GASTROINTESTINAL/NUTRITIONAL

Abdominal pain

Renal
  - CC: colicky right sided flank pain, nausea, vomiting, hematuria, CVA tenderness
  - Workup: UA, BUN/Cr, CT abdomen, renal US, KUB, blood cultures
  - Ddx: nephrolithiasis, renal cell carcinoma, pyelonephritis, GI etiology, glomerulonephritis, splenic rupture

Pancreas:
  - CC: dull epigastric pain that radiates to the back
  - Workup: CT abdomen, CBC, electrolytes, amylase, lipase, AST, ALT, bilirubin, alkphos, U/S abdomen
  - Ddx: pancreatitis, pancreatic cancer, peptic ulcer disease, cholecystitis/choledocholithiasis

Gallbladder:
  - CC: RUQ pain
  - Workup: RUQUS, CBC, CMP, HIDA scan, MRCP/ERCP, amylase, lipase, alk phos, bili
  - Ddx: cholecystitis, choledocholithiasis, hepatitis, ascending cholangitis, fitz-hugh-curtis syndrome, acute subhepatic appendicitis

Liver:
  - CC: RUQ pan, fever, anorexia, nausea, vomiting, dark urine, clay stool
  - Workup: CBC, amylase, lipase, liver enzymes, viral hepatitis serologies, UA, U/S abdomen, ERCP, MRCP
  - Ddx: acute hepatitis, acute cholecystitis, ascending cholangitis, choledocholithiasis, pancreatitis, primary sclerosing cholangitis, primary biliary cirrhosis, glomerulonephritis

Spleen:
  - CC: severe LUQ pain that radiates to left scapula w hx of infectious mono
  - Workup: CBC, CXR, CT/US of abdomen
  - Ddx: splenic rupture, splenic infarct, kidney stone, rib fracture, pneumonia, perforated peptic ulcer

Stomach:
  - CC: burning epigastric pain after meals
  - Workup: rectal exam (occult blood in stool), amylase, lipase, lactate, AST, ALT, bili, alk phos, upper endoscopy (H.pylori biopsies), upper GI series
  - Ddx: peptic ulcer disease, perforated peptic ulcer disease, gastritis, GERD, cholecystitis, mesenteric ischemia, chronic pancreatitis

Intestines:
  - CC: crampy abdominal pain, vomiting, abdominal distention, inability to pass flatus
  - Workup: rectal exam, CBC, electrolytes, CT abdomen/pelvis, colonoscopy
  - DDx: intestinal obstruction, small bowel / colon cancer, volvulus, gastroenteritis, food poisoning, ileus, hernia, mesenteric ischemia/infarction, diverticulitis
  - w/ alternating disrrhea/. Constipation: diverticulitis, Crohn’s disease, ulcerative colitis, abscess, IBS, celiac disease, GI parasitic infection (amebiasis, giardiasis)

Pelvis:
  - CC: RLQ pain, nausea, vomiting, dysuria, hematuria
  - Workup: pelvic exam, urine hCG, doppler U/S, rectal exam, UA, CBC, CT abdomen, laparoscopy, chlaymdia and gonorrhea testing
  - Ddx: ovarian torsion, appendicitis, ectopic pregnancy, ruptured ovarian cyst, pelvic inflammatory disease, bowel infarction / perforation, endometriosis, vaginitis, cystitis, pyelonephritis

Cholecystitis

Definition: gall bladder (cystic duct) obstruction by stone $\rightarrow$ inflammation / infection
  - 50-80% = E.coli

History and Physical Exam:
  - Biliary colic: episodic RUQ / epigastric pain beginning abruptly, continuous in duration, resolves slowly lasting 30m-hours assoc with nausea precipitated by fatty foods or large meals
  - Low grade fever, nausea/vomiting, palpable GB, murphy’s sign; boas sign (referred pain to subscapular area due to phrenic never irritation)
  - Hypoactive bowel sounds are an indicator that a perforation has occurred. Other symptoms include high fever, systemic signs of toxicity (tachycardia and increased respiratory rate), and increased abdominal pain with rebound tenderness.
**Diagnostics:**
- **US = initial test of choice:** thickened GB <3mm, distended GB, sludge, gallstones, pericholecystic fluid, sonographic murphy’s sign
- Abdominal XR: 10% of stones seen
- Labs: leukocytosis with left shift, increased bilirubin after 24 hours, alk phos and LFTs
- **HIDA scan (heptoiimidodiacetic acid): gold standard** – positive HIDA = nonvisualization of gallbladder in cholecystitis
  - HIDA shows gallbladder ejection fraction and if stone are present in the cystic ducts
  - If pt. is fasting, HIDA may show falsely decreased ejection fraction (if this is the case, inject with morphine or CCK)
- ERCP can identify cause, location and extent of biliary obstruction

**Therapeutics:**
- Conservative: NPO, IVF, abx (3rd gen cephalosporin + metronidazole)
- Cholecystectomy
- Meperidine preferred (morphine assoc with sphincter of oddi spasm)
- Acute acalculous cholecystitis: MC occur in seriously ill pt.
- Chronic cholecystitis: assoc with gallstones; strawberry GB (interior of GB resembles strawberry secondary to cholesterol submucosal aggregation) → porcelain GB (premalignant condition)

**Acute vs chronic:**
- Acute cholecystitis begins suddenly, resulting in severe, steady pain in the upper abdomen. Inflammation may cause the gallbladder to fill with fluid and its walls to thicken. Rarely, a form of acute cholecystitis without gallstones (acalculous cholecystitis) occurs.
- Chronic cholecystitis is gallbladder inflammation that has lasted a long time. It almost always results from gallstones and from prior attacks of acute cholecystitis. Chronic cholecystitis is characterized by repeated attacks of pain (biliary colic) that occur when gallstones periodically block the cystic duct.

**Definition:**
- MC cause is cholelithiasis or alcohol abuse
  - Chronic = due to alcohol abuse
  - Hyperlipidemia (hypertriglyceridemia), trauma, drugs, hypercalcemia, penetrating PUD, medications (ARTs) may also cause
- Chronic: loss of endocrine function

**History and Physical:**
- Epigastric pain radiating to back – **boring, constant radiating to back / other quadrant**; lessens when pt. leans forward, sits, or lies in fetal position
- N/V, fever, leukocytosis, dehydration / shock, sterile peritonitis, severe hypovolemia, ARDS, tachycardia >130bp → grave prognosis
- Hemorrhagic pancreatitis: may cause bleeding into flanks (Grey turner) or umbilical (Cullen sign)
- Chronic = fat malabsorption / steatorrhea
  - Triad of calcifications, steatorrhea, diabetes mellitus
- Left sided exudative pleural effusion

**Diagnostics:**
- Elevated amylase – may be transient / return to normal after 48-72 hrs
- Lipase = more sensitive but only with elevations 3x or greater – increases 7-14 days
- **Abdominal CT = diagnostic test of choice**
- AXR: sentinel loop = localized ileus – dilated small bowel in LUQ – colon cutoff sign
- WBC count elevated; liver enzymes may increase – mild hyperbilirubinemia / bilirubinuria, hyperglycemia, hypocalcemia
- Chronic: calcification on US / gallstones; amylase and lipase usually not elevated

**Therapeutics:**
- **Stop oral intake; fluid resuscitation – 90% recover with supportive measures only**
- Pain management (analgesics and Demerol) consider abx but not used prophylactically – when necessary, broad spectrum imipenem (necrotizing pancreatitis)
- ERCP if biliary sepsis suspected; only effective in obstructive jaundice
- Monitor for complications
- Chronic: oral pancreatic enzyme replacement; low fat diet; surgical removal of damaged part for pain control / pain control; STOP DRINKING
Indications for surgery: severe pain that limits functioning; intractable pain despite non-narcotic analgesics + absence of alcohol intake

Health Maintenance:
- STOP DRINKING

Acute vs chronic:
- Acute pancreatitis: isolated episode of abdominal pain accompanied by elevations in blood enzyme levels.
  - inflammation of the pancreas. >80% of the cases of acute pancreatitis are related to biliary stones or alcohol use. Acute pancreatitis may lead to chronic pancreatitis.
- Chronic pancreatitis is a painful disease of the pancreas in which inflammation has resolved, but with resultant damage to the gland characterized by fibrosis, calcification and ductal inflammation. It is possible for patients with chronic pancreatitis to have episodes of acute pancreatitis.

Anal Fissure

Definition:
- Painful, linear lesions in the distal anal canal most commonly found on posterior midline
- May involve full thickness of mucosa if untreated

History and Physical Exam:
- Severe tearing pain on defecation, often accompanied by hematochezia
- bright red blood often noted on stool or toilet paper
- skin tags seen in chronic
- MC = posterior midline

Diagnostics:
- severe painful BM causing pt to refrain from having BM → constipation, bright red blood per rectum, rectal pain

Therapeutics:
- >80% resolve spontaneously
- 1st line: analgesics, sitz bath, bulking agents and increased fluids to avoid straining
- 2nd line: topical nitroglycerin .4% ointment or topical styptic, such as silver nitrate 1% or 2% may help with healing; gentian violet solution 1% may help with healing; nifedipine ointment, botox

Health Maintenance:
- High fiber diet

Anorectal Abscess / Fistula

Definition:
- Often results from bacterial infection of anal ducts / glands. MC staph aureus, e.coli
- MC posterior midline

History and physical: throbbing rectal pain worse with sitting, coughing, defecation

Treatment of perirectal abscess: incision and drainage – no antibiotics

Appendicitis

Definition:
- obstruction of the appendix → inflammation / infection
- MC due to fecalith, or inflammation, malignancy, foreign body (collagen vascular dz, IBD)
- MC surgical emergency

History and Physical Exam:
- Initial sx = anorexia, peri umbilical / epigastric pain → constant and worsened RLQ pain (McBurney’s point), nausea, vomiting (vomiting usually occurs after pain) and rebound tenderness
- Diarrhea may occur but not common
- Low-grade fever is common; high grade fever = unlikely
- Retrocecal appendicitis = pain on rectal exam
- 20% = perforation / peritonitis (high grade fever, generalized abdominal pain, increased leukocytosis)
- MC 10y-30y
- Physical: rebound tenderness, rigidity, guarding
  - Rovsing sign: RLQ pain with LLQ palpation
  - Obturator: RLQ pain with internal and external hip rotation with bent knee
  - Psoas sign: RLQ pain with right hip flexion / tension (raise leg vs resistance)
  - McBurney’s point tenderness: the point 1/3 the distance from ASIS and navel

Diagnostics:
- Abdominal US is preferred initial imagining
CT is more sensitive and confirms diagnosis (will also help locate abnormally placed appendix)
Leukocytosis (10,000-20,000) – higher levels suggest perforation and peritonitis
Some microscopic hematuria and pyuria

Therapeutics:
- Appendectomy
- Reason to suspect abscess or perforation, broad spectrum abx administered before and after surgery
- A single dose of cefotetan 2 g intravenously (2nd gen cephalosporin) is recommended for preoperative administration for prophylaxis against infection. This is given to prevent intra-abdominal and wound infections. The targeted flora represent those that are found within the colon. In acute appendicitis without perforation, only a single dose within 60 minutes of the initial incision is indicated. Antibiotics are not indicated postoperatively in these uncomplicated cases of appendicitis. In patients with a penicillin or cephalosporin allergy, the recommended antibiotic regimen is clindamycin and one of the following: ciprofloxacin, levofloxacin, gentamicin, or aztreonam.

