
</table>

### Orthostatic

<table>
<thead>
<tr>
<th>Primary autonomic</th>
<th>Secondary autonomic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure autonomic, Parkinsons, Lewy body dementia</td>
<td>Diabetes, amyloidosis, uremia, spinal cord injuries</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Drug induced</th>
<th>Volume depletion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol, vasodilators, diuretics, phenothiazines, antidepressants</td>
<td>Hemorrhage diarrhea, vomiting</td>
</tr>
</tbody>
</table>

### Cardiac

<table>
<thead>
<tr>
<th>Bradydysrhythmia</th>
<th>Tachydysrhythmia</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV block, tachy-brady syndrome</td>
<td>Supraventricular tachycardia, ventricular tachycardia</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Structural heart disease</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug-induced, valvular disease, ischemia, cardiomyopathy, tamponade</td>
<td>Pulmonary embolism, aortic dissection, pulmonary hypertension</td>
</tr>
</tbody>
</table>

### Notes

- Pure autonomic, Parkinsons, Lewy body dementia
- Tachydysrhythmia
- Secondary autonomic diseases
  - Diabetes, amyloidosis, uremia, spinal cord injuries
- Drug induced
  - Alcohol, vasodilators, diuretics, phenothiazines, antidepressants
- Volume depletion
  - Hemorrhage diarrhea, vomiting
- Bradydysrhythmia
  - AV block, tachy-brady syndrome
- Tachydysrhythmia
  - Supraventricular tachycardia, ventricular tachycardia
- Structural heart disease
  - Drug-induced, valvular disease, ischemia, cardiomyopathy, tamponade
- Other
  - Pulmonary embolism, aortic dissection, pulmonary hypertension
IX. Varicose Veins

A. Varicose Veins

Patient
• Woman

Presentation
• Dull ache in legs after prolonged standing

Physical Exam
• Dilated, elongated subcutaneous veins

Diagnostic Studies
• Duplex ultrasound establishes diagnosis

Management
• Leg elevation and compression stockings

Varicose Veins
→ Subcutaneous dilated, tortuous veins > 3 mm in diameter

Notes
B. Chronic Venous Insufficiency

Pathophysiology
- Inadequate muscle pump function, incompetent valves, and venous thrombosis leading to elevated venous pressure

Patient
- Obese or older with a history of prolonged standing or family history of venous insufficiency, previous DVT

Presentation
- Leg edema, worse at the end of the day, worsened by inactivity, relieved by leg elevation

Physical Exam
- Skin changes, ulceration (most common location above the medial malleolus), edema, varicose veins

Diagnostic Studies
- History and physical are diagnostic

Management
- Leg elevation, exercise, compression therapy, ulcer care, ablation

![Diagram showing normal venous blood flow and venous insufficiency with valvular abnormality leading to venous insufficiency.](image)

Edema | Varicose veins | Skin changes | Ulcer

Notes
CHAPTER 4

Endocrinology
ADRENAL CARCINOMA
HEAT/COLD INTOLERANCE
HYPERPARATHYROIDISM
PALPITATIONS

THYROID CARCINOMA
THYROID NODULES
TREMORS
I. Adrenal Carcinoma

A. Adrenal Carcinoma

**Pathophysiology**
- **Functioning** (hormone-secreting):
  - pheochromocytomas, aldosteronomas, and cortisol-producing adenomas
- **Nonfunctioning** (not hormone-secreting): most common type, usually an incidental finding

**Diagnostic Studies**
- Plasma fractionated metanephrines or 24-hour urine metanephrines
- Serum potassium, aldosterone, plasma renin activity
- 24-hour urinary-free cortisol or dexamethasone suppression test
- DHEA-S

**Management**
- Functioning: treat disorder
- Nonfunctioning: resect if > 4 cm
## B. Evaluation of the Patient with an Incidentally-Discovered Adrenal Mass

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Suggestive clinical features</th>
<th>Laboratory testing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pheochromocytoma</strong></td>
<td>• Hypertension</td>
<td>24-hour urine</td>
</tr>
<tr>
<td></td>
<td>• Paroxysmal symptoms</td>
<td>• Fractionated metanephrines</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Fractionated catecholamines</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Blood</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Fractionated metanephrines</td>
</tr>
<tr>
<td><strong>Cushing syndrome</strong></td>
<td>• Central obesity</td>
<td>(+) symptoms of Cushing syndrome</td>
</tr>
<tr>
<td></td>
<td>• Proximal muscle weakness</td>
<td>• 24-hour urinary free cortisol</td>
</tr>
<tr>
<td></td>
<td>• Thin lip</td>
<td>(-) symptoms of Cushing syndrome</td>
</tr>
<tr>
<td></td>
<td>• Supraclavicular fat pad</td>
<td>• 1 mg overnight dexamethasone suppression test</td>
</tr>
<tr>
<td></td>
<td>• Facial plethora</td>
<td></td>
</tr>
<tr>
<td><strong>Primary aldosteronism</strong></td>
<td>• Hypertension</td>
<td><em>Plasma aldosterone concentration</em></td>
</tr>
<tr>
<td></td>
<td>• Hypokalemia</td>
<td><em>Plasma renin activity</em></td>
</tr>
<tr>
<td><strong>Adrenocortical carcinoma</strong></td>
<td>• Mass effect symptoms</td>
<td><em>Serum DHEAS</em></td>
</tr>
<tr>
<td></td>
<td>• Symptoms related to excess glucocorticoid, mineralocorticoid, androgen, or estrogen secretion</td>
<td><em>Measures of clinically-induced steroid</em></td>
</tr>
</tbody>
</table>

---

**Notes**
C. Pheochromocytoma

Pathophysiology

• **Most commonly** caused by a catecholamine-secreting tumor located in the adrenal glands

Presentation

• Headaches, flushing, tremors, and vision changes

Physical Exam

• Hypertension

Diagnostic Studies

• Assay of **urinary catecholamines and metanephrines**, and **plasma metanephrine levels**

Management

• Surgery; treat with **alpha-blocker** (phenoxybenzamine) **prior** to beta blockade to prevent unopposed alpha agonism

**Notes**

Adrenal cortex
Adrenal medulla
Pheochromocytoma
Kidney

Adrenal cortex
Adrenal medulla

1. Episodic headache
2. Diaphoresis
3. Tachycardia with hypertension

**Diagnosis**

• **Fractionated metanephrines** and **catecholamines** in 24-hour urine collection
  • Plasma fractionated metanephrines
  • Normetanephrine, norepinephrine

**Treatment**

• Surgical resection of tumor
• Phenoxybenzamine (preop)
• Phentolamine (acute hypertensive crisis)
• Sodium nitroprusside (acute hypertensive crisis)
• Nicardipine (acute hypertensive crisis)
II. Heat/Cold Intolerance

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

• PTU if Pregnant
II. Heat/Cold Intolerance

B. Graves Disease

1. **Hyperthyroidism**

   - Thyroid-Stimulating Immunoglobulins

   - Thyroid-Stimulating Hormone Receptor (TSHR)

   - Increased T3, T4 [Low TSH]

   - **Hyperthyroidism**

2. **Goiter**

3. **Eye Disease**

   - **Exophthalmos**
   - Abnormal connective tissue deposition

4. **Pretibial or Localized Edema**

   - An infiltrative dermopathy with waxy, discolored induration of the skin
C. 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
D. Hashimoto Thyroiditis

→ Chronic autoimmune thyroiditis

**Most common cause of hypothyroidism in iodine-sufficient areas of the world**

May present initially as **hyperthyroidism**

Clinical

- Fatigue
- Weight gain
- Cold intolerance
- Dry skin
- Constipation
- Menstrual irregularities
- Depression

Treatment

- Levothyroxine
E. Myxedema Coma

→ Severe hypothyroidism resulting in a decompensated metabolic state and mental status change

- Thinning hair
- Hair loss
- Puffy face
- Enlarged thyroid
- Bradycardia
- Poor appetite
- Infertility
- Heavy menstruation
- Carpal tunnel syndrome
- Cool extremities and swelling of the limbs
- Loss of eyebrow hair
- Myxedema
  Thickened, nonpitting edema of skin
- Hypothermia

Precipitating factors
- Infection
- Cold exposure
- Stroke
- Meds (amiodarone, lithium)

Laboratory findings
- Hypoglycemia, hyponatremia
- Hypoxemia, hypercapnia
- Prolonged QT, low voltage
- Pericardial effusion

Management
- Supportive (airway, rewarming)
- Hydrocortisone
- Levothyroxine (T4)
- +/- T3 supplementation

Notes
III. Hyperparathyroidism

A. Hyperparathyroidism

Pathophysiology
- Most commonly caused by an adenoma with unregulated overproduction of PTH

Diagnostic Studies
- High PTH, high calcium, low phosphorus

Primary Hyperparathyroidism

Parathyroid hormone (PTH)

Calcium

Phosphorus

Notes
### IV. Palpitations

**A. Palpitations:** A noticeably rapid, strong, or irregular heartbeat

**B. Causes of Palpitations**

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>Any dysrhythm, Cardiac and Extracardiac shunts, Valvular heart disease, Pacemaker, Atrial myxoma, Cardiomyopathy</td>
</tr>
<tr>
<td>Psychiatric disease</td>
<td>Panic attack and disorder, Generalized anxiety disorder, Somatization, Depression</td>
</tr>
<tr>
<td>Medications</td>
<td>Sympathomimetic agents, Vasodilators, Anticholinergic drugs, Beta-blocker withdrawal</td>
</tr>
<tr>
<td>Habits</td>
<td>Cocaine, Amphetamines, Caffeine, Nicotine</td>
</tr>
<tr>
<td>Metabolic disorders</td>
<td>Hypoglycemia, Thyrotoxicosis, Pheochromocytoma, Mastocytosis, Scombroid food poisoning</td>
</tr>
<tr>
<td>High output states</td>
<td>Anemia, Pregnancy, Paget disease, Fever</td>
</tr>
<tr>
<td>Catecholamine excess</td>
<td>Stress, Exercise</td>
</tr>
</tbody>
</table>

**Notes**
V. Thyroid Carcinoma

A. Thyroid Carcinoma

Pathophysiology
- Most common type is papillary
- Worst prognosis is anaplastic

Patient
- Radiation exposure is the most common risk factor

Diagnostic Studies
- Ultrasound
- Thyroid uptake scan
  - No uptake indicates it is “cold”, need to rule out cancer, consider fine needle aspiration (FNA)

Management
- Complete or partial removal of the thyroid
## Thyroid Carcinoma

<table>
<thead>
<tr>
<th>Type</th>
<th>Papillary</th>
<th>Follicular</th>
<th>Medullary</th>
<th>Anaplastic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proportion of thyroid cancer</td>
<td>85%</td>
<td>12%</td>
<td>2%</td>
<td>1%</td>
</tr>
<tr>
<td>Age of incidence</td>
<td>35–40 yrs</td>
<td>30–60 yrs</td>
<td>Isolated: 40–50 yrs</td>
<td>&gt; 60 yrs</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Genetic: 10–20 yrs</td>
<td></td>
</tr>
<tr>
<td>Cell differentiation</td>
<td>Well differentiated</td>
<td>Well differentiated</td>
<td>Intermediately differentiated</td>
<td>Poorly differentiated</td>
</tr>
<tr>
<td>Primary mode of spread</td>
<td>Lymphatic</td>
<td>Hematologic</td>
<td>Lymphatic</td>
<td>Lymphatic</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Hoarseness Lymphadenopathy</td>
<td>Hoarseness Lymphadenopathy (rare)</td>
<td>Diarrhea Flushing</td>
<td>Shortness of breath Hoarseness Lymphadenopathy</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>FNA</td>
<td>FNA and CNB to confirm</td>
<td>Fam history Previous MEN dx FNA Calcitonin level</td>
<td>CNB Surgical biopsy</td>
</tr>
<tr>
<td>Treatment</td>
<td>Surgery Radioablation TSH suppression</td>
<td>Surgery Radioablation TSH suppression</td>
<td>Surgery External beam radiation</td>
<td>Early: Surgery Late: Palliation</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Excellent</td>
<td>Very good</td>
<td>Very good</td>
<td>Poor</td>
</tr>
</tbody>
</table>

FNA: Fine needle aspiration  
CNB: Coarse needle biopsy  
MEN: Multiple endocrine neoplasia
VI. Thyroid Nodule

A. Red Flags for Thyroid Nodule

- Male gender
- Age < 20 or > 65 years
- History of head/neck irradiation
- Family history of thyroid cancer
- Hard, fixed nodule > 4 cm
- Rapid growth of the nodule
- Symptoms related to local invasion (dysphagia, hoarseness)
B. Initial Evaluation of a Patient with a Thyroid Nodule

Thyroid nodule found clinically or incidentally on imaging

- TSH, thyroid ultrasound
  - TSH normal or elevated
    - Nodule is nonfunctional
      - Meets sonographic criteria for FNA
        - Yes: Fine-needle aspiration
        - No: Monitor
  - TSH subnormal
    - Radionuclide thyroid scan
      - Measure FT4, T3
        - FT4 and T3 normal
          - Subclinical hyperthyroidism
        - FT4 or T3 or both high
          - Overt hyperthyroidism
            - Observe in most cases
            - Treat
### VII. Tremors

#### A. Tremors

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
<th>Physical Exam</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Resting</strong></td>
<td>Tremor at rest</td>
<td>• Observe at rest</td>
<td>Parkinson’s disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Frequency 4-6 Hz</td>
<td>Parkinsonism tremor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Associated with hypokinesia or rigidity (parkinsonism)</td>
<td>(medications)</td>
</tr>
<tr>
<td><strong>Postural</strong></td>
<td>Tremor with sustained posture of an extremity</td>
<td>• Ask patient to extend arms and hold</td>
<td>Essential tremor</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Increased physiologic tremor</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Wilson’s disease</td>
</tr>
<tr>
<td><strong>Intention</strong></td>
<td>Tremor during movement that increases as a target is approached</td>
<td>• No tremor at rest</td>
<td>Cerebellar disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• May be associated with cerebellar signs</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Chronic alcohol use</td>
</tr>
</tbody>
</table>

---

**Notes**
B. Essential Tremor

**Most common cause of action tremor in adults**

Activated by **voluntary movement** or when arms are held in a **fixed posture against gravity**

Amplified by **goal-directed movements**

**Clinical**
- Classically affects the hands and arms
- Typically bilateral, often slightly asymmetric
- Can also affect the head, voice, face, and trunk

**International Parkinson and Movement Disorder Society Diagnostic Criteria for Essential Tremor**
- Isolated tremor consisting of bilateral upper limb action tremor, without other motor abnormalities
- At least three years in duration
- With or without tremor in other locations (e.g., head, voice, or lower limbs)
- Absence of other neurologic signs, such as dystonia, ataxia, or parkinsonism

**Management**
- Propranolol or other beta-blockers
- Primidone
- Gabapentin
- Topiramate
- Alcohol (ethanol)
- Botulinum toxin
CHAPTER 5

Dermatology
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<th>Topic</th>
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<td>SQUAMOUS CELL CARCINOMA</td>
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<td>209</td>
<td>URTICARIA</td>
</tr>
</tbody>
</table>
I. Basal Cell Carcinoma

A. Basal Cell Carcinoma

Presentation
• Painless, slow-growing lesion on the face, ears, or neck

Physical Exam
• Pearly papule with rolled borders and telangiectasia

Diagnostic Studies
• Shave biopsy

Management
• Surgical excision

Comments
• Most common skin cancer

Risk Factors
• Inability to tan
• Intermittent sun exposure
• Cumulative sun exposure
• Fair skin
• Older age

Diagnosis
• Clinical appearance
• Biopsy

Management
• Electrodesiccation and curettage
• Surgical excision
• Radiation
• Superficial form may respond to topical immune modulator (imiquimod)
• Mohs micrographic surgery used to sclerosing and other types with poorly defined clinical margins or located within the facial mask area

Notes
Basal Cell Carcinoma cont.

Nodular ulcerative
- Most common type
- Well-circumscribed pearly papule
- Telangiectasias

Superficial
- Well-circumscribed erythematous, scaly patch
- Resembles tinea, psoriasis, or dermatitis
- Often occurs on non-sun-exposed skin

Sclerotic
- Whitish plaque with sclerosis or fibrosis on palpation

Pigmented
- Similar to nodular but are pigmented
- Most common form seen in darker skinned individuals
## II. Burns

### A. Burn Classification

<table>
<thead>
<tr>
<th>Clinical appearance</th>
<th>Thickness</th>
<th>Degree</th>
<th>Depth</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial</td>
<td>Superficial</td>
<td>1st</td>
<td>Epidermis</td>
<td>Pain, redness, mild swelling</td>
</tr>
<tr>
<td>Superficial Partial</td>
<td></td>
<td></td>
<td>Dermis: papillary region</td>
<td>Pain, blisters, splotchy skin, severe swelling</td>
</tr>
<tr>
<td>Deep partial</td>
<td></td>
<td>2nd</td>
<td>Dermis: reticular region</td>
<td>White, leathery, relatively painless</td>
</tr>
<tr>
<td>Full</td>
<td></td>
<td>3rd</td>
<td>Hypodermis (subcutaneous tissue)</td>
<td>Charred, insensate, eschar formation</td>
</tr>
</tbody>
</table>
Burn Classification cont.

**Skin Layers**
- Epidermis
- Dermis
- Subcutaneous fat
- Muscle

**Burn Depth**
- Superficial (was 1st degree)
- Superficial partial-thickness
- Deep partial-thickness
- Full partial-thickness
- Fourth degree

Notes
B. Rule of Nines

Only for 2nd and 3rd degree burns

Patient's PALM approximates 1% total body surface area

Notes
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

Comments
- Cat bite (*Pasteurella multocida*): Augmentin or doxycycline if PCN allergic
- Puncture wound (cover for *pseudomonas* if through shoe): Ciprofloxacin

Notes
B. Surgical Site Infection

**Clinical**
- Localized erythema, induration, warmth, and pain at incision site
- Purulent wound drainage and separation of the wound may occur
- Some patients have systemic evidence (e.g., fever, leukocytosis)

**Necrotizing fasciitis** (most serious wound infection)
- Copious, dishwater-like drainage
- Dusky and friable subcutaneous tissue
- Pale and devitalized fascia

Notes
IV. Discharge

BREAST/NIPPLE DISCHARGE • GU

1. BREAST/NIPPLE DISCHARGE

A. Nipple Discharge

<table>
<thead>
<tr>
<th>Discharge type</th>
<th>Characteristics</th>
<th>Common Causes</th>
</tr>
</thead>
</table>
| Physiologic (galactorrhea): Nonpathologic nipple discharge unrelated to pregnancy or breastfeeding | • Usually bilateral  
• White or clear  
• May be straw-colored, green, brown, gray | • Hyperprolactinemia  
• Medications  
• Neurogenic stimulation |
| Pathologic (suspicious): Secretory production of fluids other than milk may be due to a pathological process in the breast | • Usually unilateral  
• Localized to single duct  
• Persistent  
• Spontaneous  
• Serous, clear, yellow  
• Sanguineous  
• Serosanguineous | • Papilloma |

Notes
B. Management of Spontaneous Nipple Discharge

History and physical exam: No palpable mass

Abnormal imaging
- Refer to surgeon

Normal imaging*

- Single duct
  - Refer to surgeon

- Multiple ducts
  - Bloody
    - Refer to surgeon
  - Non-bloody
    - Medical evaluation
      - Consider galactorrhea work up
      - Continue routine screening

*Breast ultrasound recommended for all patients with nipple discharge. Mammograms are recommended for women ≥ age 30
2. GU

A. Vaginal Infections

<table>
<thead>
<tr>
<th></th>
<th>Candidiasis</th>
<th>Trichomoniasis</th>
<th>Bacterial vaginosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Discharge</strong></td>
<td>White</td>
<td>Green/yellow</td>
<td>Gray/white</td>
</tr>
<tr>
<td><strong>Color</strong></td>
<td>Thick, curdy, white discharge</td>
<td>Frothy, green/yellow discharge</td>
<td>Thin, white, grey discharge</td>
</tr>
<tr>
<td><strong>pH</strong></td>
<td>&lt; 4.5</td>
<td>&gt; 5</td>
<td>&gt; 4.5</td>
</tr>
<tr>
<td><strong>Amine odor with KOH</strong></td>
<td>Negative</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td><strong>Wet mount</strong></td>
<td>Pseudohyphae</td>
<td>Flagella, Undulating membrane</td>
<td>Bacteria, Clue cell, epithelial cell</td>
</tr>
<tr>
<td></td>
<td>WBC, Spores, Pseudohyphae</td>
<td>WBC, Motile trichomonads</td>
<td>Few WBCs, Clue cells</td>
</tr>
</tbody>
</table>

Notes
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 reaction manifestation

Notes
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)
### C. Classic Drug Reaction Patterns

<table>
<thead>
<tr>
<th>Type</th>
<th>Characteristics</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drug-induced exanthems</strong></td>
<td>• Most common cutaneous drug reaction</td>
<td></td>
</tr>
<tr>
<td><strong>Symmetrical drug-related intertriginous and flexural exanthema</strong></td>
<td>• Sharply demarcated&lt;br&gt;• Involvement of flexural or intertriginous fold&lt;br&gt;• No systemic symptoms</td>
<td></td>
</tr>
<tr>
<td><strong>Urticaria</strong></td>
<td>• Pruritic&lt;br&gt;• Raised&lt;br&gt;• Central pallor</td>
<td></td>
</tr>
<tr>
<td><strong>Cutaneous small vessel vasculitis</strong></td>
<td>• A single organ vasculitis</td>
<td></td>
</tr>
<tr>
<td><strong>Exfoliative dermatitis</strong></td>
<td>• Chronic erythema and scale involving &gt; 90% of body surface</td>
<td></td>
</tr>
<tr>
<td><strong>Stevens-Johnson syndrome</strong></td>
<td>• Severe mucocutaneous reaction&lt;br&gt;• &lt; 10% body surface</td>
<td></td>
</tr>
<tr>
<td><strong>Toxic epidermal necrolysis</strong></td>
<td>• Severe mucocutaneous reaction&lt;br&gt;• &gt; 30% body surface</td>
<td></td>
</tr>
<tr>
<td><strong>Erythema multiforme</strong></td>
<td>• Target lesions</td>
<td></td>
</tr>
<tr>
<td><strong>Drug reaction with eosinophilia and systemic symptoms (DRESS)</strong></td>
<td>• Idiosyncratic reaction&lt;br&gt;• Fever, malaise, lymphadenopathy</td>
<td></td>
</tr>
<tr>
<td><strong>Fixed drug reaction</strong></td>
<td>• Erythematous and edematous plaques with grayish center or frank bullae</td>
<td></td>
</tr>
</tbody>
</table>
VI. Melanoma

A. Melanoma

Patient
- Fair-skinned with history of severe blistering sunburns and family history of melanoma or dysplastic nevus syndrome

Presentation
- Itching, painful lesion that won’t heal

Physical Exam
- Ulcerated lesion and ABCDE
  - Asymmetry
  - Border irregularity
  - Color variation
  - Diameter
  - Evolution

Diagnostic Studies
- Biopsy: excisional or punch
- Depth is the most important factor (Breslow depth)

Management
- Excision with adequate margins; interferon reduces recurrence
Melanoma cont.

Squamous Cell Carcinoma
- Arises from superficial layers of keratinocytes
- Actinic keratosis is precursor
- Non-healing lesion that may bleed without trauma
- Location: Sun-exposed areas

Actinic Keratosis
- Also known as solar keratosis
- Atypical epidermal keratinocytes
- Precancerous for squamous cell

Melanoma
- Asymmetry
- Border irregularity
- Color variations
- Diameter > 6 mm

Basal Cell Carcinoma
- Most common skin cancer (USA)
- Pearly nodule
- "Rolled" raised edge
- Telangiectatic vessels

Notes
B. ABCDEs of Melanoma

**Asymmetry**

**Border irregularity**

**Color variations**

**Diameter**

(≥ 6 mm or 1/4 inch)

**Evolution**

Notes
C. Surgical Margins for Excision of Melanoma

<table>
<thead>
<tr>
<th>Tumor Thickness</th>
<th>Recommended Margin</th>
</tr>
</thead>
<tbody>
<tr>
<td>In situ</td>
<td>0.5 cm</td>
</tr>
<tr>
<td>≤ 1.0 mm</td>
<td>1.0 cm</td>
</tr>
<tr>
<td>1.01–2 mm</td>
<td>1–2 cm</td>
</tr>
<tr>
<td>2.01–4 mm</td>
<td>2.0 cm</td>
</tr>
<tr>
<td>&gt; 4 mm</td>
<td>2.0 cm</td>
</tr>
</tbody>
</table>

Notes
VII. Pressure Ulcers

A. Pressure Ulcer Stages

**Stage 1**
Nonblanchable erythema
- Intact skin
- May be painful

**Stage 2**
Partial thickness
- Shallow open ulcer
- Red/pink wound bed

**Stage 3**
Full-thickness skin loss
- Subcutaneous fat may be visible
- Bone, muscle, tendon not exposed
- May include tunneling

**Stage 4**
Full-thickness tissue loss
- Exposed bone, tendon, or muscle
- Slough or eschar may be present
- Undermining and tunneling

Notes
VIII. Rash

A. Rashes

- **Zinc deficiency**: perioral pustular rash

- **Paget disease**: intolerable **pruritus** and a well-demarcated, erythematous, eczematous rash

- **Herpes zoster**: classic **vesicular** lesions which develop **unilaterally** in a dermatomal distribution

- **Herpes simplex**: clear vesicles on an erythematous base with **crusting**
B. "Erythema" Rashes

**Erythema multiforme**
- Target-like lesions
- Infectious, medication, autoimmune

**Erythema marginatum**
- Macule with central clearing
- Spares the face
- Rheumatic fever

**Erythema nodosum**
- Inflammatory nodules
- Infectious, autoimmune, medication, pregnancy

**Erythema migrans**
- Bull's eye appearance
- Lyme disease

**Erythema infectiosum**
- Slapped cheek appearance
- Circumoral pallor
- Parvovirus B19 infection
IX. Redness/Erythema

**Erythema migrans**
- Bull’s eye lesion
- Associated with Lyme disease

**Erythema multiforme**
- Target lesion
- Viral, drugs, autoimmune

**Nummular eczema**
- Dermatitis
- Scales

**Granuloma annulare**
- Benign inflammatory dermatitis

---

Notes
### X. Squamous Cell Carcinoma

#### A. Squamous Cell Carcinoma

**Patient**
- History of HPV, chronic sun exposure, exposure to arsenic or radiation

**Presentation**
- Non-healing lesion that sometimes bleeds

**Physical Exam**
- Red, scaly, hyperkeratotic nodular, papule or plaque that does not itch.
  - **Most common** on lips, hands, neck, head (sun-exposed areas)

**Diagnostic Studies**
- Clinical exam, skin biopsy confirms

**Management**
- Wide local excision, radiation therapy

---

**Indurated and ulcerated papule**

- Arises from superficial layers of keratinocytes
- Actinic keratosis is precursor
- Non-healing lesion that may bleed without trauma
- Location: Sun-exposed areas
XI. 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 and 1:10,000 for IV route (Peds: epinephrine 0.01 mg/kg SC/IV)
Urticaria cont.