**Bariatric Surgery**

Definition/diagnostics:
- only proven method to maintain weight loss and reduce obesity-related morbidities and mortalities; usually last resort due to risks
- guidelines: BMI >40 (100lb over ideal body weight); BMI >35 with medical problem sequelae of obesity; failed other non-surgical programs; must be psychologically stable and able to follow post-op instructions; obesity NOT EXPLAINED by medical organic cause (ie endocrine)

Therapeutics: in US most common are RNYGB, AGB, VSG, BPD/DS
- Restrictive procedures: less weight loss, not as much morbidity
  - **Adjustable gastric banding (AGB): SMALLEST AMOUNT WEIGHT LOSS**
    - Indications: BMI >30-35 with comorbid conditions; ideal for volume eater; trains you to eat / chew slower
    - Proximal gastric pouch created using inflatable band and access port placed
    - 35-45% excess body weight lost in first few years
    - Fewer complications but need for more follow up for band adjustment
    - Complications: regurgitation, prolapse, must be seen annually for upper GI band checking; less risk but less weight loss
  - Vertical banded gastroplasty (VBG)
  - **Sleeve gastrectomy (SG): 85% stomach removed and stomach takes shape of sleeve; weight loss less than RNYGB but higher than AGB – MEDIUM AMOUNT WEIGHT LOSS**
    - Reduces stomach to <25% of original volume by resection of large portion along greater curvature including entire fundus
    - Ghrelin made in fundus (hunger hormone) → decreased with removal
    - Indications: BMI >35
    - Complications: lack of hunger, 70% weight loss at 2y
  - Malabsorptive: more weight loss than restrictive but issues with malnutrition / nutrient absorption
    - Biliopancreatic diversion (BPD + Biliopancreatic diversion with/without duodenal switch (BPD/DS)
      - Not popular bc of malnutrition issues associated; about 70-90% weight loss
  - Combo of restrictive / malabsorptive – MOST WEIGHT LOSS
    - **Roux-en-Y Gastric Bypass (RYGB): MC used bariatric surgery for tx of severe obesity in US – proximal gastric pouch created by transecting stomach; 75-85% excess body weight lost in first two years; less severe nutrition problems**
      - Bypass of most of stomach, entire duodenum, and part of small intestine
      - Pouch is restrictive, causing fullness; roux limb limits absorption
      - Indications: BMI >35
      - Complications: dumping syndrome, lifelong micronutrient supplementation (B1, B12, folate, vitamin C) and vitamins A,D,E,K and minerals

Complications:
- Early: anastomotic leak, DVT, PE, bleeding, infection, splenic injury
- Late: malnutrition / nutritional problems, marginal ulcer and anastomotic strictures, internal hernia, cholelithiasis, band slippage, band erosion, esophageal dilatation, dumping syndrome (usually occurs when pt attempts to eat large amount of simple sugars – they move from stomach to bowel too quickly → fullness, cramping/pain, n/v/d, seating, flushing, rapid heartbeat)

**Bowel Obstruction**
Definition:
- Most small bowel obstructions are caused by adhesions or hernias; other causes: neoplasm, inflammatory bowel disease, volvulus
  - Post-surgical adhesions MC (60%); hernias, Crohn’s disease, malignancy (although malignancy is MC cause of large bowel obstruction in may cause SBO)
- Large bowel obstruction more likely caused by neoplasm; others causes are strictures, hernias, volvulus, intussusception, fecal impaction
- Complete strangulation of bowel: infarction, necrosis, peritonitis, death

History and Physical Exam:
- SBO: abdominal pain, distention, vomiting of partially digested food, obstipation (severe constipation)
  - Crampy, abdominal pain, vomiting, usually follows the pain, diarrhea (early finding); mild pain → severe
  - High pitched tinkles on auscultations and visible peristalsis → hypoactive bowel sounds in late obstruction
  - Bowel sounds = high pitched and in rushes; later on = absent
- LBO = distention and pain, afebrile and tachycardic; shock may ensue

Diagnostics:
- Dehydration and electrolyte imbalance
- Upright radiographs → air fluid levels and multiple dilated loops of bowel
  - XR: air fluid levels in step ladder patterns; dilated bowel loops
- If radiography inconclusive, abdominal CT with contrast should be obtained

Therapeutics:
- 1. NPO, nasogastric suctioning, IV fluids, monitoring
- 2. Partial obstruction in hemodynamically stable pt may be managed with IV hydration and nasogastric decompression
- 3. Urgent surgical consultation is necessary when mechanical obstruction is suspected, especially of the large bowel (esp. if strangulated)
- Pain management necessary for pt. with bowel obstruction

Metabolic alkalosis would be the most likely acid-base disturbance to develop postoperatively in SBO. Volume contraction combined with gastric fluid loss is the most common cause. During this type of surgery, aggressive third-spacing of fluid into the peritoneal cavity, as well as the intestinal lumen, is the cause of volume contraction. Postoperatively, nasogastric decompression of the stomach causes acid loss. As the stomach works to replenish hydrochloric acid, bicarbonate (HCO₃⁻) is released into the serum. The patient also loses potassium from the gastric fluid during nasogastric suctioning. The combination of the volume contraction and the postoperative measures causes a hypochloremic, hypokalemic metabolic alkalosis.

Compared to a complete bowel obstruction requiring intestinal decompression and electrolyte regulation, most early postoperative bowel obstructions are partial and resolve spontaneously. Patients who experience ileus, return to normal bowel function, and then present with obstructive symptoms are more likely to have a true postoperative bowel obstruction

**Volvulus**
- Twisting of any part of the bowel in itself; MC = sigmoid, cecum
- s/sx: abdominal pain, distention, n/v, fever, tachycardia
- management:
  - 1. Endoscopic decompression
  - 2. Surgical correction

**Primary Sclerosing Cholangitis**
- Autoimmune, progressive cholestasis with diffuse fibrosis of intrahepatic and extra hepatic ducts – RARE
- MC associated with inflammatory bowel disease – 90% have ulcerative colitis +/- Crohn’s
- MC men 20-40yo
- Clinical manifestations progressive jaundice, pruritus, RUQ pain, hepatomegaly, splenomegaly
- Diagnosis: increased ALP (3-5x normal), increased GGT (cholestasis), increased ALT, AST, increased Bili, increased IgM; +P-ANCA
  - ERCP = GOLD STANDARD (increased risk developing cholangiocarcinoma)
- Management: liver transplant; meds (steroids, immune meds) = no significant benefit

**Ascending Cholangitis:**
- Definition: biliary tract infection secondary to obstruction by gallstone
  - Dx: ERCP
  - Charcot’s triad: fever/chills, RUQ pain, jaundice, increased liver enzymes
Reynold’s pentad: shock + AMS

Tx: abx (PCN + aminoglycoside), decompression of biliary tree via ERCP stone extraction

Cholelithiasis

Definition: gallstones in the gall bladder (NO INFLAMMATION)

90% cholesterol

History and Physical Exam:

Risk factors = 5Fs: fat, fair, female, forty, fertile: OCP’s (increased estrogen), Native Americans, bile stasis, chronic hemolysis, cirrhosis, infection, rapid weight loss, IBD, TPN, fibrates, increased triglycerides

MC asymptomatic (may be incidental finding)

Diagnostics:

Ultrasound = test of choice

Biliary colic: episodic RUQ / epigastric pain beginning abruptly, continuous in duration, resolves slowly lasting 30m-hours associated with nausea precipitated by fatty foods or large meals

Therapeutics:

If asymptomatic may observe or use oral bile dissolution treatment

Cholecystectomy in symptomatic pt (usually laparoscopic)

Complications:

Choleodocholithiasis: gallstones in biliary tree → +/- biliary colic or jaundice

Dx = MRCP

Tx = stone extraction via ERCP

Diarrhea

Definition: increased frequently or volume of stool 3+ liquid or semisolid stools daily for at least 2-3 consecutive days

Causes: infectious, toxic, dietary, other GI dz

Food borne / waterborne: norovirus, rotavirus, staph aureus, clostridium perfringens, vibrio (cholera), E. coli, giardia lamblia, cryptosporidium, cyclospora, salmonella, E. coli, shigella, campylobacter

History and Physical:

Include all current meds and illnesses among others who have shared meals with the patient + travel history!!