Raised, erythematous plaques

Central pallor

Pruritus

Allergen + Mast cell Histamine release

Common Causes of Urticaria

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<thead>
<tr>
<th>Drugs</th>
<th>Infection</th>
<th>Environmental</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Penicillin</td>
<td>• Epstein-Barr virus</td>
<td>• Heat</td>
</tr>
<tr>
<td>• Sulfur</td>
<td>• Hepatitis B virus</td>
<td>• Cold</td>
</tr>
<tr>
<td>• Aspirin</td>
<td>• Coxsackie virus</td>
<td>• Exercise</td>
</tr>
<tr>
<td>• Local anesthetics</td>
<td>• Parasitic infections</td>
<td></td>
</tr>
<tr>
<td>• Diuretics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• NSAIDs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Morphine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Codeine</td>
<td></td>
<td></td>
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<tr>
<td>• Progesterone</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Food</th>
<th>Other</th>
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</thead>
<tbody>
<tr>
<td>• Fish</td>
<td>• Latex</td>
</tr>
<tr>
<td>• Eggs</td>
<td>• Pregnancy</td>
</tr>
<tr>
<td>• Nuts</td>
<td>• Malignancy</td>
</tr>
<tr>
<td>• Shellfish</td>
<td></td>
</tr>
<tr>
<td>• Fruits</td>
<td></td>
</tr>
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Management

• Supportive care
• Antihistamines
• Glucocorticoids (if associated with angioedema)
CHAPTER 6

Neurology
CHAPTER 6
Neurology

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234 VASCULAR DISORDERS (CAROTID DISEASE)
I. Change in Speech

A. Aphasia

Pathophysiology
- Inability to comprehend or formulate language because of damage to specific brain regions
- Caused by stroke, hemorrhage, carotid disease, vascular disorders

Broca Aphasia
- Motor speech area
- Located in the frontal lobe
- Expressive aphasia (non-fluent aphasia)
- Broca is Broken

Wernicke Aphasia
- Sensory speech area
- Located in the parietal and temporal lobe
- Receptive aphasia (fluent aphasia), can't understand
- Wernicke is “What?”

Comments
- Recent thyroidectomy: recurrent laryngeal nerve injury
B. Types of Aphasia

1. Fluent?
   Is speech fluent?

2. Comprehends?
   Can you comprehend spoken messages?

3. Repeats?
   Can the person repeat words or phrases?

Global aphasia, Mixed transcortical aphasia, Broca aphasia, Transcortical motor aphasia, Wernicke aphasia, Transcortical sensory aphasia, Conduction aphasia, Anomic aphasia

Notes
C. Dysarthria

Pathophysiology
- Motor speech disorder ("slurred")
- Caused by brain injury or thrombotic or embolic stroke

Degenerative Diseases
- Parkinsonism, amyotrophic lateral sclerosis (ALS), multiple sclerosis, Huntington disease, Niemann-Pick disease, and Friedreich ataxia

Toxic and Metabolic Conditions
- Wilson disease, central pontine myelinolysis (when sodium deficiency or hyponatremia is corrected too rapidly)
II. Change in Vision

A. Perioperative Vision Loss

Pathophysiology
• Central retinal artery occlusion, ischemic optic neuropathy, and cerebral vision loss

Presentation
• Transient blurring of vision
  • Ocular ointments, excessive drying of the cornea, or corneal trauma
• Complete or partial visual loss
  • Surgical trauma, embolic events, acute anemia, hypotension
  • Anterior ischemic optic neuropathy
    • Ischemia to the ciliary blood vessels that supply the front, or anterior, portion of the optic nerve
    • Painless abrupt reduction in vision and optic disc pallor and swelling
### B. Painful Vision Loss vs. Painless Vision Loss

<table>
<thead>
<tr>
<th>Painful Vision Loss</th>
<th>Painless Vision Loss</th>
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<tbody>
<tr>
<td>Acute angle closure glaucoma</td>
<td>Retinal detachment</td>
</tr>
<tr>
<td>Optic neuritis</td>
<td>Central retinal artery occlusion</td>
</tr>
<tr>
<td>Temporal arteritis</td>
<td>Central retinal vein occlusion</td>
</tr>
<tr>
<td>Corneal abrasion/ulcer</td>
<td>Vitreous hemorrhage</td>
</tr>
<tr>
<td>Anterior uveitis</td>
<td>Amaurosis fugax</td>
</tr>
<tr>
<td>Iritis</td>
<td>Macular degeneration</td>
</tr>
<tr>
<td>Endophthalmitis</td>
<td>Cerebrovascular accident</td>
</tr>
</tbody>
</table>

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Notes
## III. Epidural Hematoma

### A. Epidural Hematoma

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

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

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

**Management**
- Emergent evacuation

<table>
<thead>
<tr>
<th><strong>Etiology</strong></th>
<th>Most common in adolescents and young adults</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Traffic accidents, falls, assaults</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Clinical</strong></th>
<th>Blood between dura and skull</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Loss of consciousness followed by lucid interval then deterioration</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Management</strong></th>
<th>Hematoma evacuation</th>
</tr>
</thead>
</table>
Epidural Hematoma cont.

- **Scalp**
- **Skull**
- **Fracture in skull**
- **Epidural hemorrhage**
- **Epidural hematoma**
- **Dura**
- **Compressed lateral ventricle**
- **Midline shift**
- **Biconvex/lens shaped epidural hematoma**

**Notes**
IV. Motor and/or Sensory Loss

SPINAL CORD INJURIES • NEUROPATHIES

1. SPINAL CORD INJURIES

A. 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

Notes
Brown-Séquard Syndrome cont.

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Mechanism</th>
<th>Clinical</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior cord</td>
<td>Flexion or vascular</td>
<td>Complete loss of motor, pain, &amp; temperature below injury, but retains proprioception and vibratory sensation</td>
<td>Poor</td>
</tr>
<tr>
<td>Central cord</td>
<td>Forced hyperextension</td>
<td>Sensory and motor deficit Upper &gt; Lower Extremities</td>
<td>Average</td>
</tr>
<tr>
<td>Brown-Séquard</td>
<td>Penetrating trauma</td>
<td>Ipsilateral loss of motor, vibratory sensation, and proprioception with contralateral loss of pain and temperature sensation</td>
<td>Good</td>
</tr>
</tbody>
</table>

**Notes**

- Ipsilateral loss of motor, vibratory, and proprioception
- Contralateral loss of pain and temperature sensation
B. 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

<table>
<thead>
<tr>
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<th>Clinical</th>
<th>Prognosis</th>
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<td>Ipsilateral loss of motor, vibratory sensation, and proprioception with contralateral loss of pain and temperature sensation</td>
<td>Good</td>
</tr>
</tbody>
</table>
C. Central Cord Syndrome

Patient
- History of hyperextension injury

Presentation
- Bilateral motor/sensory deficits
- Affecting upper extremities more significantly than lower

Comments
- Most common incomplete spinal cord syndrome

Notes
- Most commonly caused by extension injury

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Mechanism</th>
<th>Clinical</th>
<th>Prognosis</th>
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<td>Brown-Séquard</td>
<td>Penetrating trauma</td>
<td>Ipsilateral loss of motor, vibratory sensation, and proprioception with contralateral loss of pain and temperature sensation</td>
<td>Good</td>
</tr>
</tbody>
</table>
2. NEUROPATHIES

A. Guillain-Barré Syndrome

→ Acute immune-mediated polyneuropathies

Pathophysiology
- Most commonly identified precipitant
  *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

Campylobacter jejuni is most commonly identified precipitant
[Follows respiratory or GI illness]

1. Immune response to preceding infection
2. Cross-reacts with peripheral nerve components due to molecular mimicry
3. Resulting in demyelination

Diagnosis
- Clinical
- Lumbar puncture:
  Albuminocytologic dissociation
  (Elevated protein with normal/mild pleocytosis)

Management
- Supportive
- Measurement of vital capacity and negative inspiratory force (NIF)
- Plasmapheresis
- IVIG

Absent or depressed deep tendon reflexes

Progressive, ascending, symmetric muscle weakness

Notes
B. Meralgia Paresthetica

→ Lateral femoral cutaneous nerve entrapment

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

Clinical
• Burning pain
• Paresthesia
• Hypesthesia

Patient often rubs the outer thigh when describing symptoms

Notes
C. Complex Regional Pain Syndrome

Patient
- History of **previous extremity injury** or fracture

Presentation
- **Light touch causes extreme pain** and **alldynia** (pain felt from a non-painful 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

Onset of symptoms

“STAMP”

- **Sensory**
  - hypoa-/hyperalgesia
  - hypo-/hyperesthesia
- **Trophic**
  - skin, hair, nail changes
- **Autonomic**
  - swelling
  - edema
  - sweating
- **Motor**
  - weakness
  - contractures
  - atrophy
- **Pain**
D. Diabetic Neuropathy

Presentation
- Polyneuropathy
- Numbness, tingling
- “Stocking-glove” distribution

Management
- Pregabalin, duloxetine, TCAs

<table>
<thead>
<tr>
<th>Distal Symmetric Polyneuropathy of Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Large myelinated fibers</strong></td>
</tr>
<tr>
<td>Function</td>
</tr>
<tr>
<td>- Pressure</td>
</tr>
<tr>
<td>- Balance</td>
</tr>
<tr>
<td>Symptoms</td>
</tr>
<tr>
<td>- Numbness</td>
</tr>
<tr>
<td>- Tingling</td>
</tr>
<tr>
<td>- Poor balance</td>
</tr>
<tr>
<td>Exam</td>
</tr>
<tr>
<td>- Ankle reflexes: reduced/absent</td>
</tr>
<tr>
<td>- Vibration perception: reduced/absent</td>
</tr>
<tr>
<td>- 10-g monofilament: reduced/absent</td>
</tr>
<tr>
<td>- Proprioception: reduced/absent</td>
</tr>
</tbody>
</table>
V. Subarachnoid Hemorrhage

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)

Nimodipine
- A dihydropyridine (DHP) calcium channel blocker that prevents 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
### B. Time Following Subarachnoid Hemorrhage vs. CT Scan Sensitivity

<table>
<thead>
<tr>
<th>Time Following Subarachnoid Hemorrhage</th>
<th>CT Scan Sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 12 hrs</td>
<td>98% - 100%</td>
</tr>
<tr>
<td>24 hrs</td>
<td>93%</td>
</tr>
<tr>
<td>6 days</td>
<td>57%</td>
</tr>
</tbody>
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**Notes**
Notes
VI. Subdural Hematoma

A. 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
VI. Subdural Hematoma

Subdural Hematoma cont.

**SUBdural hematoma**
Blood collection between the dura and arachnoid matter

Crescent-shaped hematoma

Bridging veins
Alcoholics and Elderly

Notes
VII. Cerebral Hemorrhage (Review)

A. Cerebral Hemorrhages

Subdural hematoma
- Crescent-shaped
- Blood collection between dura and arachnoid matter
- Tear in bridging veins
- Alcoholics and elderly are prone

Epidermal 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
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
B. Stroke Distribution: 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

**Middle cerebral artery**

**Posterior cerebral artery**
C. Stroke Distribution: Middle Cerebral Artery

**Lateral**

- Anterior cerebral artery
- Middle cerebral artery
- Posterior cerebral artery

**Medial**

**Middle cerebral artery**
- Paralysis of contralateral face, arm, and leg
- Sensory impairment over contralateral face, arm, and leg
- Homonymous hemi- or quadrantopia
- Paralysis of gaze to opposite side
- Aphasia, dysarthria

Notes
D. Stroke Distribution: 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
E. Carotid Artery Stenosis

Pathophysiology
- Plaque buildup in the carotid artery

Management
- Aspirin, statin, BP control, and lifestyle changes
- **Symptomatic** carotid stenosis of 70 to 99 percent and a life expectancy of at least five years is an indication for a **carotid endarterectomy (CEA)**

Atherosclerotic plaque reduces blood flow in the internal carotid artery
F. Carotid Endarterectomy

→ Reduces the risk of ischemic stroke in patients with symptomatic carotid artery atherosclerosis

Indications for Carotid Endarterectomy

Recently symptomatic carotid stenosis of 70 to 99% and a life expectancy of > 5 years who meet following conditions:

- A surgically accessible carotid lesion
- Absence of clinically significant cardiac, pulmonary, or other disease that would greatly increase the risk of anesthesia and surgery
- No prior ipsilateral endarterectomy
- Perioperative risk of stroke and death with CEA for the surgeon or center is < 6%

Low-dose aspirin (81 to 325 mg/day) is recommended for all patients who are having a carotid endarterectomy. Aspirin should be started prior to surgery and continued for at least three months after surgery.