Secretory (large volumes without inflammation) indicates infection, pancreatic insufficiency, ingestion of preformed bacterial toxins, laxative use

Inflammatory (bloody diarrhea with fever, dysentery) indicates invasive organisms or IBD

Abx associated is almost always caused by c. diff colitis which in the most severe cases causes the classic psuedomembranous colitis

Diagnostics:

WBC in stool = inflammatory process

Cultures for bacterial agents, microscopy for parasites, toxin identification (if enterotoxic E. coli or C. diff I suspected) can identify infectious agents in stool

Therapeutics:

IV fluid repletion = mainstay of gastroenteritis management; PO preferred

Diet: bland low residue (BRAT / crackers, boiled vegetables, soup)

Anti-motility agents: in pt. <65 with severe signs of volume depletion

Bismuth salicylate (Pepto / kapectate): antimicrobial properties against bacterial and viral pathogens; salicylate: anti-secretory, anti-inflammatory properties

Safe in patients with dysentery

s/e: dark colored stools / darkening of tongue

Cl: children with viral dz (salicylate intake = increased likelihood reye’s syndrome – increased ICP, hepatomegaly, liver failure; tx = water restrict / lower ICP with mannitol)

Opioid agonist (diphenoxylate / atropine): bind gut wall opioid receptors and inhibit peristalsis; CNS effects; opiates cause constipation

Ind: noninvasive diarrhea

s/e: avoid in patients with acute dysentery

Anticholinergies (Phenobarb): inhibit Ach related GI motility

Do not give anti-motility drugs to pt with invasive diarrhea – may cause toxicity from bacteria

Anti-emetics:

ondansetron (Zofran): blocks serotonin receptors

dopamine blockers (Compazine / Reglan): block dopamine receptors with antiemetic effects

s/e: QT prolongation, anticholinergic / antihistamine s/e (drowsiness)

extrapyramidal sx, dystonic reactions, tardive dyskinesia, parkinsonism
• give diphenhydramine IV or add anticholinergic agent (benztrapine)
  • neuroleptic malignant syndrome: life threatening disorder – mental status changes, extreme muscle
tiredness, tremor, fever, autonomic instability (tachycardia)
• give dopamine agonist for reversal – bromocriptine / levodopa / carbidopa
  o Supportive therapy is sufficient for most patients with viral or bacterial diarrhea
  o Abx may be indicated with severe diarrhea / systemic symptoms (shigella, campylobacter, c. diff)
    o Metronidazole, oral vanco, fidaxomicin (difidic) are abx of choice for abx induced C. diff colitis
  o Tx of the underlying cause = required for noninfectious diarrhea
  o Bismuth loperamide (immodium)

Health Maintenance:
  o C. diff prevention: careful handwashing, alcohol-based sanitizers are ineffective in preventing transmission of spores

Constipation

Definition:
  o Normal bowel function ranges from three stools/day to three stools/week
  o Decrease in stool volume and increase in stool firmness accompanied by straining
  o >50yo with new onset constipation should be evaluated for colon cancer

History and Physical: +/- tender, hard abdomen

Diagnostics: +/- KUB, hypercalcemia

Therapeutics:
  o Increase in insoluble fiber (up to 10-20g/day) and fluid intake (up to 1.5 to 2L/day) and increased exercise

Health Maintenance:
  o Lasting longer than 2 weeks or with constipation refractory to modifications in diet, exercise, and fluid intake should
    undergo further investigation to detect the underlying cause
  o If treatable underlying cause is found, constipation will resolve with treatment of the disease process

Diverticular Disease

• Diverticula: small mucosal herniations protruding through intestinal and smooth muscle layer along natural openings of the
  vasa recta of the colon; sigmoid colon MC area due to highest intraluminal pressure; onset usually >40y
• Diverticulosis: uninflamed diverticula (assoc w low fiber diet***, constipation and obesity); usually asymptomatic but MC
  causes of acute lower GI bleeding
  o MC area = sigmoid
• Diverticulitis: inflamed diverticula secondary to obstruction/infection (fecaliths) $\rightarrow$ distention
  s/s: fever, LLQ pain, nausea, vomiting, diarrhea, constipation, flatulence, bloating
  dx: CT = test of choice; increased WBCs, +guaiac
  management:
    o diverticulitis: clear liquid diet, broad spectrum abx (cipiro / Bactrim) + metronidazole
    o diverticulosis: high fiber diet, fiber supplements; bleeding stops in 90% (+/- vasopressin if not)

Esophageal Neoplasms

• Squamous cell: 90-95% worldwide assoc w tobacco and alcohol use, decreased fruits/vegetable intake, achalasia, hot beverage
  ingestion, exposure of esophagus to noxious stimuli in men, nitrates
  o Decreased incidence with NSAIDs and coffee consumption
  o MC in upper 1/3 esophagus; peaks 50-70yo; increased incidence African Americans
• Adenocarcinoma – 50-80% in the US; presents in younger patients and usually presents early
  o Usually a complication of GERD / Barret’s esophagus, obesity
  o Lower 1/3 esophagus
  o Most serious complication of barrett’s is esophageal adenocarcinoma
• clinical manifestations: solid food dysphagia (fluids / soft foods usually tolerated initially); odynophagia
  o weight loss, chest pain, anorexia, cough, hoarseness, reflux, hematemesis, +/-Vircho’s node
  o hypercalcemia in patients w squamous cell (due to ectopic PTH related protein tumor secretion)
• diagnosis: upper endoscopy with biopsy = test of choice
• management: esophageal resection, XRT, chemotherapy
  **commonly spreads to mediastinum; endoscopy screening n pts w Barrett’s every 3-5 years
  o Barrett’s = transition of squamous to columnar epithelium

Esophageal Strictures
- **Esophageal web**: thin membranes in mid-upper esophagus, may be congenital
  - Plummer-vinson syndrome: dysphagia + esophageal webs + iron deficiency anemia; atrophic glossitis, angular cheilitis, koilonychias, splenomegaly
- **Schatzki ring**: mucosa lower esophageal constrictions at squamocolumnar junction; **MC assoc with hiatal hernia**
- **s/s**: dysphagia (esp to solids)
- **dx**: barium esophagram (swallow): dx test of choice for esophageal webs
- **management**: endoscopic dilation of the area

**Gastric Carcinoma**

- adenocarcinoma = MC worldwide 99%; MC males >40 and usually present in late disease
- **r/f**: H.PYLORI = MOST IMPORTANT R/F – salted, cured, smoked, pickled foods containing nitrites; pernicious anemia, chronic atrophic gastritis, achlorhydria, smoking, ETOH, blood type A
- **s/s**: indigestion, weight loss, early satiety, abdominal pain / fulness, nausea, post-prandial vomiting, dysphagia, melena, hematemesis; iron deficiency anemia
  - signs of metastasis: virchow’s node, sister mary joseph’s node, ovarian METS, palpable nodule on rectal exam (blumer’s shelf); left axillary lymph node involvement (irish sign)
- **dx**: upper endoscopy with biopsy; linitis plastica – diffuse thickening of stomach wall d/t cancer infiltration (worst type)
- **tx**: gastrectomy, XRT, chemo; poor prognosis

**Disease encasement of the hepatic artery is considered an indicator of unresectability of gastric cancer.** Gastric cancer is typically surgically resected along with regional lymph nodes with complete disease eradication representing the best chance for positive outcomes. During gastric and lymph node resection, complete abdominal exploration should occur. **Indicators of unresectability** include vascular involvement of the aorta, hepatic artery, or proximal splenic artery. Distant metastases are also an indicator of unresectability, although locoregional metastases are not always unresectable. Lymph nodes that are located in the aorto-caval region, in the porta hepatis, or behind the pancreas are also considered unresectable.

**GERD**

**Definition:**
- transient relaxation of LES → gastric acid reflux → esophageal mucosal injury

**History / Physical Exam:**
- **typical symptoms**: heartburn (pyrosis) hallmark often retrosternal and post prandial (MC 30-60min post eating, increased in supine position and often relieved with antacids); regurge (water brash or sour taste in mouth), dysphagia, cough at night (acid aspiration into the lungs causes lung irritation)
  - halitosis, cough, hiccupping, sore throat, laryngitis, atypical chest pain
  - **atypical symptoms**: hoarseness, aspiration pneumonia, “asthma” (bronchospasm from lung contact with acid), noncardiac chest pain, weight loss
  - **ALARM SYMPTOMS**: dysphagia, odynophagia, weight loss, bleeding

**Complications:**
- esophagitis, esophagus stricture, barrett’s esophagus, esophageal adenocarcinoma
- barrett’s: esophageal squamous epithelium replaced by precancerous metaplastic columnar cells from the cardia of the stomach

**Diagnostics:**
- clinical diagnosis based on history especially if present with classic, simple symptoms
- **endoscopy: often 1st used**: useful to evaluate persistent symptoms, GERD with culture, malignancy, new sx >50y etc
  - use with: pt older than 45 with new onset sx, long standing or frequently recurring symptoms, failure to respond to therapy or symptoms indicating more severe conditions like anemia, dysphagia, or recurrent vomiting
  - make sure to eval / rule out MI
- **esophageal manometry**: decreased LES pressure – often done if normal upper endoscopy
- **24h ambulatory pH monitoring**: gold standard (not usually done)

**Therapeutics:**
1. As needed pharm therapy: antacids and OTC H2 receptor blockers: if alarm of atypical sx upper endoscopy is next appropriate step
2. Initiation of scheduled pharmacologic therapy: PPIs are drug of choice in severe disease
3. H2 blocker at bedtime and PPI in the daytime may be helpful in pt with significant nighttime symptoms
4. Avoid: beta agonist, alpha adrenergic antagonist, nitrates, calcium channel blockers, anticholinergics, theophylline, morphine, meperidine, diazepam, barbiturate agents (decrease LES pressure)
5. Nissen fundoplication if refractory

**Health Maintenance:**
6. Lifestyle modifications: elevate head of bed by six inches, avoid recumbency for 3 hours after eating, eating small meals, avoid fatty/spicy, citrus, chocolate, caffeinated products, peppermint; decrease fat and alcohol intake, weight loss, smoking cessation

7. Predisposing factors: obesity, pregnancy, diabetes, hiatal hernia, connective tissue disorders

Scientific Concepts:
- multifactorial: increased gastric acid, incompetent lower esophageal sphincter (LES), esophageal motility disorders and delayed gastric emptying +/- hiatal hernia
- reflux esophagitis is result of recurrent reflux of gastric contents into distal esophagus bc of mechanical or functional abnormality
- present in ~10% of the population
- infants: 50%
- protective factors: gravity, lower esophageal sphincter tone, esophageal motility, salivary flow, gastric emptying, tissue resistance
- can → barrett’s which can predispose to malignancy

Hematemesis / GI bleed

- **Diagnosics:** usually pt with upper GI bleeding
- **s/sx:** vomiting blood, coffee ground emesis, melena (black/tarry stools), orthostatic dizziness, confusion, angina, severe palpitations
- **Eval:** HEMODYNAMIC STABILITY
- **Causes:** peptic ulcer, esophageal ulcer, Mallory-weiss tear, variceal hemorrhage/portal hypertensive gastropathy, malignancy
- **Physical exam:** look for signs of hypovolemia
  - Mild/moderate (<15%): resting tachycardia
  - moderate (>15%): orthostatic hypotension
  - severe (>40%): supine hypotension
- **Labs:** CBC, chemistries, liver tests, coags, ?cardiac enzymes/ECGs for those at risk for MI
- **Treatment:**
  - NGT lavage: if unclear pt has ongoing bleeding
  - IV access: 2 large bore (18 gauge) IVs, FLUIDS!!
  - Transfusion:
  - RBC: if unstable, high-risk and hgb <9 OR if stable and hgb <7; goal to maintain hgb >7
  - Platelets: if actively bleeding with platelets <50K
  - Coags: if INR >2, should give FFP
  - TIPS: stent in the liver to shunt blood away from portal vein into hepatic vein to bypass cirrhotic liver parenchyma; main complication – encephalopathy and accumulation of toxic substances in the brain since liver isn’t acting as a filter anymore

Hemorrhoids

**Definition:** enlarged venous plexus that increases with increased venous pressure: worse with pregnancy, defecation (especially if constipated), prolonged sitting, obesity

**History and Physical:**
- **internal:** intermittent rectal bleeding = MC, hematochezia BRBPR (toilet paper / toilet water), vague anal discomfort, rectal pain with internal suggest complication; purple nodules if prolapses; uncomplicated internal are neither palpable or tender
  - stage I internal: confined to anal canal and may bleed with defecation
  - stage II: protrude from anal opening but reduce spontaneously; bleeding and mucoid discharge may occur
  - stage III: require manual reduction after bowel movement; may develop pain and discomfort
  - stage IV: chronically protrude and risk strangulation
- **external:** MC perianal pain, aggravated with defecation, tender palpable mass; thrombosis may be precipitated by cough / heavy lifting
  - visible peri-anally

**Diagnostics:**
- visual inspection, digital rectal exam, fecal occult blood testing
- proctosigmoidoscopy, colonoscopy in pts with hematochezia to r/o proximal sigmoid dz

**Therapeutics:**
- conservative (stage I and II): high fiber diet, increased fluids, warm sitz baths, bulk laxatives
  - higher stage = suppositories and anesthetic and astringent properties
  - topical rectal hydrocortisone for pruritus and discomfort +/- analgesics
- surgical (stage IV): if failed conservative management, debilitating pain, strangulation
perioperative complications and is associated with a higher risk of readmission. Management strategies include medical therapy, such as proton pump inhibitors, and surgical intervention, such as bariatric surgery.}

**Conclusion:**

The prevalence of paraesophageal hiatal hernias is increasing, likely due to the rising obesity rate. This condition warrants careful evaluation and management to prevent complications and improve patient outcomes. Future research should focus on understanding the natural history of paraesophageal hiatal hernias and developing novel treatment approaches.
Sliding hiatus hernia is another type of hiatus hernia that is described as displacement of the gastroesophageal junction above the diaphragmatic hiatus by greater than 2 cm. By definition, this type of hernia differs from paraesophageal hiatal hernias in that the stomach remains in its position.

**Inflammatory Bowel Disease**

<table>
<thead>
<tr>
<th></th>
<th>UC</th>
<th>Crohn’s</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
<td>• Onset sudden or gradual</td>
<td>• Some genetic predisposition</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Cause = unknown; M = F; 15-35</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Onset gradual; waxes and wanes</td>
</tr>
<tr>
<td><strong>History / Physical</strong></td>
<td></td>
<td>Abdominal cramps, diarrhea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Low grade fever, polyarthritis, fatigue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Abdominal cramps, diarrhea</td>
</tr>
<tr>
<td><strong>Area affected</strong></td>
<td>• Limited to colon (begins in rectum with contiguous spread proximally to colon)</td>
<td>Any segment of GI tract; mouth → anus</td>
</tr>
<tr>
<td></td>
<td>• Rectum always involved</td>
<td>• MC in terminal ileum / right colon (RLQ pain)</td>
</tr>
<tr>
<td><strong>Depth</strong></td>
<td>• Mucosa and sub mucosa only</td>
<td>• transmural</td>
</tr>
<tr>
<td><strong>Clinical manifestations</strong></td>
<td>• Abdominal pain; LLQ MC, colicky</td>
<td>abdominal pain; RLQ pain (crampy); weight loss more common in Crohn’s</td>
</tr>
<tr>
<td></td>
<td>• tenesmus, urgency</td>
<td>• diarrhea with no visible blood usually</td>
</tr>
<tr>
<td></td>
<td>• blood diarrhea hallmark (stools with mucus/pus), hematochezia MC in UC</td>
<td>• perianal dz; fistulas, stricture, abscesses, granulomas</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td>• primary sclerosing cholangitis; colon CA; toxic megacolon (more common in UC)</td>
<td>• malabsorption: B12 and iron deficiency</td>
</tr>
<tr>
<td></td>
<td>• smoking decreases risk for UC</td>
<td>• smoking makes worse</td>
</tr>
<tr>
<td><strong>Colonoscopy</strong></td>
<td>• uniform inflammation +/- ulceration in rectum /colon = “sandpaper” appearance</td>
<td>• skip lesions (normal areas btwn inflamed areas) with cobblestone appearance</td>
</tr>
<tr>
<td></td>
<td>• pseudo polyps</td>
<td></td>
</tr>
<tr>
<td><strong>Barium studies</strong></td>
<td>• stovepipe sign (loss of hastral marking)</td>
<td>• string sign (Ba flow through narrowed inflamed / scarred area due to transmural strictures</td>
</tr>
<tr>
<td><strong>Labs</strong></td>
<td>• P-ANCA (more common in UC)</td>
<td>• ASCA</td>
</tr>
<tr>
<td><strong>Surgery</strong></td>
<td>curative</td>
<td>noncurative</td>
</tr>
</tbody>
</table>

Both UC and CD: arthritis (seronegative spondyloarthropathies, ankylosing spondylitis), episcleritis

Systemic: fevers, sweats, weight loss, malaise, fatigue, erythema nodosum, pyoderma gangreosum

**Diagnostics:**
- Crohn’s: upper GI series with small bowel follow through is test of choice in acute disease
  - Colonoscopy is most valuable tool for establishing diagnosis / determining extent / guiding treatment
  - Avoid contrast studies
  - Biopsy will show involvement of entire bowel wall; granulomas are frequent
  - Blood test: increased ESR, anemia, nutritional and electrolyte imbalance during exacerbation
  - Anti-saccharomyces cerevisiae antibody and perinuclear antineutrophil cytoplasmic antibody may distinguish crohn and UC
- UC: flex sigmoidoscopy test of choice in acute disease
  - Radiograph may show colonic dilation
  - Colonoscopy = contraindicated!!! → perforation
  - Barium enema = CI → may cause toxic megacolon
  - Anemia, increased sed rate, decreased albumin
  - Perinuclear antineutrophil cytoplasmic antibody and anti-saccharomyces cerevisiae antibody may be used to distinguish UC from crohn’s

**Therapeutics:**
- Elemental diet
  - Crohn’s: supplement with vitamin B12, folic acid, vitamin D
  - Smoking cessation
Surgery not curative in Crohn’s; curative in UC

Aminosalicylates (sulfasalazine, mesalamine) ➔ corticosteroids ➔ immune modifying agents

1. 5-aminosalicylates: anti-inflammatory agents; good for flares and remission
   a. Oral mesalamine: especially active in terminal small bowel and colon; long acting works throughout entire small intestine and colon; **best for maintenance**
   b. Topical mesalamine: rectal suppositories and enemas: topical are effective in distal colon
   c. Sulfasaline: works primarily in the colon; s/e: **higher side effect profile** with sulfasalazine (hepatitis, pancreatitis, allergic reaction, fever, rash); give folic acid with sulfasalazine

2. Corticosteroids: rapid acting anti-inflammatory drugs used for acute flares only ➔ oral and topical; long term risk = osteoporosis, increased infections, weight gain, edema, cataracts

3. Immune modifying drugs: 6-mercaptopurine, azathioprine and methotrexate = steroid sparing

4. Anti-tnf drugs: inhibits proinflammatory cytokines (-mab)

5. Initial treatment for uncomplicated Crohn’s disease is **immunosuppressant therapy**. If this is not effective, surgery may be required, especially for complications of perforation, hemorrhage, and toxic colitis. The distinction between Crohn’s disease and ulcerative colitis is important as chronic treatment approaches vary. Resection is strongly recommended for ulcerative colitis patients who are young, have frequent recurrence, or are steroid-dependent. Avoidance of surgery is desired in patients with Crohn’s disease due to the natural history of recurrence. Indications for surgery in both Crohn’s disease and ulcerative colitis include intractable or fulminant disease, massive hemorrhage, colonic obstruction, cancer prophylaxis, colon dysplasia, or cancer. Indications for surgery specific to ulcerative colitis include toxic megacolon, colonic perforation, or extracolonic disease. Indications for surgery specific to Crohn’s disease include stricture and obstruction, refractory fistula, abscess, or perianal disease unresponsive to medical therapy.

**Scientific Concepts:**

- **Etiology = idiopathic** (most likely immune reaction to GI tract flora); MC in Caucasians 15-35y

**Jaundice**

**Definition:**

- Yellowing of skin, nail beds, sclera by bilirubin deposition as consequence of hyperbilirubinemia; not a disease but sign of disease
- Occurs with increased bilirubin overproduction (hemolysis), decreased hepatic bilirubin uptake, impaired conjugation, biliary obstruction, hepatitis
- **Usually first sign is scleral icteris (conjunctival is first, first sign tho)**

**Causes:**

- Extravascular hemolysis / ineffective erythropoiesis, gilbert syndrome, crigler-najjar dsyndrome, dubin-johnson, biliary tract obstruction, viral hepatitis, physiologic jaundice of newborn

**History and Physical:**

- **Hemolytic = prehepatic**
  - Increased indirect / unconjugated bilirubin, mild hyperbilirubinemia
  - Dark urine due to hemoglobinuria; dark stool
- **Obstructive = post hepatic**
  - Cholestasis = bile duct blockage ➔ increased conjugated bili
  - Ex. Cholestasis / pancreatic CA
  - Increased direct/ conjugated hyperbilirubinemia
  - GGT and ALP elevated
  - Dark urine = increase direct bilirubin
  - Acholic stools = biliary obstruction (white)
- **Hepatocellular (intra hepatic)**
  - Increased indirect and direct bilirubin; ALT and AST markedly elevated
  - Dark urine = increased direct bilirubin
  - ETOH hepatitis: AST > ALT 2:1
  - Acute hepatitis: increased ALT and AST > 1000; ALT >AST usually
  - Chronic hepatitis: increased ALT:AST but <500