Notes
CHAPTER 7

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</tbody>
</table>
I. Acid/Base Disorders

A. Acute Respiratory and Metabolic Acidosis and Alkalosis

Pathophysiology
- **Respiratory Acidosis**: lungs fail to excrete \( \text{CO}_2 \) (Breathing too slow, holding on to \( \text{CO}_2 \))
- **Respiratory Alkalosis**: excessive elimination of \( \text{CO}_2 \) (Breathing too fast, blowing of \( \text{CO}_2 \))
- **Metabolic Acidosis**
  - How to calculate ion gap: \( \text{Na}^- - (\text{Cl}^- + \text{HCO}_3^-) = 8-16 \text{ mEq/L} \)
  - Increased ion gap metabolic acidosis
    - A CAT MUDPILE
    - Low anion gap
      - Loss of bicarbonate

Diagnostic Studies
- Normal ABG values:
  - pH: 7.35–7.45
  - \( \text{PaCO}_2 \): 35–45
  - \( \text{HCO}_3^- \): 22–26

Comments
- **Winter Formula** is used to evaluate respiratory compensation when metabolic acidosis is present in a patient; \( \text{pCO}_2 = (\text{HCO}_3^- \times 1.5) + 8 \pm 2 \)
- **Delta ratio** is used to assess elevated anion gap metabolic acidosis and to evaluate whether a mixed acid-base disorder is present
I. Acid/Base Disorders

Arterial pH

pH < 7.35
- Acidemia
  - HCO₃⁻ < 24 or PCO₂ > 40
    - Metabolic acidosis
    - Respiratory compensation
    - PCO₂ < 40
  - Respiratory acidosis
    - Renal compensation
    - HCO₃⁻ > 24

pH > 7.45
- Alkalemia
  - HCO₃⁻ > 24 or PCO₂ < 40
    - Metabolic alkalosis
    - Respiratory compensation
    - HCO₃⁻ < 24
  - Respiratory alkalosis
    - Renal compensation
    - PCO₂ > 40

Notes
B. Respiratory Acidosis

pH: < 7.35; PaCO₂: > 45

**Acute (Acute respiratory failure)**
- PaCO₂ is elevated (> 45 mm Hg)
- Accompanying acidemia (pH < 7.35)

**Chronic (COPD, Obesity hypoventilation syndrome)**
- PaCO₂ 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 PaCO₂)

2. **Renal compensation** occurs over 3 to 5 days [Excretion of carbonic acid, bicarbonate reabsorption] (3 to 5 mEq/L for each 10 mm Hg increase in PaCO₂)

**Change in pH**

**Acute:** \( \Delta pH \ 0.008 \times (40 - \text{PaCO}_2) \)

**Chronic:** \( \Delta pH \ 0.003 \times (40 - \text{PaCO}_2) \)
C. Respiratory Alkalosis

pH: > 7.45; PaCO$_2$: < 35

**Common Causes**
- Hyperventilation syndrome
- Anxiety, pain
- Fever, psychosis
- Pulmonary embolism
- Heart failure
- Mechanical ventilation
- Stroke
- Pregnancy
- Hyperthyroidism
- Salicylate toxicity
D. Metabolic Acidosis

pH: < 7.35; HCO$_3^-$: < 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 formula**
   
   \[ pCO_2 = 1.5 \times (HCO_3^-) + 8 +/- 2 \]
   
   If measured pCO$_2$ is > Winter formula pCO$_2$
   then concomitant respiratory acidosis
   If measured pCO$_2$ is < Winter formula pCO$_2$
   then concomitant respiratory alkalosis

2. **Anion gap = Na – [Cl$^-+HCO_3^-]$**
   
   > 11 is elevated

3. **Delta ratio**

   \[ \frac{\Delta \text{ Anion gap}}{\Delta \text{ HCO}_3^-} \]
   
   \( \Delta \text{ Anion gap} = \text{Measured anion gap} - \text{Normal anion gap} \)
   
   \( \Delta \text{ HCO}_3^- = \text{Normal [HCO}_3^-] - \text{Measured [HCO}_3^-] \)
### E. Major Causes of Metabolic Acidosis According to Mechanism and Anion Gap

<table>
<thead>
<tr>
<th>Mechanism of acidosis</th>
<th>Increased anion gap</th>
<th>Normal anion gap</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Increased acid production</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactic acidosis</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Ketoacidosis</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Starvation</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Alcohol associated</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Ingestions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methanol</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Aspirin</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Toluene (if early or if kidney function is impaired)</td>
<td>Toluene ingestion (if late and if renal function is preserved; due to excretion of sodium and potassium hippurate in the urine)</td>
<td></td>
</tr>
<tr>
<td>Diethylene glycol</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>Propylene glycol</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td>D-lactic acidosis</td>
<td>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⁻)</td>
<td></td>
</tr>
<tr>
<td>Pyroglutamic acid (5-oxoproline)</td>
<td></td>
<td>-</td>
</tr>
<tr>
<td><strong>Loss of bicarbonate or bicarbonate precursors</strong></td>
<td>-</td>
<td></td>
</tr>
<tr>
<td><strong>Decreased renal acid excretion</strong></td>
<td>Chronic kidney disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chronic kidney disease and tubular dysfunction (but relatively preserved glomerular filtration rate)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Type 1 (distal) renal tubular acidosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Type 4 renal tubular acidosis (hypoaldosteronism)</td>
</tr>
</tbody>
</table>
F. Causes of High-Anion Gap Metabolic Acidosis

Anion gap = Na - [Cl\(^-\) + HCO\(_3^\)]

A CAT MUDPILE

Aspirin
Carbon monoxide, Cyanide, Caffeine
Acetaminophen
Theophylline
Methanol, Metformin
Uremia
Diabetic ketoacidosis (Alcoholic ketoacidosis)
Propylene glycol
Isoniazid, Ibuprofen, Iron
Lactic acidosis
Ethylene glycol
G. Metabolic Alkalosis

pH: > 7.45; HCO₃⁻: > 26

<table>
<thead>
<tr>
<th>Major Causes of Metabolic Alkalosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gastrointestinal hydrogen loss</strong></td>
</tr>
<tr>
<td>Vomiting or nasogastric suction</td>
</tr>
<tr>
<td>Congenital chloride diarrhea</td>
</tr>
<tr>
<td><strong>Renal hydrogen loss</strong></td>
</tr>
<tr>
<td>Primary mineralcorticoid excess</td>
</tr>
<tr>
<td>Mineralcorticoid excess-like states</td>
</tr>
<tr>
<td>• Licorice ingestion</td>
</tr>
<tr>
<td>• Liddle syndrome</td>
</tr>
<tr>
<td>• Apparent mineralcorticoid excess</td>
</tr>
<tr>
<td>Loop or thiazide diuretics</td>
</tr>
<tr>
<td>Bartter or Gitelman syndrome</td>
</tr>
<tr>
<td>Posthypercapnic alkalosis</td>
</tr>
<tr>
<td>Hypercalcemia and the milk-alkali syndrome</td>
</tr>
<tr>
<td><strong>Intracellular shift of hydrogen</strong></td>
</tr>
<tr>
<td>Severe hypokalemia</td>
</tr>
<tr>
<td>Villous adenoma</td>
</tr>
<tr>
<td>Laxative abuse</td>
</tr>
<tr>
<td><strong>Alkali administration with reduced renal function</strong></td>
</tr>
<tr>
<td><strong>Contraction alkalosis</strong></td>
</tr>
</tbody>
</table>

Notes
II. Bladder Carcinoma

A. Bladder Cancer

Pathophysiology
• Most common type is transitional cell carcinoma

Patient
• Older, with a history of smoking or exposure to industrial dyes

Presentation
• Painless hematuria

Diagnostic Studies
• Urine cytology, cystoscopy

Notes
Most common cancer involving the urinary system

Risk Factors
• Cigarette smoke (most important)
• Industrial chemicals
• Aniline dye
• Chlorination
• Arsenic
• Chronic cystitis

Diagnosis
• Cystoscopy (gold standard)
• Urine cytology
• CT urography
III. Chronic Renal Failure (Shunts/Access)

A. Chronic Kidney Disease
(Chronic renal failure)

Pathophysiology
• Permanent loss of renal function > 3 months
• Most commonly caused by DM and hypertension

Physical Exam
• Uremic frost (urea crystals deposit on skin)
• Cardiovascular: HTN, pericarditis, CHF, atherosclerosis
• Volume overload, pulmonary edema

Diagnostic Studies
• Decreased erythropoietin with anemia of chronic disease leads to normocytic anemia
• Hypovitaminosis D leads to secondary hyperparathyroidism, contributing to renal osteodystrophy
• Labs will show platelet dysfunction, broad waxy casts, anion gap metabolic acidosis, hyperkalemia, hyperphosphatemia, hypocalcemia
• Stages:
  • Stage 1: normal kidney function (eGFR ≥ 90 ml/min) and persistent (≥ 3 months) proteinuria
  • Stage 2: mild reduction in kidney function (eGFR 60-89 ml/min) and persistent proteinuria
  • Stage 3: moderate reduction in kidney function (eGFR 30-59 ml/min)
  • Stage 4: severe reduction in kidney function (eGFR 15-29 ml/min)
  • Stage 5: kidney failure (eGFR < 15 ml/min); requires dialysis or transplant for survival [end-stage renal disease]

Management
• Depends on stage; includes diet modification (Sodium, potassium and protein restriction), transplant, dialysis
### B. Management of Chronic Kidney Disease

<table>
<thead>
<tr>
<th>Stage</th>
<th>GFR</th>
<th>Treatment options</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&gt; 90 mL/min</td>
<td>• Observation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Treat reversible causes</td>
</tr>
<tr>
<td>2</td>
<td>60 - 89 mL/min</td>
<td>• Observation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Monitor creatinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Attempt to control blood pressure, and other risk factors</td>
</tr>
<tr>
<td>3a</td>
<td>45 - 59 mL/min</td>
<td>• Observation</td>
</tr>
<tr>
<td>3b</td>
<td>30 - 44 mL/min</td>
<td>• Monitor creatinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Attempt to control blood pressure, and other risk factors</td>
</tr>
<tr>
<td>4</td>
<td>15 - 29 mL/min</td>
<td>• Plan and create access site for dialysis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Assess for possible transplant</td>
</tr>
<tr>
<td>5</td>
<td>&lt; 15 mL/min</td>
<td>• Start renal replacement therapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Dialysis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Transplant</td>
</tr>
</tbody>
</table>

---

Notes
C. Types of Dialysis

- Hemodialysis
  - Need AV fistula access
- Peritoneal dialysis
D. Continuous Ambulatory Peritoneal Dialysis

- Solution bag
- Catheter
- Peritoneal dialysis solution
- Drainage bag
- Peritoneum
### E. Arteriovenous Fistula Complications

<table>
<thead>
<tr>
<th>Complication</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dialysis Associated Steal Syndrome (DASS)</strong></td>
<td>- Occurs secondary to retrograde flow from artery distal to AV anastomosis</td>
</tr>
<tr>
<td></td>
<td>- Most common when a large artery supplies blood through fistula into a large, low pressure vein</td>
</tr>
<tr>
<td><strong>Hemorrhage</strong></td>
<td>- Often due to platelet dysfunction, supratherapeutic anticoagulation, or fistula abnormalities</td>
</tr>
<tr>
<td></td>
<td>- Hemostasis via direct pressure, topical hemostatic agents, desmopressin, suture, tourniquet</td>
</tr>
<tr>
<td><strong>Infection</strong></td>
<td><em>Staph. aureus</em> is most common</td>
</tr>
<tr>
<td><strong>Stenosis</strong></td>
<td>- Upper extremity and chest wall edema</td>
</tr>
<tr>
<td></td>
<td>- Outflow stenosis (bounding pulse, absent thrill)</td>
</tr>
<tr>
<td></td>
<td>- Inflow stenosis (weakened radial pulse and high pitched bruit)</td>
</tr>
<tr>
<td></td>
<td>- Vascular surgery consultation</td>
</tr>
<tr>
<td><strong>Thrombosis</strong></td>
<td>- Most commonly due to venous outflow stenosis, venous stasis, or compression</td>
</tr>
<tr>
<td></td>
<td>- Absence of a bruit (hear) and thrill (feel)</td>
</tr>
<tr>
<td></td>
<td>- Vascular surgery consultation</td>
</tr>
<tr>
<td></td>
<td>- Thrombectomy or thrombolysis</td>
</tr>
<tr>
<td><strong>Aneurysm or pseudoaneurysm</strong></td>
<td>- Aneurysms form secondary to repetitive cannulation and weakening of vessel walls</td>
</tr>
<tr>
<td></td>
<td>- Pseudoaneurysms are rare</td>
</tr>
<tr>
<td></td>
<td>- Vascular surgery consultation</td>
</tr>
</tbody>
</table>

---

**Notes**
IV. Dysuria

A. Overview of Urinary Casts

**Cellular Casts**

**Red blood cell cast**

- Always pathological
- Glomerular damage
- Glomerulonephritis
- Vasculitis

**White blood cell cast**

- Inflammation or infection
- Pyelonephritis
- Allergic interstitial nephritis
- Nephrotic syndrome
- Post-streptococcal glomerulonephritis

**Epithelial cell cast**

- Desquamating epithelial cells of tubule lining
- Large, round nuclei
- Acute tubular necrosis
- Toxic ingestion
- Viral infection (CMV, Hepatitis)
Overview of Urinary Casts cont.