**Diagnostics:**

- Bilirubin >2.5mg/dL
- Increased bilirubin without increased LFTs = suspected familial bilirubin disorders (gilbert’s, dubin-johnsons) and hemolysis
- Labs: serum total and unconjugated bilirubin, alk phos, AST/ALT, PT/INR, albumin
  - Normal alk phos / aminotransferases: not likely due to hepatic injury / biliary tract disease
  - Increased alk phos out of proportion to AST/ALT: biliary obstruction / intrahepatic cholestasis
  - AST/ALT elevation: intrinsic hepatocellular disease
Treatment: depends on cause

1. **Causes of postoperative hepatic jaundice**: Drugs, hypotension, hypoxia, sepsis, hepatitis, "sympathetic" hepatic inflammation from adjacent right lower lobe infarction of the lung or pneumonia, preexisting cirrhosis, right-sided heart failure, hepatic abscess, pylephlebitis (thrombosis of portal vein), Gilbert syndrome, Crigler-Najjar syndrome, Dubin-Johnson syndrome, fatty infiltrate from TPN

2. What blood test results would support the assumption that hemolysis was causing jaundice in a patient?
   a. Decreased—Haptoglobin, Hct
   b. Increased—LDH, reticulocytes Also, fragmented RBCs on a peripheral smear

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**Melena / Hematochezia** (GI bleed)

**Melena**: black tarry stool
- Causes: upper GI bleed, gastric cancer, duodenal ulcer, right sided colon cancer, portal hypertension with esophageal varices, severe erosive esophagitis, Mallory-Weiss syndrome
  - Peptic ulcer = upper abdominal pain
  - Esophageal ulcer: odynophagia/dysphagia, GERD
  - Mallory-weiss: emesis, excessive coughing
  - Variceal hemorrhage / portal hypertensive gastropathy: jaundice, ascites
  - Malignancy: dysphagia, early satiety, involuntary weight loss, cachexia
- Dx: EGD; blood/stool/breath test for H.pylori

**Hematochezia**: BRBPR (bright red blood per rectum)
- Causes: lower GI bleed, hemorrhoids, anal fissures, polyps, proctitis, rectal ulcers, colorectal cancer
  - Hemorrhoids: painless bleeding w wiping
  - Anal fissures: severe rectal pain with defecation
  - Proctitis: rectal bleeding + abd pain
  - Polyps: painless rectal bleeding, no red flag signs
  - Colorectal cancer: painless rectal bleeding + change in bowel habits, pt is 50-80yo
- Dx: colonoscopy, enteroscopy, barium XR, radionuclide scanning, angiography, laparotomy

**Labs for both**: CBC, chemistries, liver tests, coags
- Active bleeding = normocytic RBC; chronic bleeding = microcytic RBC or iron deficiency anemia

Treatment: similar for both
- Endoscopic thermal probe: burning blood vessel / tissue causing ulcer
- Endoscopic clips: close a bleeding blood vessel / other source of bleeding in tissue in GI tract
- Endoscopic injection: injection of liquid near source of bleeding to stop flow of blood
- Angiographic embolization: injects particles into affected blood vessels
- Endoscopic intravariceal cyanojection: special glue injected near affected area to stop bleeding of large stomach essels
- Band ligation: small rubber bands around hemorrhoids or swollen eins (esophageal varices) to cut off blood supply
- Octreotide, somatostatin preferred for better safety profiles / less incidence side effects
- Sclerotherapy: decrease risk for rebleeding in pt with esophageal varices

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**Mesenteric Infarction / Ischemia**

**Definition**: when artery supplies blood becomes blocked / narrowed; superior mesenteric artery = MC site
**Presentation**: acute onset pain out of proportion with history of afib, MI, CHF or young person with hx of oral contraceptive pills / illicit drugs
**Causes**: blockage in arteries from tumor/blood clot; narrowing of arteries supplying blood to bowel from atherosclerosis
**s/sx**: acute onset severe diffuse abd pain out of proportion to exam – usually someone with afib; possibly with vomiting / constipation + blood in stool
**dx**: XR shows thumb-printing of small bowel or right colon due to submucosal bleeding; CT, colonoscopy, angiography, or MRangiography
  - mesenteric angiography is considered gold standard
**tx**: early surgical eval for emergent operative intervention is mandatory for revascularization
• chronic: postprandial epigastric pain usually within first hour after eating that resolves in 2-3 hr in pt with history of PVD, smoker, or DM

Nausea/Vomiting

Postoperative nausea and vomiting (PONV) – affects 30% children and adults after anesthesia

- sequelae: wound dehiscence, esophageal rupture, aspiration, dehydration, increased intracranial pressure, pneumothorax
- emetogenic drugs: nitrous oxide, opioids (dose-related), phystostigmine
- propofol = LEAST emetogenic general anesthetic
- risk score: female gender, nonsmoker, history of motion sickness/previous PONV, expected administration of postop opioids → increased by 10 (0), 20 (1), 40 (2), 60 (3), 80% (4) with each increasing number of risk factors present
- treatment:
  - preop fasting (2-6 hours)
  - antiemetics: scopolamine patch, dexamethasone, ondansetron (avoid with QTc prolongation)
  - rescue antiemetics: prochlorperazine, droperidol

Pancreatic Carcinoma

- epidemiology: 4th leading cause death, M>W, risk increases with age; greater incidence in smokers, **cigarette smoking and alcohol abuse
- MC ductal adenocarcinoma located at pancreatic head
- presentation: painless jaundice = pathognomonic, weightloss/epigastric abd pain, clay-colored stools, jaundice + palpable non-tender gallbladder (Courvoisier’s sign), virchow’s node = lymph in left supraclavicular fossa
- diagnosis: abdominal CT scan (75% shows tumor at the head of the pancreas) – ERCP, pancreatic biopsy, abdominal MRI, elevated serum bilirubin, abnormal LFTs
- CA 19-9 present in about 80% of patients with pancreatic cancer
- Treatment: only about 20% can be removed by pancreaticoduodenectomy (whipple); when can’t be removed with surgery → combination of radiation therapy and chemotherapy

Pancreatic Pseudocyst

- Definition: cystic collection of tissue, fluid, and necrotic debris surrounding the pancreas
- Causes: acute/chronic pancreatitis, trauma to chest (steering wheel trauma), classically occur 2-3 weeks after acute pancreatitis
- Sx: abdominal pain
- Physical exam: abdominal mass
- Diagnosis: CT = test of choice
- Tx: if pseudocyst persists for 4-6 weeks or continues to enlarge → percutaneous drainage, surgical decompression (pancreaticogastrostomy), cyst fluid drained
- Complications: can become infected and lead to peritonitis

Peptic Ulcer Disease

Definition: secondary to imbalance of 1. Decreased mucosal protective factors 2. Increased damaging factors (acid); gastric ulcers more common in elderly

History and Physical:
  - Etiology: h. pylori, NSAIDs, Zollinger Ellison syndrome (suspect GI malignancy in nonhealing GU-ZES and gastric cancer)
  - Asymptomatic, dyspepsia, worse at night
    - Ulcer-like or acid dyspepsia: relief with food, antacids, anti-secretory agents; worse before meals or 2-5h after meals; nocturnal symptoms
  - Food provoked: pain 1-2 hours p meals and weight loss
  - GI bleed: PUD = MC cause of upper GI bleed

Diagnostics:
  - Endoscopy = gold standard / most accurate diagnostic test
    - Bx to r/o malignancy – alarm sx: >50yo, dyspepsia, history of UG, anorexia, wt loss, anemia, dysphagia
  - Upper GI series: all GU seen on UGI serious should be followed with endoscopy to r/o malignancy
  - H. pylori testing:
    - Endoscopy with biopsy = gold standard + rapid urease test
    - Urea breath test to confirm eradication after therapy
    - H. pylori stool antigen = >90% specific – confirm eradication after therapy
    - Serologic antibodies: confirm infection not eradication
Complications: bleeding (melena, hematemesis, dizziness), perforation (sudden onset severe, diffuse abdominal pain, rigid abdomen, rebound tenderness), penetration (pain radiating to back), obstruction (vomiting)

Therapeutics:
- H. pylori: clarithromycin + amoxicillin + PPI (CAP)
- H. pylori negative: PPI, H2RA, misoprostol, antacids, bismuth compounds, sucralfate
  - PPI: drug of choice / most effective drug to treat PUD – 30m before meals; s/e = B12 deficiency
  - H2 blocker: cimetidine = lots of drug interactions + anti-androgen effects
  - **Misoprostol**: good for preventing NSAID related ulcers but not for healing already existing ulcers; CI in premenopausal women bc abortifacient
    - Antacid: milk of magnesia → diarrhea; amphogel → constipation; Maalox, Mylanta
    - Bismuth compounds: antibacterial and cytoprotective; s/e = darkening tongue / stool, constipation
    - Sucralfate: forms viscous adhesive ulcer coating; promotes healing / protects mucosa; may reduce bioavailability of H2RA, PPI
  - Parietal cell vagotomy if refractory
  - **Weight loss** is experienced by up to 30% of patients post-surgically. Patients may limit food intake due to early satiety. Distention or discomfort may occur shortly after even a moderate-sized meal as the residual gastric pouch is smaller. Patients should be counseled to eat smaller and more frequent meals. Additionally, weight loss can be a result of other postsurgical complications such as maldigestion or dumping syndrome.

**Pilonidal Cysts**
- Chronic **pilonidal disease**, for which the **definitive treatment requires surgical excision of all sinus tracts**. Pilonidal disease occurs when the skin and subcutaneous tissue at or near the upper region of the natal cleft of the buttocks becomes infected, often in the setting of ongoing inflammation and damage of local hair follicles. This problem is **most common in young adults** and often occurs in patients with **sedentary occupations, obesity, or a positive family history**. Patient presentations can range from asymptomatic to chronic pain and drainage. Acute abscess formation can also occur. Pilonidal disease is a clinical diagnosis that does not require any imaging or laboratory procedures.
- **Incision and drainage** is necessary in the setting of an acute pilonidal abscess, but is not the definitive step in management due to high recurrence rates after incision and drainage treatment alone.

**Pyloric Stenosis**
Presentation: infants feed well for first 2-3 weeks then present with nonbilious vomiting after most or every feeding
- Pediatric pt <3mo, projectile vomiting, dehydration
- **Physical exam** = palpable epigastric olive-shaped mass (pathognomonic)
- **dx**: ultrasound → double track; barium studies reveal “string sign” or “shoulder sign”
- **labs**: hypochloremic, hypokalemic – metabolic alkalosis
- **tx**: surgical incision of hypertrophied pyloric muscle → Ramstedt procedure (pyloromyotomy)

**Small Bowel Carcinoma**
- Small bowel cancer is RARE – delay in diagnosis is common, adenocarcinoma is MC, highest in duodenum
- **Diagnosis**: delays are common → discovery of disease at late stage and poor treatment outcomes
- **s/sx**: abdominal pain (intermittent and crampy), n/v, anemia, overt GI bleeding, jaundice, weight loss, other/no sx
- **Risk factors**: hereditary cancer syndromes – HNPPC aka Lynch syndrome, cystic fibrosis, crohn’s disease, intake of alcohol/refined sugar/red meat/ salt-cured and smoked foods
- **dx**: CT, wireless capsule endoscopy, push enteroscopy, double-balloon endoscopy; screening for fecal occult blood
  - tumor marker: CEA
- **tx**: localized of the small bowel are best managed with wide segmental surgical resection with adjuvant chemotherapy

**Toxic Megacolon**
- **definition**: usually complication of inflammatory bowel disease, such as UC and Crohn’s and/or some infections → C.dif
  - Life threatening form of colon distention; pt presents with fever, distended abdomen with peritonitis and shock
- **Diagnosis**: abdominal plain-film radiography shows colonic dilation, KUB shows dilated colon >6cm
  - at least 3 of the following: fever >101.5F, HR >120, neutrophilic leukocytosis >10.5 x 10^9, anemia
- **Treatment**: decompression of colon is required – in some cases, colostomy or even complete colonic resection may be required
PREOPERATIVE/POSTOPERATIVE CARE

Acid Base Disorders

Baseline values: easy way to remember – 24/7 40/40
- 24 (HC03 → base)
- 7.4 pH
- 40 (CO2 → acid)

1. LOOK AT PH (7.35-7.45 = normal)
   a. <7.35 = acidosis
   b. >7.45 = alkalosis

2. LOOK AT PCO2 → normal, low, or high (35-45 = normal)
   a. Increased CO2, decreased pH = respiratory acidosis
      i. pH low, high PCO2, normal bicarb
      ii. lungs fail to excrete CO2: slow breathing, pulmonary disease, neuromuscular disease, drug-induced hypoventilation (opiates, barbiturates)
      iii. can result in hyperkalemia - potassium exchange for hydrogen ions in intracellular space causing increase in potassium
   b. Decreased CO2, increased pH = respiratory alkalosis
      i. pH high, low PCO2, normal bicarb
      ii. excessive elimination of CO2 → hyperventilation, pulmonary embolism, fever, hyperthyroid, anxiety, salicylate intoxication, septicemia
      iii. can result in hypokalemia / hypokalcemia → paresthesias, carpopedal spasm, tetany
   c. No change in CO2 in relation to pH → look at HCO3

3. LOOK AT HCO3 → normal, low, high (20-26 = normal)
   a. Decreased HCO3, decreased pH = metabolic acidosis
      i. Low pH, normal PCO2, low bicarb
      ii. CALCULATE ANION GAP
         1. Na – (Cl + HCO3) = 10-16
         2. Increased anion gap (>16) → MUDPILES
            a. Methanol, uremia, diabetic ketoacidosis, paraldehyde, infection, lactic acidosis, ethylene glycol, salicylates
         3. Low anion gap (<10) = loss of bicarb
            a. Diarrhea, pancreatic / biliary drainage, renal tubular acidosis
   b. Increased HCO3, increased pH = metabolic alkalosis
      i. High pH, normal PCO2, high bicarb
      ii. Loss of hydrogen (vomiting), bulimia, overdose of antacids, addition of bicarb (hyperalimentation therapy)

Cardiac Disease

- 6 predictors of surgical cardiac complications (likelihood increases with increased number of factors)
  o Ischemic heart disease, CHF, cerebrovascular dz, high-risk operation, pre-op tx with insulin, pre-op creatinine >2
  o Other r/f: age, smoking, abnormal cardiac stress test, long-term beta-blocker therapy, COPD
- Hx of MI = 5-10% increased risk of post-op MI
  o Pre-op EKG on pt >40, avoid surgery with current unstable angina, control stage 2 HTN prior to surgery, take antihypertensive meds day of surgery, provide prophylactic abx therapy with rheumatic heart disease; send to cardio for clearance
- Noninvasive stress test before noncardiac operations for pt with: active cardiac conditions, pt who need vascular operations
- Coronary revascularization before noncardiac operations with: left main coronary artery stenosis, stable angina with 2 vessel coronary disease, stable angina with two vessel disease, significant LAD coronary artery stenosis with EF <50%, high risk unstable angina or non STE MI / acute STEMI

Compartment Syndrome

- Increased pressure in muscle compartment usually due to injury (fracture of long bone)
- Sx: severe pain, numbness, decreased ROM
  - Loss of two point discrimination = earliest sign
  - 5p’s: pain out of proportion, pallor, paresthesia, pulselessness, paralysis, poikilothermia
- Volkmann’s contracture: permanent flexion contracture of hand at risk → claw-like deformity; passive extension of fingers is restricted
- Dx: compartment pressure – normal = 0-8mmHg; >30 = compartment syndrome
- Tx: fasciotomy = only treatment

### Deep Venous Thrombosis
- **Definition**: caused by clot formation in veins
- **r/f**: Virchow’s triad – stasis (post-surgical, immobility, venous insufficiency), hypercoagulability (factor V leiden, cancer, OCP + smoking, pregnancy), trauma (surgery, cellulitis)
- **presentation**: edema of one extremity, positive Homan’s sign (extend leg and push foot towards the head → elicits pain)
- **dx**: venous duplex ultrasound = 1st line
  - d-dimer: r/o DVT in low risk pt
  - venography = gold standard
- **tx**: IV heparin then bridge to warfarin
  - recurrent DVT = lifetime anticoagulation

### Electrolyte Disorders

#### Hyponatremia
- **Definition**: serum sodium of < 135 mmol/L
- Peripheral and presacral edema, pulmonary edema, JVD, hypertension, decreased hematocrit, decreased serum protein, decreased BUN/CR
- **Presentation: Muscle cramps and seizures**
  - Hypervolemic hyponatremia – CHF, nephrotic syndrome, renal failure, cirrhosis
  - Euvolemic hyponatremia – SIADH (steroids, hypothyroid)
  - Hypovolemic hyponatremia – sodium loss (renal, non-renal)
- **Treatment**:
  - Asymptomatic → free water restriction
  - moderate hyponatremia → IV normal saline, loop diuretics may be added
  - severe hyponatremia → hypertonic (3%) saline
- **Serum Na should be corrected slowly** — by ≤ 10 mEq/L over 24 h to avoid osmotic demyelination syndrome

#### Hypernatremia
- **Definition**: serum sodium of > 145 mmol/L
- Etiology: Diarrhea, burns, diuretics, hyperglycemia, diabetes insipidus, a deficit of thirst
- Poor skin turgor, dry mucous membranes, flat neck veins, hypotension, increased BUN/CR ratio > 20:1
  - Decrease circulating volume = decrease of flow to kidneys means more bound urea in the blood which means ↑ BUN
- **Treatment**: intravenous (IV) 5% dextrose in water (D5W). Rapid overcorrection causes cerebral edema and pontine herniation.*

**Diabetes insipidus** - Low urine sodium (but high serum sodium) and polyuria usually indicate diabetes insipidus
  - **Neurogenic (central)** is caused by deficient secretion of vasopressin (ADH - anti-piss-hormone) from the posterior pituitary
  - **Nephrogenic DI** is caused by kidneys that are unresponsive to normal vasopressin levels - usually inherited X-linked or from lithium or renal disease
  - Urine osmolality of less than 250 despite hyponatremia, indicated Diabetes Insipidus

#### Hyperkalemia
- **Definition**: serum potassium of > 5.5 mEq/L
<table>
<thead>
<tr>
<th>Condition</th>
<th>Definition</th>
<th>Presentation</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypokalemia</td>
<td>serum potassium of &lt; 3.5 mEq/L</td>
<td>Muscle cramps, constipation, flattened/inverted T waves, U waves</td>
<td>Potassium repletion</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>remember to <strong>NOT use dextrose-containing fluids</strong> as this will stimulate insulin release and shift potassium within the cell which worsens the hypokalemia</td>
</tr>
<tr>
<td>Hypocalcemia</td>
<td>serum total calcium &lt; 8.4 mg/dL, ionized fraction of calcium &lt; 4.4 mg/dL</td>
<td>QT prolongation, Trousseau’s sign, Chvostek’s sign</td>
<td>IV calcium gluconate or calcium chloride</td>
</tr>
<tr>
<td></td>
<td>Labs: ↓ Ca+, ↓ PTH, ↑ phosphate EKG = Prolonged QT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypomagnesemia</td>
<td>serum magnesium &lt; 1.8 mg/dL</td>
<td>Muscle weakness, hyperreflexia, prolonged QT, PR and wide QRS, ventricular tachycardia, torsades de pointes</td>
<td>IV magnesium sulfate (acute) or oral magnesium oxide (chronic)</td>
</tr>
<tr>
<td>Hypermagnesemia</td>
<td>serum magnesium &gt; 2.6 mg/dL</td>
<td>Muscle weakness, prolonged QT, PR and wide QRS</td>
<td>IV isotonic saline, loop diuretics can be considered</td>
</tr>
<tr>
<td>Hyperphosphatemia</td>
<td>serum phosphate &gt; 4.5 mg/dL</td>
<td>Etiology: Chronic kidney disease</td>
<td>calcium carbonate, restrict potassium</td>
</tr>
<tr>
<td></td>
<td>Etiology: Typically asymptomatic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypophosphatemia</td>
<td>serum phosphate &lt; 2.5 mg/dL</td>
<td>Weakness, muscle and bone pain, osteomalacia, rickets</td>
<td>IV phosphate replacement</td>
</tr>
</tbody>
</table>
**Hematologic Disease**