**Acellular Casts**

**Hyaline cast**
- Most common type of cast
- Tamm-Horsfall protein
- Dehydration or exercise

**Waxy cast**
- Low urine flow
- Chronic kidney disease

**Granular cast**
- Always pathological
- Glomerular damage
- Glomerulonephritis
- Vasculitis

**Fatty cast**
- Hyaline cast with fat globule inclusion
- Yellowish-tan
- Pathognomonic for nephrotic syndrome

---

### Notes

IV. Dysuria
B. Cystitis (Urinary Tract Infection)

Pathophysiology
- Most commonly caused by *Escherichia coli*

Presentation
- Low-grade fever, increased urinary frequency, dysuria, and suprapubic or abdominal pain

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: TMP-SMX, nitrofurantoin, or fluoroquinolone for seven days

Etiology
- *E. coli*
- *Klebsiella*
- *Proteus*
- *Enterococcus*
- *Staph saprophyticus* (Female adolescents)
- Group B *Streptococcus* (Neonates)
- *Pseudomonas*
- *Adenovirus*

Diagnosis
- Urine culture > 100,000 colonies (from reliable source)

Clinical
- Suprapubic pressure and flank pain (referred)
- Vomiting
- Dysuria, urgency, frequency
- Enuresis

Notes
C. Acute Pyelonephritis

Pathophysiology
- Most commonly caused by E. coli

Presentation
- Fever, dysuria, flank pain, nausea and vomiting

Management

Oral (outpatient)
- Ciprofloxacin, levofloxacin
- Trimethoprim-sulfamethoxazole

Parenteral (mild to moderate)
- Ceftriaxone
- Ciprofloxacin, levofloxacin
- Aztreonam

Parenteral (severe)
- Cefepime
- Piperillin-tazobactam
- Meropenem

Microbiology

Ascending (most common)
- E. coli (75% to 95%)
- Proteus mirabilis
- Klebsiella pneumoniae
- Staphylococcus saprophyticus

Descending (less common)
- Septicemia
- Infective endocarditis

Physical Exam
- CVA tenderness

Management
- Fluoroquinolone or TMP-SMX
- If an inpatient or pregnant: ampicillin/gentamicin or third-gen cephalosporin

Fever
Chills
Dysuria
Frequency
Urgency
Flank pain
CVA tenderness
Nausea and vomiting

Notes
IV. Dysuria

D. 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

Gonococcal urethritis
- \( N. \textit{gonorrhoeae} \)

Nongonococcal urethritis
- \( C. \textit{trachomatis}, \textit{Mycoplasma genitalium} \)
E. 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)

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

Notes
F. 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

Comments
• Avoid vigorous prostatic massage as this can lead to septicemia
G. 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

Microbiology

- *Neisseria gonorrhoeae*
- *Chlamydia trachomatis*

Treatment

- Ceftriaxone
- Doxycycline

*Treat for 4-6 weeks*
H. Chronic Prostatitis

- Treat with fluoroquinolones or TMP-SMX for 6–12 weeks

Chronic Bacterial Prostatitis

→ Reflux of urine from urethra into intraprostatic ducts

Microbiology

- *E. coli* (most common)
- *Proteus mirabilis*
- *Enterococcus faecalis*
- *Klebsiella pneumoniae*
- *Pseudomonas aeruginosa*

Clinical

- Recurrent UTI
- Low back and perineal pain
- Urinary retention
- Tender boggy prostate

Treatment

- Trimethoprim-sulfamethoxazole
- Fluoroquinolone

Notes
V. Edema

A. Edema

**Pathophysiology**
- Swelling due to fluid overload most commonly due to loss of albumin

**Physical Exam**
- Most common locations of pitting edema: periorbital, lower extremity, and genital edema

**Diagnostic Studies**
- Urine may show renal tubular cell casts

**Management**
- Treat underlying condition

---

Notes
B. Nephrotic Syndrome

Pathophysiology
• Glomerular damage results in increased urinary protein loss

Presentation
• Proteinuria, hypoalbuminemia, edema, hyperlipidemia

Diagnostic Studies
• Transudative pleural effusion
• Urinalysis: proteinuria > 3.5 g on 24-hour urine, fatty casts, oval fat bodies
• Biopsy: hypocellular minimal change disease loss of podocytes on microscopy

Low serum protein ➔ Decreased oncotic pressure ➔ Results in edema

1. Hypoproteinemia
2. Edema
3. Proteinuria

• Most cases are minimal change nephrotic syndrome (MCNS)
• Most common 2 to 8 years old
• Decreased urine output, Abdominal pain, weight gain

Notes
Nephrotic Syndrome cont.

<table>
<thead>
<tr>
<th>Nephrotic Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary</strong></td>
</tr>
<tr>
<td><strong>Minimal change disease</strong></td>
</tr>
<tr>
<td>- More common in children</td>
</tr>
<tr>
<td>- May be preceded by URI or immunization</td>
</tr>
<tr>
<td>- Responds to steroids</td>
</tr>
<tr>
<td><strong>Focal segmental glomerulosclerosis (FSGS)</strong></td>
</tr>
<tr>
<td>- More common in African Americans</td>
</tr>
<tr>
<td>- Also seen in sickle cell disease, IV drug use, HTN, DM</td>
</tr>
<tr>
<td>- Associated with higher frequency of renal failure</td>
</tr>
<tr>
<td>- Treated with steroids, cyclosporin A, cyclophosphamide</td>
</tr>
<tr>
<td><strong>Membranous nephropathy</strong></td>
</tr>
<tr>
<td>- More common in Caucasians, aged 30-50 years</td>
</tr>
<tr>
<td>- Seen in HBV, HCV, syphilis, malaria, SLE, gold, and penicillamine use</td>
</tr>
<tr>
<td>- Approx. 24% associated with neoplasm</td>
</tr>
<tr>
<td>- Presents with proteinuria with occasional microhematuria and hypercoagulability (renal vein thrombosis)</td>
</tr>
<tr>
<td>- Spontaneous remission (25%)</td>
</tr>
<tr>
<td><strong>Membranoproliferative glomerulonephritis (MPGN)</strong></td>
</tr>
<tr>
<td>- Can present with nephrotic or nephritic features</td>
</tr>
<tr>
<td>- Assoc. with infection, autoimmune disease, HCV, and cryoglobulins</td>
</tr>
<tr>
<td>- Low C3</td>
</tr>
<tr>
<td>- Associated with 50% mortality rate or progression to ESRD within 5 years of biopsy</td>
</tr>
<tr>
<td><strong>Secondary</strong></td>
</tr>
<tr>
<td><strong>Diabetic nephropathy</strong></td>
</tr>
<tr>
<td>- Leading cause of ESRD in the United States</td>
</tr>
<tr>
<td>- Onset 5-10 years after diagnosis of type 1 DM</td>
</tr>
<tr>
<td>- Treatment includes tight glycemic control and ACE inhibitors</td>
</tr>
<tr>
<td><strong>Other</strong></td>
</tr>
<tr>
<td>- Malignancy (lymphoma or myeloma), infections (HIV, HBV, HCV, syphilis, leprosy, malaria), SLE, amyloidosis, sickle cell disease, preeclampsia</td>
</tr>
</tbody>
</table>

Notes
VI. Fluid and Electrolyte Disorders

A. Fluid Maintenance

<table>
<thead>
<tr>
<th></th>
<th>Maintenance Fluid Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>First 10 kg</td>
<td>4 mL/hr</td>
</tr>
<tr>
<td>Second 10 kg</td>
<td>2 mL/hr</td>
</tr>
<tr>
<td>Additional kgs</td>
<td>1 mL/kg/hr</td>
</tr>
</tbody>
</table>

Notes
### B. Types of Fluid Solutions

<table>
<thead>
<tr>
<th>Solution</th>
<th>0.9% Sodium Chloride</th>
<th>Lactated Ringer's</th>
<th>5% Albumin</th>
<th>Plasma Lyte</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium (mEq/L)</td>
<td>154</td>
<td>130</td>
<td>130 to 160</td>
<td>140</td>
</tr>
<tr>
<td>Potassium (mEq/L)</td>
<td>0</td>
<td>4</td>
<td>≤ 2</td>
<td>5</td>
</tr>
<tr>
<td>Chloride (mEq/L)</td>
<td>154</td>
<td>109</td>
<td>Varies</td>
<td>98</td>
</tr>
<tr>
<td>Lactate (mEq/L)</td>
<td>0</td>
<td>28</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Acetate (mEq/L)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>27</td>
</tr>
<tr>
<td>pH</td>
<td>5.5</td>
<td>6.5</td>
<td>6.9</td>
<td>7.4</td>
</tr>
<tr>
<td>Osmolarity (mOsm/L)</td>
<td>308</td>
<td>273</td>
<td>300</td>
<td>294</td>
</tr>
</tbody>
</table>

---

**Notes**
VII. Nephrolithiasis

A. Nephrolithiasis

Pathophysiology

- **Most commonly** caused by **calcium oxalate**
  - Struvite: staghorn calculi, urease-producing bacteria
  - Uric acid: radiolucent on X-ray, gout
  - Cystine: children with metabolic diseases
- **Most common** location is the ureterovesicular junction (UVJ)

Presentation

- Flank pain radiating to groin

Physical Exam

- Restless patient with CVA tenderness

Diagnostic Studies

- Helical CT is diagnostic

Management

- < 5 mm: likely to pass spontaneously
- > 8 mm: unlikely to pass; **lithotripsy**, **ESWL**
- **Emergent stenting** if stone is obstructing (associated with infection)
B. Kidney Stone

**Diagnosis**
- Plain X-ray
- Ultrasound
- CT scan

**Clinical**
- Hematuria
- Abdominal/flank pain
- Urinary frequency
- Dysuria
- Fever (sometimes)

**Management**
- Supportive
- IV hydration
- Tamsulosin
- Renal/Urology consultation

**Notes**
### C. Classification of Kidney Stones

<table>
<thead>
<tr>
<th>Type</th>
<th>Incidence</th>
<th>Crystal shape</th>
<th>X-ray findings</th>
<th>Clinical risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium oxalate</td>
<td>+++</td>
<td>Envelope</td>
<td>• Radiopaque • Spherical</td>
<td>• Men in 30s and 40s</td>
</tr>
<tr>
<td>Calcium phosphate</td>
<td>+</td>
<td>Amorphous</td>
<td>• Radiopaque • Spherical</td>
<td>• Primary hyperparathyroidism • Distal RTA • Alkali treatment</td>
</tr>
<tr>
<td>Uric acid</td>
<td>++</td>
<td>Diamond, rhomboid</td>
<td>• Radiolucent</td>
<td>• Gout • Diabetes • IBD</td>
</tr>
<tr>
<td>Struvite</td>
<td>++</td>
<td>Coffin-lid</td>
<td>• Radiopaque • Spherical • Staghorns possible</td>
<td>• Neurogenic bladder • Infection with urea-splitting bacteria</td>
</tr>
<tr>
<td>Cystine</td>
<td>+</td>
<td>Hexagonal</td>
<td>• Radiolucent • Faintly radiopaque</td>
<td>• Cystinuria associated with autosomal recessive disorder</td>
</tr>
</tbody>
</table>

---

Notes
VIII. Orthostatic Hypotension

A. Orthostatic hypotension

- After 2-5 minutes: a drop in blood pressure of ≥ 20 mm Hg systolic, ≥ 10 mm Hg diastolic, or both

- Treat underlying cause

Notes

Sitting → Standing

1. Fall in systolic blood pressure ≥ 20 mm Hg

OR

2. Fall in diastolic blood pressure ≥ 10 mm Hg
IX. Renal Cell Carcinoma

A. Renal Cell Carcinoma

Pathophysiology
- **Most common** type is **clear cell**

Patient
- History of **smoking**

Presentation
- **Flank pain, flank mass, hematuria**

Management
- Nephrectomy

*Originates within the renal cortex*

**Classic TRIAD**
1. Flank pain
2. Hematuria
3. Palpable abdominal renal mass

**Paraneoplastic symptoms**
- Anemia
- Hepatic dysfunction
- Fever
- Hypercalcemia
- Cachexia
- Amyloidosis
- Polymyalgia rheumatica
X. Renal Vascular Disease

A. Renal Artery Stenosis

Pathophysiology
- **Narrowing** of one or both of the renal arteries
- Most often caused by atherosclerosis or fibromuscular dysplasia

Physical Exam
- May hear a renal artery **bruit** on auscultation

Diagnosis
- Renal **arteriography** is **gold standard** for diagnosis

Management
- Percutaneous transluminal angioplasty (**PTA**) plus stent placement or with surgical bypass of the stenotic segment