- DVT diagnosed in 20% general surgery pt and 30% colorectal pt without prophylaxis
- **TX:**
  - Compression devices
  - Subcutaneous heparin and LMWH are equivalent in reducing both DVT and PE
    - LMWH has simpler dosing and decreased risk heparin-induced-thrombocytopenia BUT more expensive and ? dose-related increased risk of bleeding
    - Fondaparinux is new Xa inhibitor with efficacy for prophylaxis
    - No enough evidence to say one is better than the other
  - Very low risk: early ambulation
  - Low risk: mechanical prophylaxis with intermittent compression devices
  - Moderate: LMWH or low-dose unfractionated heparin or IPC
  - High risk: IPC + LMWH or low-dose heparin
  - Extended course (4 weeks) or LMWH may be indicated for pt undergoing abdominal resection / pelvic malignancy
- Risks with treatment: wound hematomas (MC risk), mucosal bleeding, reoperation

| ACNP's suggested risk stratification for perioperative thromboembolism* |
|---------------------------------|-----------------|-----------------|
| **Risk category**               | **Mechanical heart valve** | **Venous thromboembolism** |
| High (≥10%/yr risk of ATE or ≥10%/yr risk of VTE) | Any mechanical mitral valve | CHADS, score of 5 or 6<br>Recent (< 3 mo) stroke or TIA | Recent (< 3 mo) VTE<br>Severe thrombophilia |
| Moderate (6%-10%/yr risk of ATE or 4%-10%/yr risk of VTE) | Bileaflet aortic valve and one of the following: atrial fibrillation, prior stroke/TIA, hypertension, diabetes, heart failure, age > 75 | CHADS, score of 3 or 4 | VTE within past 3–12 mo<br>Recent VTE<br>No other thrombembolic conditions<br>Active cancer |
| Low (≤4%/yr risk of ATE or ≤2%/yr risk of VTE) | Bileaflet aortic valve without atrial fibrillation and no other risk factors for stroke | CHADS, score of 0–2<br>(and no prior stroke or TIA) | Single VTE within past 12 mo<br>and no other risk factors |


**TABLE 2**

**Bridging for patients taking warfarin**

**Preoperatively**
- Give last warfarin dose on day –6
- Start bridging on day –3 with one of the following:
  - Enoxaparin 1 mg/kg twice daily or 1.5 mg/kg once daily
  - Dalteparin 100 IU/kg twice daily or 200 IU/kg once daily
  - Tinzaparin 175 IU/kg once daily
  - Nadroparin 85 IU/kg twice daily
- Give last bridging dose on the morning of day –1
- Do not routinely check the international normalized ratio on the day before surgery (≥ 90% will be < 1.5) except for very high-risk cases or patients having neuraxial anesthesia
- Do not continue aspirin; stop 7 days preoperatively, and resume 7 days postoperatively
- Give the patient precise instructions for the bridging plan

**Postoperatively**
- Give double dose of warfarin on the first 1–2 days postoperatively
- Resume bridging when hemostasis is secured:
  - 24 hours after low-bleeding-risk surgery
  - 48–72 hours after high-bleeding-risk surgery
- Do not use therapeutic-dose bridging at all for:
  - Cardiac surgery
  - Intracranial or spinal surgery
  - Cancer surgery (eg, Whipple procedure)
  - Reconstructive surgery (eg, skin grafting)

**Fluid / Volume Disorders**

- ~1% pt have reduced kidney function post-op
  - Reduce risk by pushing fluids, avoiding NSAIDs, exposure to IV contrast minimized/avoided
  - Blood transfusions may be necessary before surgery, esp with active hemorrhage / anemia
- **CALCULATING FLUIDS:** daily sensible / insensible loss in adult = 1500-2500mL depending on age, gender, weight
  - Pt weight (kg) x 30 = fluid over 24 hours
  - Increase for fever, hyperventilation
  - Measure intake and output / weight pt daily
  - General rule: 2000-2500mL of 5% dextrose in normal saline or LR solution daily
  - Re-evaluate IV fluid orders q24 hours
- Don’t have to measure electrolytes postop, unless extra fluid loss, sepsis, preexisting electrolyte abnormalities, renal insufficiency
• Don’t add potassium during first 24 hours because K is already increased during surgery (from stress) with increased aldosterone activity
• Otherwise, 20 mEq potassium added to each liter if pt has good urine output
• Replace post-op ionized serum calcium in pt with thyroidectomy or parathyroidectomy

• Indications for catheter placement: long procedure, urologic / low pelvic surgery, need to monitor fluid balance

**Metabolic Disease**

• Pt should be assessed at admission for hx of diabetes (with blood glucose testing)
• Blood glucose may be elevated preoperatively in DM pt esp with physical trauma + emotional stress
  - Perioperative hyperglycemia treated with IV short-acting insulin or SQ sliding scale insulin
• Elevated postop blood glucose in diabetic pt → increased chance postop infection and longer hospital stays and higher risk heart disease
  - Risk surgical site infection increases with degree of hyperglycemia, levels >140 = predictor of site infection
• IV insulin = best for perioperative glucose control bc of rapid onset, short half-life, immediate availability
• Post-op glycemia control
  - Normal: 90-100mg/dL – control with IV insulin
  - Moderate control: 120-200mg
• Post-op monitoring for: hyperglycemia, hypoglycemia, infection, poor healing, wound issues, CVD (double risk for men, quadruple for women)

**Pulmonary Disease**

• MC perioperative complications = pulmonary
• r/f: operative site and presence of pre-existent lung disease
• OPTIMALLY STOP SMOKING AT LEAST 8 WEEKS BEFORE SCHEDULED SURGERY
• COPD:
  - Treat aggressively to get to best possible baseline
  - Minimum of one week of therapy: smoking cessation, administration of antibiotics for purulent sputum, bronchodilators if indicated
• Asthma: poorly controlled = increased risk for postop complications; well-controlled = confers little additional risk
  - Poorly controlled: step-up in asthma therapy (course of steroids if FEV1 or PEFR are below predicted value)
  - Elective surgery → free of wheezing with PEFR > 80% of predicted / personal best prior to surgery
  - For those who need endotracheal intubation → administer inhaled rapid-acting beta agonist 2-4 puffs / neb treatment 30 min prior to intubation
  - 1-2 days systemic steroids sometimes advised to prevent acute bronchoconstriction at time of intubation
• Pulmonary fibrosis / restrictive lung disease: treat infection, remove sputum, stop smoking
• Acute lower respiratory tract infection (tracheitis, bronchitis, pneumonia): CONTRAINDICATIONS TO ELECTIVE SURGERY
  - Emergency surgery: humidification of inhaled gas, remove lung secretions, bronchodilators, antibiotics
• Diagnosis: done by PFTs before hospital admission
  - Do with exceptional dyspnea, exercise tolerance, cough, production of sputum, hx of smoking, previous pulm complications, asthma, age, body weight
  - Mild pulmonary compromise for non-abdominal / thoracic surgery don’t need PFTs
  - Test via simple spirometry with forced expiratory outflow
  - If airflow reduced → measure response to bronchodilators / get an ABG
    • Increased risk if FEV1 <50% or PaCO₂ > 45
• Treatment:
  - Preop: cigarette cessation, optimize underlying condition, patient education
    • Antibiotics for lower respiratory tract infections (purulent sputum / change in sputum character)
  - Intraop: increased risk for surgeries >3-4 hours; upper abdominal / open AAA repair, open thoracotomy, head/neck surgeries = GREATEST risk for postop complications
    • Lung protective ventilation for those undergoing abdominal surgery → low tidal ventilation of PEEP at 6-8cm water to reduce adverse pulmonary events
  - Postop: lung expansion, incentive spirometry, CPAP for some patients, early mobilization
**Substance Use Disorder**

- Key features: substance taken in large amounts over longer periods than intended, unsuccessful efforts to cut down, lot of time spent obtaining substance / recovering from effects, craving, recurrent despite hazards, tolerance / withdrawal
- Alcohol / drug dependence = 5-10% of population; males > females (15% of pt seen in primary care have at-risk pattern of drug/alcohol abuse)
- Surgical issues: venous access, arterial injury, DVT, abscess formation, tissue compression, crush injury, ischemia (compartment syndrome), poor wound healing, altered consciousness, difficulty with pain management
- Patients with known opioid use disorder on methadone should be advised to continue the use of methadone, including on the day of surgery to avoid precipitating withdrawal. For this reason, it is advised to continue chronic narcotics on the day of surgery.

**Dx:** screen for alcohol / drug use in ALL patients → quantity, frequency

- > 2 drinks per day in men and > 1 drink per day in women or anyone > 65 yrs
- CAGE questions: need to cut down; annoyed by people criticizing behaviors, guilt about use, eye opener right in the morning
- Blood alcohol content (2 drinks = 0.08%)
- Tox screen
- MCV, GGT, AST/ALT can be useful to diagnose / monitor

**Treatment:** hx of drug abuse = contraindication to surgery

- Avoid surgery if pt is acutely intoxicated if possible
- Pharmacological agents: Naltrexone (antagonist), acamprosate, disulfiram (causes nausea, headache, flushing, respiratory distress when combined with alcohol), topiramate, methadone (opioid agonist), buprenorphine (partial agonist), naltrexone
  - Opioids work on mu receptor
  - Combine pharmacotherapy with counseling
  - Treatment of comorbid psychiatric disorders improves outcome

**Anesthesia:**

- Opioid use: predicting analgesic needs = difficult
- Sympathomimetic drug use (cocaine, amphetamine) → greater pressor response to stimuli like oro-tracheal intubation and surgical incision → may need more anesthetic
- Volatile solvents: can mimic alcohol intoxication – chronic use → cardiomyopathy and dysrhythmia
  - Sensitivity to sympathomimetic agents and myocardial depression with volatile anesthetic agents
- Patient controlled anesthesia (PCA pumps) usually involve morphine – risky for opioid-abusing patients / those in recovery to relapse
  - Avoid IV bolus of opioids

**Tobacco Use / Dependence**

- Epidemiology: 1 in 5 smokes cigarettes in US, ~ 30% those undergoing general surgery smoke
- Smoking within 1 year of surgery: increased postop complications, increased hospital cost, higher resource use, decreased wound healing ability
- Optimally stop smoking at least 8 weeks before surgery
  - HIGHEST rate of pulm complications for those who had stopped smoking 1-8 weeks pre-op
  - Recent smoking cessation may pose greater risk pulm complication bc of increased cough / sputum production
  - Short-term cessation → reduction in vasoconstriction / irregularly heart activity; decreased risk of blood clot, better wound healing, improved PFTs
- Perioperative nicotine replacement = controversial → nicotine gum / lozenge helpful even on morning of surgery for a preop fast from cigarettes
- Use of NRT in hospitals not necessary to treat withdrawal symptoms but may be good setting to initiate treatment
- To help quit: bupropion 1-2 weeks before quit date