Clinical
- Renal artery stenosis: caused by atherosclerosis or fibromuscular dysplasia
- Most cases are asymptomatic
- Recalcitrant hypertension
- Fibromuscular dysplasia – most common in young females

Management Options
- Control of hypertension with ACE inhibitor or ARB
- Percutaneous angioplasty with or without stent placement
- Surgical revascularization or resection

Notes
# XI. Testicular Carcinoma

## A. Testicular Cancer

### Patient
- Man, **15 – 35 years old**

### Presentation
- Testicular lump

### Physical Exam
- **Painless**, hard, **fixed** mass

### Diagnostic Studies
- Increased **beta-hCG, alpha-fetoprotein (AFP), or lactate dehydrogenase (LDH)**
- Imaging starts with **ultrasound**

### Comments
- **Most common** risk factor: **cryptorchidism**
- **Most common** tumor: **seminoma**

### Notes

**Most common solid malignancy affecting males between ages 15 and 35**

---

### Clinical
- Nodule or **painless** swelling of one testicle
  - **Metastatic disease**
    - Neck mass (supraclavicular lymph node metastasis)
    - Cough or dyspnea (pulmonary metastasis)
    - Anorexia, nausea, vomiting, GI bleeding (retroduodenal metastasis)
    - Lumbar back pain (retroperitoneal disease)
    - Bone pain (skeletal metastasis)
    - CNS or PNS symptoms (cord or nerve involvement)
    - Extremity swelling (iliac or caval venous obstruction)

### Diagnostic
- Scrotal ultrasound
- CT scan

### Serum Markers
- Seminoma (Beta-hCG)
- Non-Seminoma (Beta-hCG, Alpha-fetoprotein)
B. Testicular Cancer Tumor Markers

**Germ cell**

- **Seminoma**: β-hCG

**Non-Seminoma**

- **Choriocarcinoma**: β-hCG
- **Yolk sac**: α-fetoprotein
- **Embryonal**: β-hCG, α-fetoprotein
- **Teratoma**: No marker
- **Teratocarcinoma**: β-hCG, α-fetoprotein

Notes
# XII. Urinary Retention

## A. Selected Causes of Urinary Retention

<table>
<thead>
<tr>
<th>Cause</th>
<th>Women</th>
<th>Men</th>
<th>Both</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Obstructive</strong></td>
<td>• Cystocele</td>
<td>• BPH</td>
<td>• Aneurysmal dilation</td>
</tr>
<tr>
<td></td>
<td>• Rectocele</td>
<td>• Meatal stenosis</td>
<td>• Bladder calculi</td>
</tr>
<tr>
<td></td>
<td>• Uterine prolapse</td>
<td>• Paraphimosis</td>
<td>• Bladder neoplasm</td>
</tr>
<tr>
<td></td>
<td>• Pelvic mass</td>
<td>• Penile constricting bands</td>
<td>• Fecal impaction</td>
</tr>
<tr>
<td></td>
<td>• Uterine fibroid</td>
<td>• Phimosis</td>
<td>• GI malignancy</td>
</tr>
<tr>
<td></td>
<td>• Ovarian cyst</td>
<td>• Prostate cancer</td>
<td>• Urethral strictures</td>
</tr>
<tr>
<td></td>
<td>• Retroverted impacted uterus</td>
<td></td>
<td>• Edema</td>
</tr>
<tr>
<td><strong>Infectious and inflammatory</strong></td>
<td>• Acute vulvovaginitis</td>
<td>• Balanitis</td>
<td>• Bilharziasis</td>
</tr>
<tr>
<td></td>
<td>• Vaginal lichen planus</td>
<td>• Prostatic abscess</td>
<td>• Cystitis</td>
</tr>
<tr>
<td></td>
<td>• Vaginal lichen sclerosis</td>
<td>• Prostatitis</td>
<td>• Echinococcosis</td>
</tr>
<tr>
<td></td>
<td>• Vaginal pemphigus</td>
<td></td>
<td>• Guillain-Barré syndrome</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td>• Postpartum complication</td>
<td>• Penile trauma</td>
<td>• HSV</td>
</tr>
<tr>
<td></td>
<td>• Urethral sphincter dysfunction</td>
<td></td>
<td>• Lyme disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Periurethral abscess</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Transverse myelitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Tubercular cystitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Varicella-zoster virus</td>
</tr>
</tbody>
</table>

**Notes**
XIII. Wilms Tumor

A. Wilms Tumor (Nephroblastoma)

Patient
- Younger than 15 years old

Presentation
- Abdominal pain, anorexia, abdominal distention, vomiting, or hematuria

Physical Exam
- Abdominal mass

Physical Exam
- CT scan is diagnostic

Management
- Surgical resection, chemotherapy, and radiation therapy

Comments
- Most common solid renal tumor of childhood

Clinical
- Most patients present with asymptomatic abdominal mass or swelling
- Other symptoms can include abdominal pain, hematuria, fever, hypertension
- Physical exam: Firm, nontender mass eccentrically located, rarely crosses midline

Diagnosis
- Ultrasound (Initial imaging study)
- CT scan, MRI for further evaluation
- Surgical excision or biopsy (Definitive)

Most common renal malignancy in children < 15 years old

Notes
CHAPTER 8

Hematology
CHAPTER 8
Hematology

282  ANEMIA

297  EASY BRUISING/BLEEDING

306  FATIGUE
I. Anemia

A. Causes of Anemia

<table>
<thead>
<tr>
<th>Low mean corpuscular volume (microcytic anemia: MCV &lt; 80 fL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Iron-deficiency anemia</td>
</tr>
<tr>
<td>- Thalassemic disorders</td>
</tr>
<tr>
<td>- Anemia of inflammation/anemia of chronic disease (late; uncommon)</td>
</tr>
<tr>
<td>- Sideroblastic anemia (e.g., congenital, lead, alcohol, drugs; uncommon)</td>
</tr>
<tr>
<td>- Copper deficiency, zinc poisoning (rare)</td>
</tr>
<tr>
<td>- Hemolysis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Normal mean corpuscular volume (normocytic anemia: MCV 80–100 fL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Acute blood loss</td>
</tr>
<tr>
<td>- Iron-deficiency anemia (early)</td>
</tr>
<tr>
<td>- Anemia of inflammation/anemia of chronic disease (e.g., infection, inflammation, malignancy)</td>
</tr>
<tr>
<td>- Bone marrow suppression (may also be macrocytic)</td>
</tr>
<tr>
<td>- Chronic renal insufficiency</td>
</tr>
<tr>
<td>- Hypothyroidism</td>
</tr>
<tr>
<td>- Hypopituitarism</td>
</tr>
<tr>
<td>- Hemolysis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Increased mean corpuscular volume (macrocytic anemia: MCV &gt; 100 fL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Excessive ethanol use</td>
</tr>
<tr>
<td>- Folate deficiency</td>
</tr>
<tr>
<td>- Vitamin B12 deficiency</td>
</tr>
<tr>
<td>- Myelodysplastic syndromes</td>
</tr>
<tr>
<td>- Acute myeloid leukemias</td>
</tr>
<tr>
<td>- Reticulocytosis</td>
</tr>
<tr>
<td>- Drug-induced anemia (e.g., hydroxyurea, AZT, chemotherapeutic agents)</td>
</tr>
<tr>
<td>- Liver disease</td>
</tr>
<tr>
<td>- Hypothyroidism</td>
</tr>
</tbody>
</table>
B. Iron Deficiency Anemia

Presentation
• Weakness, dizziness, and fatigue

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

Clinical

Symptoms
• Fatigue
• Weakness
• Headache
• Irritability
• Exercise intolerance
• Exertional dyspnea
• Pica and ice craving

Signs
• Pallor
• Dry or rough skin
• Blue sclerae
• Atrophic glossitis
• Cheilosis
• Koilonychia
• Esophageal web

Laboratory

<table>
<thead>
<tr>
<th>RBC count</th>
<th>Hgb</th>
<th>ARC</th>
<th>MCV</th>
<th>MCH</th>
<th>Iron</th>
<th>Ferritin</th>
<th>TS</th>
<th>Transferrin</th>
<th>RDW</th>
<th>TIBC</th>
</tr>
</thead>
</table>

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

Notes
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)

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

Ringed sideroblast
Granules of iron accumulate in the mitochondria surrounding the nucleus
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)

Exposure results from ingestion of lead-containing material (e.g., paint chips)
• Subtle, insidious, nonspecific sx
• Headache
• Peripheral motor neuropathy
• HTN, anemia, gout
• Cognitive impairment

Succimer is treatment
"It SUCCS to eat LEAD"

Notes
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
I. Anemia

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, malabsorption

**I. Hematopoiesis**

1. **Myeloid**
   - Granulocytes
   - Monocytes
   - Erythroblasts
   - Megakaryocytes

2. **Lymphoid**
   - Lymphocytes
   - Plasma cells

**Notes**
### G. Anemia of Chronic Disease

**Presentation**
- Fatigue

**Diagnostic Studies**
- **Normocytic anemia**, **low** serum iron, **high** or normal **ferritin**

<table>
<thead>
<tr>
<th></th>
<th>Iron Deficiency Anemia</th>
<th>Anemia of Chronic Disease</th>
<th>Thalassemia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MCV</strong></td>
<td>Low/normal</td>
<td>Low/normal</td>
<td>Low/normal</td>
</tr>
<tr>
<td><strong>RDW</strong></td>
<td>High</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Iron</strong></td>
<td>Low</td>
<td>Low</td>
<td>Normal/high</td>
</tr>
<tr>
<td><strong>TIBC</strong></td>
<td>High</td>
<td>Normal/low</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Ferritin</strong></td>
<td>Low</td>
<td>Normal/high</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Transferrin saturation</strong></td>
<td>Low</td>
<td>Low/Normal</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Transferrin</strong></td>
<td>High</td>
<td>Normal</td>
<td>Normal</td>
</tr>
</tbody>
</table>

_MCV_ = mean corpuscular volume  
_RDW_ = red cell distribution width  
_TIBC_ = total iron binding capacity
H. 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

Etiology
• Autoimmune mechanisms
• Direct injury to hematopoietic stem cells
• Viral infection
• Clonal and genetic disorders
• Drug reaction

Clinical
• Recurrent infections (neutropenia)
• Mucosal hemorrhage (thrombocytopenia)
• Fatigue (anemia)

Aplastic Anemia
Pancytopenia + bone marrow hypoplasia/aplasia

Loss of hematopoietic stem cells

Bone marrow
Platelets
Red blood cells
White blood cells

Normal
Aplastic anemia

Notes
I. 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

**Alpha Thalassemia** → Occurs when one or more of the **four alpha globin chain** genes fails function
J. Beta Thalassemia Minor

**Diagnostic Studies**
- Microcytic and hypochromic cells, *increased* Hgb A2, *target cells*

**Management**
- Genetic counseling, avoid iron

K. 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

---

Notes
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

Notes
I. Anemia

L. Hemolytic Anemia

Premature breakdown of RBCs

- Autoimmune Hemolytic Anemia
  - Positive direct Coombs test
  - Increased reticulocyte count, increased LDH, decreased haptoglobin, and increased bilirubin (indirect)

- Hereditary Spherocytosis
  - Positive osmotic fragility test
  - Increased reticulocyte count
  - Presence of spherocytes

- G6PD deficiency
  - Oxidative stress
  - Increased reticulocyte count
  - X-linked
  - Heinz bodies and Bite cells on smear
  - Triggers: Fava beans, antimalarials, sulfonamides
Hereditary Spherocytosis
Autosomal dominant

Dense and hyperchromic hereditary hemolytic anemia

Notes
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

G6PD Deficiency \rightarrow \text{No NADPH} = \text{No Glutathione} (free radical destroyer)

 Oxidative stress \rightarrow \text{Hemolytic anemia}

• Causes a nonimmune hemolytic anemia (infection, medications, fava beans)
• Protects against malaria
• Heinz bodies on smear

Notes
Hemolytic Anemia Review

<table>
<thead>
<tr>
<th>Hemolytic Anemia</th>
<th>Extravascular</th>
<th>Intravascular</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Etiologies</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Warm autoimmune hemolytic anemia</td>
<td></td>
<td>• Cold autoimmune hemolytic anemia</td>
</tr>
<tr>
<td>• Hypersplenism</td>
<td></td>
<td>• Acute hemolytic transfusion reaction</td>
</tr>
<tr>
<td>• Delayed hemolytic transfusion reaction</td>
<td></td>
<td>• Microangiopathic hemolysis</td>
</tr>
<tr>
<td>• Hemoglobinopathies</td>
<td></td>
<td>• G6PD deficiency</td>
</tr>
<tr>
<td>• Paroxysmal nocturnal hemoglobinuria</td>
<td></td>
<td>• Hemoglobinopathies</td>
</tr>
<tr>
<td>Site of RBC destruction</td>
<td>Macrophage (spleen, liver)</td>
<td>Blood vessels</td>
</tr>
<tr>
<td>Haptoglobin</td>
<td>Low</td>
<td>Very Low</td>
</tr>
<tr>
<td>LDH</td>
<td>High</td>
<td>Very high</td>
</tr>
<tr>
<td>Urine hemosiderin</td>
<td>Negative</td>
<td>Positive</td>
</tr>
<tr>
<td>Urine hemoglobin</td>
<td>Negative</td>
<td>Positive (in severe cases)</td>
</tr>
<tr>
<td>Peripheral smear</td>
<td>Spherocytes</td>
<td>Schistocytes</td>
</tr>
</tbody>
</table>

Notes
II. Easy Bruising/Bleeding

A. Preoperative testing:

- PT, aPTT, platelet count

Notes
### B. Clinical Features of Clotting Disorders

<table>
<thead>
<tr>
<th>Bleeding characteristics</th>
<th>Type of bleeding disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Thrombocytopenia or platelet function defects</td>
</tr>
<tr>
<td>Major sites of bleeding</td>
<td>Mucocutaneous (e.g., mouth, nose, gastrointestinal tract, urinary tract, menorrhagia)</td>
</tr>
<tr>
<td>Petechiae</td>
<td>Common</td>
</tr>
<tr>
<td>Ecchymoses</td>
<td>Generally small and superficial; may be significant, depending upon the degree of thrombocytopenia</td>
</tr>
<tr>
<td>Excessive bleeding after minor cuts</td>
<td>Yes</td>
</tr>
<tr>
<td>Excessive bleeding with surgery or invasive procedures</td>
<td>Often immediate Degree varies with severity of the defect (e.g., no excess bleeding with mild thrombocytopenia, severe bleeding with certain platelet function defects such as GT)</td>
</tr>
</tbody>
</table>

---

**Notes**
C. 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

Notes
D. 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

Notes
• Hemophilia B
  • X-linked recessive
  • Decreased synthesis of Factor IX (9)
  • Treat with recombinant Factor IX
  • Christmas disease
E. Von Willebrand Disease

**Most common inherited bleeding disorder**

**Pathophysiology**
- **Autosomal dominant**

**Patient**
- History of a *parent* with similar symptoms

**Presentation**
- Mucosal hemorrhage or bleeding that is difficult to control

**Diagnostic Studies**
- Decreased factor VIII, prolonged bleeding time

**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)

**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)

**Treatment**
- Desmopressin (DDAVP)
F. 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
### G. Pretest Probability Scoring for Heparin-Induced Thrombocytopenia (The 4T Score)

<table>
<thead>
<tr>
<th>Category</th>
<th>2 Points</th>
<th>1 Point</th>
<th>0 Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombocytopenia</td>
<td>Platelet count fall &gt; 50% AND Platelet nadir ≥ 20,000</td>
<td>Platelet count fall 30%–50% OR Platelet nadir 10–19,000</td>
<td>Platelet count fall &lt; 30% OR Platelet nadir &lt; 10,000</td>
</tr>
<tr>
<td>Timing of platelet count fall</td>
<td>Clear onset between days 5 and 10 OR Platelet fall ≤ 1 day (prior heparin exposure w/in 30 days)</td>
<td>Consistent with days 5–10 fall, but not clear or onset after day 10 OR Fall ≤ 1 day (prior heparin exposure 30–100 days ago)</td>
<td>Platelet count fall &lt; 4 days without recent heparin exposure</td>
</tr>
<tr>
<td>Thrombosis or other sequelae</td>
<td>New thrombosis or skin necrosis at heparin injection sites or acute systemic reaction after intravenous heparin bolus</td>
<td>Progressive or recurrent thrombosis or nonnecrotizing (erythematous) skin lesions or suspected thrombosis</td>
<td>None</td>
</tr>
<tr>
<td>Other causes for thrombocytopenia</td>
<td>None apparent</td>
<td>Possible</td>
<td>Definite</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Total Points</th>
<th>Probability of Heparin-Induced Thrombocytopenia</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 3</td>
<td>Low probability</td>
</tr>
<tr>
<td>4–5</td>
<td>Intermediate probability</td>
</tr>
<tr>
<td>≥ 6</td>
<td>High probability</td>
</tr>
</tbody>
</table>

H. 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

Disseminated coagulation
  - Microvascular thrombosis
  - Microangiopathic hemolytic anemia

Ongoing fibrinolysis
  - High
    - PT
    - aPTT
    - Thrombin clotting time

  - Low
    - Platelets
    - Fibrinogen

  - High
    - D-dimers
    - Fibrin complexes
    - Thrombin clotting time
Coagulation Disorders Review

<table>
<thead>
<tr>
<th>Disorders of Coagulation</th>
</tr>
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<tbody>
<tr>
<td><strong>Disorder</strong></td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td>Hemophilia A</td>
</tr>
<tr>
<td>Hemophilia B</td>
</tr>
<tr>
<td>von Willebrand disease</td>
</tr>
<tr>
<td>DIC</td>
</tr>
<tr>
<td>Polycythemia vera</td>
</tr>
<tr>
<td>Protein C and S deficiency</td>
</tr>
<tr>
<td>Factor V Leiden</td>
</tr>
</tbody>
</table>

Notes
CHAPTER 9
Pulmonology
<table>
<thead>
<tr>
<th>Page</th>
<th>Condition</th>
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</thead>
<tbody>
<tr>
<td>309</td>
<td>HEMOPTYSIS</td>
</tr>
<tr>
<td>311</td>
<td>LUNG CARCINOMA</td>
</tr>
<tr>
<td>313</td>
<td>PLEURAL EFFUSION</td>
</tr>
<tr>
<td>315</td>
<td>PNEUMONIA (POSTOPERATIVE)</td>
</tr>
<tr>
<td>318</td>
<td>PNEUMOTHORAX</td>
</tr>
<tr>
<td>321</td>
<td>SHORTNESS OF BREATH (DYSPNEA)</td>
</tr>
<tr>
<td>322</td>
<td>WEIGHT LOSS AND FATIGUE</td>
</tr>
</tbody>
</table>
I. 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

Notes
B. Causes of Hemoptysis

BATTLE CAMP

Bronchitis
Bronchiectasis
Aspergilloma
Tumor
Tuberculosis
Lung abscess
Embol (pulmonary)
Coagulopathy
Autoimmune disorders
AV malformation
Alveolar hemorrhage
Mitral stenosis
Pneumonia
II. Lung Carcinoma

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
II. Lung Carcinoma

Lung Cancer

Small cell lung cancer (SCLC)
  - Starts centrally

Non-small cell lung cancer (NSCLC)

  Adenocarcinoma
    - Most common
    - Peripherally located

  Squamous cell carcinoma
    - Starts centrally
    - Hypercalcemia

  Large cell carcinoma

Notes
III. Pleural Effusion

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** is used to differentiate between transudative and exudative effusions

Notes

Loss of cardiophrenic angle

Loss of diaphragmatic and apex of heart silhouette

Blunting of costophrenic sulci
### III. Pleural Effusion

<table>
<thead>
<tr>
<th>Light’s Criteria</th>
<th>Transudate</th>
<th>Exudate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pleural:Serum Protein</td>
<td>&lt; 0.5</td>
<td>≥ 0.5</td>
</tr>
<tr>
<td>Pleural:Serum LDH</td>
<td>&lt; 0.6</td>
<td>≥ 0.6</td>
</tr>
<tr>
<td>Pleural fluid LDH</td>
<td>&lt; 2/3 upper limit of normal</td>
<td>&gt; 2/3 upper limit of normal</td>
</tr>
</tbody>
</table>

**Main Causes**
- Heart failure
- Cirrhosis
- Nephrotic syndrome
- Pulmonary embolism
- Malignancy
- Bacterial/Viral pneumonia
- Tuberculosis
- Pulmonary embolism
- Pancreatitis
- Esophageal rupture
- Collagen vascular disease
- Chylothorax/Hemothorax

**Notes**
IV. Pneumonia (Postoperative)

A. Postoperative pneumonia

- Related to hospital- or ventilator- acquired pneumonia

- Most common pathogens: *Pseudomonas aeruginosa*, Methicillin-sensitive *Staphylococcus aureus* (MSSA), Methicillin-resistant *S. aureus* (MRSA)
## B. Reducing Ventilator-Associated Pneumonia

### Airway Management
- Minimize duration of ventilation; perform daily assessments of readiness to wean from ventilation
- Interrupt sedation daily
- Use non-invasive positive-pressure ventilation whenever possible
- Use a cuffed continuous aspiration of subglottic secretions (CASS) tube
- Use orotracheal tubes over nasotracheal tubes, unless contraindicated

### Oral Care
- Comprehensive oropharyngeal cleaning and decontamination with antiseptic solution

### Gastric Reflux Prevention
- Elevate head of bed 30 to 45 degrees to prevent aspiration

### Equipment Maintenance
- Change ventilator circuit when it becomes visibly soiled or malfunctions

---

**Notes**
### C. Medical Treatment of Pneumonia

#### Community-acquired Pneumonia (CAP)

- **Infection acquired in the community**
  - **Outpt:** Macrolide or doxycycline
  - **Chronic dz, immunocomp, or ABX in past 3 months:** Respiratory fluoroquinolone or beta-lactam + macrolide or doxycycline
  - **Inpt:** Resp fluoroquinolone or beta-lactam + macrolide or doxycycline

#### Hospital-acquired Pneumonia (HAP)

- **Occurs ≥ 48 hours after admission and did not appear to be incubating at the time of admission**
  - **(-) MDR risk factors:** Monotherapy with beta-lactam, ertapenem, or resp fluoroquinolone
  - **(+ MDR risk factors:** Antipseudomonal beta-lactam or carbapenem + resp fluoroquinolone
  - **(+) MRSA risk factors:** Add vancomycin, consider consolidation to two agents: vancomycin + antipseudomonal beta-lactam

#### Ventilator-associated Pneumonia (VAP)

- **Develops more than 48 to 72 hrs after endotracheal intubation**
  - **Initial ABX Therapy:** Same as HAP for MDR RF

#### Aspiration Pneumonia

- **Increased risk for anaerobes**
  - **Initial ABX Therapy**
    - No consensus guideline
    - Rec: Antipseudomonal beta-lactam, carbapenem, or cephalosporin
    - Resp fluoroquinolone or antipseudomonal beta-lactam + clindamycin or metronidazole

---

**Notes**
V. Pneumothorax

A. 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., thoracentesis) or therapeutic (e.g., central venous line) procedure

Traumatic
Result of blunt or penetrating trauma

Lung collapse
B. Spontaneous Pneumothorax

Patient
- Young, tall, thin man

Physical Exam
- 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
C. Tension Pneumothorax

Patient
• History of chest trauma

Physical Exam
• 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

Notes
### VI. Shortness of Breath (Dyspnea)

#### A. Physical Examination Signs of Dyspnea

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

A. Conditions and Common Clinical Findings

**Anemia:** SOB, pallor, brittle nails, tachycardia, lightheadedness, fatigue

**Anorexia:** weight loss, irregular menstruation, underweight

**Chronic Fatigue Syndrome:** fatigue, anxiety, muscle pain

**Chronic Obstructive Pulmonary Disease:** fatigue, shortness of breath, dry cough

**HIV:** fatigue, fever, weight loss, night sweats

**Hyperthyroidism:** weight loss, fatigue, excessive sweating

**Interstitial Lung Disease:** SOB, tachypnea, weight loss, chronic cough, fatigue

**Pulmonary Hypertension:** fatigue, inability to exercise, edema

**Sleep Apnea:** fatigue, episodes of apnea, weight gain, snoring

**Tuberculosis:** SOB, hemoptysis, fatigue, fever, weight loss, night sweats

**Type 1 Diabetes:** weight loss, fatigue, hunger

**Type 2 Diabetes:** weight loss, fatigue, excessive thirst, excessive hunger
CHAPTER 10

Obstetrics/Gynecology
CHAPTER 10
Obstetrics/Gynecology

325  ADENOPATHY

327  BENIGN BREAST DISEASE
Fibroadenomas
Fibrocystic Breast Disease

329  BREAST CARCINOMA

332  NIPPLE DISCHARGE

334  SKIN CHANGES
I. Adenopathy

A. Axillary Adenopathy

*Most common* adenopathy in breast disease is **Axillary Adenopathy**
B. Sentinel Node Biopsy

Tumor with injected labeling substance

Sentinel lymph node with absorbed labeling substance

Notes
II. Benign Breast Disease

FIBROADENOMAS • FIBROCYSTIC BREAST DISEASE

A. Fibroadenoma

Patient
• Woman of childbearing age

Presentation
• Painless, firm, solitary, mobile, slowly growing breast mass

Management
• Watchful waiting or surgical excision

Comments
• Most common breast tumor in adolescent women

Painless, firm, solitary, mobile, slowly growing breast mass
• Women of childbearing age
• Most common breast tumor in adolescent women
• Partially hormone-dependent, regresses after menopause
• Rule out malignancy
• Conservative management or surgical excision
B. Fibrocystic Breast Disease

Pathophysiology
• Caused by fluctuating estrogen levels during menstrual cycles

Patient
• 30 – 50 years old

Presentation
• Intermittent breast pain and tenderness that peaks before menstruation

Diagnostic Studies
• Ultrasound will show dense, prominent, fibroglandular tissue with cysts but no discernable mass

Management
• Well-fitted and supportive bras, heat pack to the breasts, or over-the-counter pain relievers

Comments
• Most common lesion of the breast
• Fibrocystic changes are generally benign and do not increase risk for breast cancer

• Noncancerous breast lumps
• Intermittent breast pain and tenderness
• Symptoms peak before each menstruation
• Symptoms improve after menopause
• Mammography, ultrasound, aspiration
III. Breast Carcinoma

A. Breast Cancer

Pathophysiology
• Most common type is invasive ductal carcinoma

Patient
• History of prior ovarian or breast cancer, a family history of breast or ovarian cancer, or a genetic predisposition such as BRCA1/BRCA2

Diagnostic Studies
• Diagnosis is made by mammography and core biopsy

Management
• Breast-conserving therapy (lumpectomy followed by radiation therapy) or mastectomy (with or without radiation therapy)
  • Surgery should involve a sentinel lymph node biopsy
  • Adjuvant therapy depending on tumor and patient characteristics

Comments
• Second most frequent cause of cancer death among women in the United States
• Screening (USPSTF):
  • Average risk: biennial screening mammography starting at the age of 50
  • Breast self-examination should not be routinely encouraged
B. Concerning Findings on Breast Exam

- **Lump**: hard, immovable, single dominant lesion with irregular borders (upper outer quadrant)
- Retracted nipple
- Skin dimpling
- Nipple discharge
- Erythema
- Skin changes
## C. Types of Invasive Breast Cancer

<table>
<thead>
<tr>
<th>Types of Invasive Breast Cancer</th>
<th>Proportion of all invasive breast cancers</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Invasive ductal carcinoma (IDC)</td>
<td>70–80%</td>
<td>Prognosis varies based on stage and grade of tumor</td>
</tr>
<tr>
<td>Invasive lobular carcinoma (ILC)</td>
<td>10–15%</td>
<td>Prognosis varies based on stage and grade of tumor</td>
</tr>
<tr>
<td>Medullary carcinoma</td>
<td>1–5%</td>
<td>More common among younger women and women with <em>BRCA1</em></td>
</tr>
<tr>
<td>Mucinous (colloid) carcinoma</td>
<td>1–5%</td>
<td>More common among older women Tends to have a good prognosis</td>
</tr>
<tr>
<td>Papillary carcinoma</td>
<td>&lt; 1%</td>
<td>More common among postmenopausal women Tends to have a good prognosis</td>
</tr>
<tr>
<td>Tubular carcinoma</td>
<td>1–5%</td>
<td>Rare for cancer to spread to lymph nodes or other parts of body</td>
</tr>
<tr>
<td>Inflammatory carcinoma</td>
<td>1–5%</td>
<td>Aggressive, often presents with advanced disease, overlying skin is thickened and red (peau d’orange)</td>
</tr>
</tbody>
</table>

**Notes**
IV. Nipple Discharge

A. Nipple Discharge

<table>
<thead>
<tr>
<th>Discharge type</th>
<th>Characteristics</th>
<th>Common Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physiologic</strong> (galactorrhea):</td>
<td>- Usually bilateral</td>
<td>- Hyperprolactinemia</td>
</tr>
<tr>
<td>Nonpathologic nipple discharge</td>
<td>- White or clear</td>
<td>- Medications</td>
</tr>
<tr>
<td>unrelated to pregnancy or</td>
<td>- May be straw-colored, green, brown,</td>
<td>- Neurogenic stimulation</td>
</tr>
<tr>
<td>breastfeeding</td>
<td>gray</td>
<td></td>
</tr>
<tr>
<td><strong>Pathologic</strong> (suspicious):</td>
<td>- Usually unilateral</td>
<td>- Papilloma</td>
</tr>
<tr>
<td>Secretory production of fluids</td>
<td>- Localized to single duct</td>
<td></td>
</tr>
<tr>
<td>other than milk may be due to</td>
<td>- Persistent</td>
<td></td>
</tr>
<tr>
<td>a pathological process in the</td>
<td>- Spontaneous</td>
<td></td>
</tr>
<tr>
<td>breast</td>
<td>- Serous, clear, yellow</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Sanguineous</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Serosanguine</td>
<td></td>
</tr>
</tbody>
</table>

**Intraductal papilloma** is a common cause of **bloody** nipple discharge

Notes
B. Management of Spontaneous Nipple Discharge (Non-Lactating)

History and physical exam: No palpable mass

- Abnormal imaging
  - Refer to surgeon

- Normal imaging*
  - Single duct
    - Refer to surgeon
  - Multiple ducts
    - Bloody
      - Refer to surgeon
    - Non-bloody
      - Medical evaluation
        - Consider galactorrhea work up
        - Continue routine screening

*Breast ultrasound recommended for all patients with nipple discharge. Mammograms are recommended for women ≥ age 30.
V. Skin Changes

A. Melasma

Pathophysiology
- Most commonly caused by hormonal changes

Patient
- Woman who is pregnant or using oral contraceptives

Presentation
- Complaining of discoloration on parts of the face

Physical Exam
- Dark, irregular, well demarcated hyperpigmented macules and patches

Management
- Treatment is sunscreen and sun avoidance

Management
- Photoprotection (avoidance, sunscreen)
- Skin-lightening agents (e.g., hydroquinone, azelaic acid)
- Topical retinoids
- Chemical peels

Risk Factors
- Female sex
- Reproductive age
- Darker skin tones
- Pregnancy
- Oral contraceptives
- UV radiation
- Genetic predisposition

Comments
- During pregnancy, it is called chloasma

Hyperpigmented brown flat macular patch

Notes
B. Paget Disease of the Breast
   → Rare cancer of the breast

Pathophysiology
   • Type of rare, malignant breast cancer

Presentation
   • Nipple and areola changes

Physical Exam
   • May see bloody discharge or nipple retraction

Diagnostic Studies
   • Diagnosis is made clinically
   • Breast imaging or biopsy can be helpful

Management
   • Breast conserving surgery with radiation
   • Mastectomy

Hallmark
   A scaly, raw, vesicular, or ulcerated lesion that begins on the nipple and then spreads to the areola

Clinical
   • Pain, burning, or pruritus may present before clinically apparent

Diagnosis
   • Skin biopsy and histology
   • Mammography
   • Ultrasound
   • MRI

Management
   • Simple mastectomy (historic standard)
   • Breast-conserving treatment (often selected)

Paget cell
   Atypical cells with large nuclei and vacuolated cytoplasm

Notes
### Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>BB</td>
<td>Beta Blocker</td>
</tr>
<tr>
<td>CCB</td>
<td>Calcium Channel Blocker</td>
</tr>
<tr>
<td>HTN</td>
<td>Hypertension</td>
</tr>
<tr>
<td>BP</td>
<td>Blood Pressure</td>
</tr>
<tr>
<td>HR</td>
<td>Heart Rate</td>
</tr>
<tr>
<td>ACE</td>
<td>Angiotensin-Converting Enzyme</td>
</tr>
<tr>
<td>ACEI</td>
<td>Angiotensin-Converting Enzyme Inhibitor</td>
</tr>
<tr>
<td>ARB</td>
<td>Angiotensin II Receptor Blocker</td>
</tr>
<tr>
<td>CKD</td>
<td>Chronic Kidney Disease</td>
</tr>
<tr>
<td>MI</td>
<td>Myocardial Infarction</td>
</tr>
<tr>
<td>HDL</td>
<td>High-Density Lipoprotein</td>
</tr>
<tr>
<td>LDL</td>
<td>Low-Density Lipoprotein</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>JVD</td>
<td>Jugular Venous Distension</td>
</tr>
<tr>
<td>HTN</td>
<td>Hypertension</td>
</tr>
<tr>
<td>CHF</td>
<td>Congestive Heart Failure</td>
</tr>
<tr>
<td>EF</td>
<td>Ejection Fraction</td>
</tr>
<tr>
<td>LLSB</td>
<td>Lower Left Sternal Border</td>
</tr>
<tr>
<td>RUQ</td>
<td>Right Upper Quadrant</td>
</tr>
<tr>
<td>CT</td>
<td>Computerized Tomography</td>
</tr>
<tr>
<td>AAA</td>
<td>Abdominal Aortic Aneurysm</td>
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<tr>
<td>CTA</td>
<td>Computerized Tomography Angiography</td>
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<tr>
<td>MRA</td>
<td>Magnetic Resonance Angiography</td>
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<td>AVM</td>
<td>Arteriovenous Malformation</td>
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<td>ESR</td>
<td>Erythrocyte Sedimentation Rate</td>
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<tr>
<td>ASAP</td>
<td>As Soon As Possible</td>
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<td>CRP</td>
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<td>DVT</td>
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<td>NSAID</td>
<td>Non-Steroidal Antiinflammatory</td>
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<td>HPV</td>
<td>Human Papillomavirus</td>
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<td>STI</td>
<td>Sexually Transmitted Infection</td>
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<td>HSV</td>
<td>Herpes Simplex Virus</td>
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<td>PCR</td>
<td>Polymerase Chain Reaction</td>
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<td>AIDS</td>
<td>Acquired Immunodeficiency Syndrome</td>
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<td>Human Herpesvirus</td>
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<td>KOH</td>
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<td>CN</td>
<td>Cranial Nerve</td>
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<td>HCTZ</td>
<td>Hydrochlorothiazide</td>
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<td>Immunoglobulin</td>
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<td>IM</td>
<td>Intramuscular</td>
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<td>I&amp;D</td>
<td>Incision And Drainage</td>
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<td>PCN</td>
<td>Penicillin</td>
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<tr>
<td>HIDA</td>
<td>Hepatic Iminodiacetic Acid</td>
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<td>AMS</td>
<td>Altered Mental Status</td>
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<td>Antibodies Against The Yeast Saccharomyces Cerevisiae</td>
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<td>VCUG</td>
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<td>p-ANCA</td>
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<td>NGT</td>
<td>CSF</td>
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Abbreviation:
- 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 Cystourethrograph
- UA: Urinalysis
- TMP-SMX: Trimethoprim-Sulfamethoxazole
- CVA: Costovertebral Angle
- 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-Phosphatase
- 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
### Abbreviations

<table>
<thead>
<tr>
<th>AFB</th>
<th>Acid-Fast Bacilli</th>
<th>EEG</th>
<th>Electroencephalogram</th>
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<tr>
<td>PPD</td>
<td>Purified Protein Derivative</td>
<td>TLC</td>
<td>Total Lung Capacity</td>
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<tr>
<td>VDRL</td>
<td>Venereal Disease Research Laboratory</td>
<td>FEV</td>
<td>Forced Expiratory Volume</td>
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<tr>
<td>RPR</td>
<td>Rapid Plasma Reagin</td>
<td>FVC</td>
<td>Forced Vital Capacity</td>
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<td>DNA</td>
<td>Deoxyribonucleic Acid</td>
<td>LFT</td>
<td>Liver Function Tests</td>
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<td>PIP</td>
<td>Proximal Interphalangeal Joints</td>
<td>PA</td>
<td>Pulmonary Arterial</td>
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<tr>
<td>DIP</td>
<td>Distal Interphalangeal Joints</td>
<td>PID</td>
<td>Pelvic Inflammatory Disease</td>
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<td>PMN</td>
<td>Polymorphonuclear Neutrophils</td>
<td>IUD</td>
<td>Intrauterine Device</td>
</tr>
<tr>
<td>AP</td>
<td>Anteroposterior</td>
<td>IVF</td>
<td>In Vitro Fertilization</td>
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<tr>
<td>MTP</td>
<td>Metatarsophalangeal Joint</td>
<td>FHR</td>
<td>Fetal Heart Rate</td>
</tr>
<tr>
<td>RF</td>
<td>Rheumatoid Factor</td>
<td>TSH</td>
<td>Thyroid Stimulating Hormone</td>
</tr>
<tr>
<td>CK</td>
<td>Creatine Kinase</td>
<td>FSH</td>
<td>Follicle Stimulating Hormone</td>
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<td>EMG</td>
<td>Electromyography</td>
<td>LH</td>
<td>Luteinizing Hormone</td>
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<td>MCP</td>
<td>Metacarpophalangeal Joint</td>
<td>OCP</td>
<td>Oral Contraceptive Pill</td>
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<tr>
<td>CCP</td>
<td>Cyclic Citrullinated Peptide</td>
<td>USPSTF</td>
<td>U.S. Preventive Services Task Force</td>
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<tr>
<td>DMARD</td>
<td>Disease-Modifying Antirheumatic Drugs</td>
<td>TAH</td>
<td>Total Abdominal Hysterectomy</td>
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<td>HLA</td>
<td>Human Leukocyte Antigen</td>
<td>BSO</td>
<td>Bilateral Salpingo Oophorectomy</td>
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<tr>
<td>ROM</td>
<td>Range Of Motion</td>
<td>LMP</td>
<td>Last Menstrual Period</td>
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<td>Sacroiliac</td>
<td>PAPP-A</td>
<td>Pregnancy-Associated Plasma Protein A</td>
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<td>TCA</td>
<td>Tricyclic Antidepressant</td>
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<td>Nonstress Test</td>
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<tr>
<td>ICP</td>
<td>Intracranial Pressure</td>
<td>SOB</td>
<td>Shortness of Breath</td>
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<td>NMDA</td>
<td>N-Methyl-D-Aspartate</td>
<td>CBT</td>
<td>Cognitive Behavioral Therapy</td>
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