**Post-operative fever**

- 5 W’s: wind, water, wound, walking, wonder drugs/whopper.
  - **Wind** (atelectasis): first 24-48 hours
    - Bronchial breathing / ipsilateral tracheal deviation could occur
    - Tx: prevent with smoking cessation at least 2 weeks before surgery, incentive spirometry, mycolytic, expectorant, inhaled beta-agonists
  - **Water (UTI):** 48-72 hours → MOST COMMON
- Usually from catheter / GU instrumentation
- MC nosocomial infections in the hospital
- Tx: antibiotic therapy based on urine culture / sensitivity; remove infected indwelling catheter
  - **Wound (infection): >72 hours**
    - Staph = MC organism implicated
  - **Walking (thrombophlebitis): >72 hours**
    - Superficial and deep can occur → venography = most accurate for diagnosis in lower leg
    - Dx: ultrasound = initial diagnostic tool
    - Anticoagulant using heparin/ LMWH
  - **Wonder drugs: 1 week**
    - Anesthetic, sulfa drugs
  - **Whopper (abscesses): 1+ week**
    - Can cause ileus / anastomotic leak
    - Tx: percutaneous drainage / surgical debridement with abx

- **Diagnosis:** physical exam, CXR, UA, blood cultures, CBC, abdominal CT, cultures (sputum, wound, abscess)

**Wounds / infections**

- Usually appears 5-10 weeks after surgery
- s/sx: fever (first sign), pain at incision site, erythema, drainage, induration, warm skin
- **common bacteria:** staph aureus (20%), e. coli (10%), enterococcus (10%), clostridium – bronze-brown weeping, tender wound
- **dx:** CBC (leukocytosis / leukopenia), blood cultures, imagine studies (CT to locate abscess)
- **tx:** remove skin sutures/staples, rule out fascial dehiscence, pack wound open, send wound culture, administer abx
  - **delayed course:** usually wounds that open due to infection are left to heal by secondary intention

**At risk for infection risks:** diabetes

**ASA score:**

<table>
<thead>
<tr>
<th>ASA Classification</th>
<th>Definition</th>
<th>Examples, incidence but not limited to:</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASA I</td>
<td>A normal healthy patient</td>
<td>Healthy, non-smoking, no or minimal alcohol use</td>
</tr>
<tr>
<td>ASA II</td>
<td>A patient with mild systemic disease</td>
<td>Mild diseases only without substantive functional limitations. Examples include (but not limited to): current smoker, social alcohol drinker, pregnancy, obesity (BMI &gt; 30), well-controlled DMHNT, mild lung disease</td>
</tr>
<tr>
<td>ASA III</td>
<td>A patient with severe systemic disease</td>
<td>Substantive functional limitations; One or more moderate to severe diseases. Examples include (but not limited to): poorly controlled DM or HTN, COPD, morbid obesity (BMI &gt; 40), active hepatitis, alcohol dependence or abuse, implanted pacemaker, moderate reduction of ejection fraction, ESRD undergoing regularly scheduled dialysis, premature infant PCA &lt; 60 weeks, history (&gt;3 months) of MI, CVA, TIA, or CAD/stents.</td>
</tr>
<tr>
<td>ASA IV</td>
<td>A patient with severe systemic disease that is a constant threat to life</td>
<td>Examples include (but not limited to): recent (&lt; 3 months) MI, CVA, TIA, or CAD/stents, ongoing cardiac ischemia or severe valve dysfunction, severe reduction of ejection fraction, sepsis, DIC, ARD or ESRD not undergoing regularly scheduled dialysis</td>
</tr>
<tr>
<td>ASA V</td>
<td>A moribund patient who is not expected to survive without the operation</td>
<td>Examples include (but not limited to): ruptured abdominal/thoracic aneurysm, massive trauma, intracranial bleed with mass effect, ischemic bowel in the face of significant cardiac pathology or multiple organ/system dysfunction</td>
</tr>
<tr>
<td>ASA VI</td>
<td>A declared brain-dead patient whose organs are being removed for donor purposes</td>
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</tbody>
</table>

**CARDIOVASCULAR**

**Aortic Aneurysm / Dissection**

- AAA trifecta = back pain, pulsatile mass, hypotension (usually male >60yo); sometimes syncope; males = 8x more likely
  - Involves all 3 layers – intima, media, adventitia
  - Screening: ultrasound, if male >65 and ever a smoker
Diagnosis: ultrasound = initial imaging; CT for further eval; angiography = gold standard
- >3cm if considered aneurysmal
- Monitor annually if >3-4.4 cm; monitor q6mo if >4.5-5.0 cm and refer to vascular surgery; monitor q3mo if 5-5.4 cm; >5.5 cm or >0.5 cm expansion in 6 months = immediate surgical repair even if asymptomatic

Treat:
- Surgical repair if >5.5 cm or expands >0.6 cm per year
- Treat with beta blocker

Dissection = severe, tearing (ripping, knife-like) chest pain radiating to the back between scapula; pulse variation between right and left arm
- Just 2 layers – intima, media
- Ascending aorta = surgical emergency; descending = medical therapy (beta blocker)
- Dx: CXR shows widened mediastinum; MRI angiography is gold standard for evaluation
- Tx: lower blood pressure
  - Type A (proximal aortic dissection): surgical management
  - Type B (distal aortic dissection): medical management initially, surgery only if needed

Arterial Embolism / Thrombosis
- Caused by arterial occlusion: 6p's: pain, paralysis, pallor, paresthesia, poikilothermia, pulselessness
- Risk factors: afib, mitral stenosis
  - MC site of arterial occlusion by embolus = common femoral artery
  - MC cause embolus from the heart = afib
  - Sources of embolism: heart (85%), aneurysms, atheromatous plaque
- Lower extremity > upper extremity
- Dx: angiography = gold standard; ECG, echocardiogram to look for clot, MI, valve vegetation
- Tx: anticoagulated with IV heparin (bolus followed by constant infusion)
  - surgical embolectomy via cutdown/fogarty balloon (bypass if embolectomy failed)
    - Fogarty balloon catheter – catheter with balloon tip that can be inflated with saline; used for embolectomy
  - Immediate preop management: anticoagulated with IV heparin; A-gram
    - Look for these postoperative after reperfusion of limb: compartment syndrome, hyperkalemia, renal failure from myoglobinuria, MI

Arterial / Venous Ulcer Disease
- Venous ulcer: chronic defects of the skin that last longer than 4 weeks → usually on lower leg just above ankle
  - Physical exam: irregularly shaped wound with well-defined borders, painless, brown-stained skin, dry, itchy, reddened
  - r/f: obesity, age, family hx chronic venous insufficiency, hx of DVT
  - dx: hx and clinical signs and symptoms
    - measurement of ankle-brachial index (ABI): ABI <0.9
    - eval for diabetes
    - consider tissue for culture, bone scan for osteomyelitis
    - vasculitis suspected – biopsy
    - no healing after 4 weeks – biopsy to rule out malignancy
    - imaging: duplex sonography
  - tx: first line = below-knee compression stockings
    - surgical debridement, regular brisk walking 30 min a day, elevate feet above heart
    - surgery: overall prognosis is poor
    - nonhealing ulcers – wound care clinic

Chest pain; history of angina
Chest pain or discomfort, heaviness, pressure, squeezing, tightness that is increased with exertion or emotion
- Predictable, relieved by rest and/or nitroglycerine
- Chest pain or substernal pressure
- Lasts < 10-15 min
- Relieved with rest or NTG
- Signs: Levine sign—clenched first over the sternum and clenched teeth when describing chest pain

Workup:
- **EKG:** normal, Q-waves (prior MI)
- **Cardiac stress test** demonstrates reversible wall motion abnormalities/ ST depression >1 mm
- **Coronary angiography** provides a definitive diagnosis
- If severely symptomatic despite medical therapy and being considered for PCI
- Patients with troublesome symptoms difficult to diagnose
- Angina symptoms in a patient who has survived a cardiac death event and patients with ischemia on noninvasive testings

### Treatment:
- **Nitroglycerin** sublingual → IV NTG
- Beta-blockers
- Severe: **angioplasty** and **bypass**

### Prognosis:
- Depends on LVEF: < 50% (increased mortality)
- Vessel(s) Involved: left main (poor, 2/3 of the heart)

### Unstable angina

**Chest pain or discomfort,** heaviness, pressure, squeezing, tightness that is **increased with exertion** or **emotion**
- **Previously stable** and predictable symptoms of angina that are **now more frequent, increasing or present at rest**
- Chronic angina—**increasing** in frequency, duration, or intensity of pain
- New-onset angina—**severe** and **worsening**
- **Angina at rest**
- **O2 demand unchanged**, supply decreased, secondary to low resting coronary flow

### Workup:
- **EKG:** ST-segment or T-wave abnormalities
- Cardiac enzymes: **normal** troponin, CK-MB

### Treatment:
- Admit to the unit with **continuous cardiac monitoring,** establish IV access, O2
- Pain control with NTG and morphine
- **ASA** and/or clopidogrel - (Plavix reduces the incidence of MI in patients with unstable angina compared with ASA alone 9-12 months of therapy)
- LMWH continued for at least 2 days
- **β-Blockers**
- LMWH
- Replace electrolytes
- If the patient responds to medical therapy → **stress test** to determine if catheterization/revascularization necessary
- **Revascularization** if symptoms persist despite medical therapy
- Reduce risk factors: stop smoking, weight loss, treat DM/HTN
- **ACE inhibitors** and **statins** - start patients with unstable angina or NSTEMI with high LDL on **HMG-CoA reductase inhibitor** (statin)

### Prinzmetal variant angina

**Coronary artery vasospasms causing** transient ST-segment elevations, **not associated with clot**
- Smooth muscle **constriction (spasm)** of the coronary artery **without obstruction** → leads to MI, ventricular arrhythmias, sudden death
- Known triggers: hyperventilation, cocaine or tobacco use, provocative agents (acetylcholine, ergonovine, histamine, serotonin)
- Nitric oxide deficiency → increased activity of potent vasoconstrictors and stimulators of smooth muscle proliferation 50-yr-old, females

### Nonexertional chest pain similar to unstable angina

- Preservation of exercise capacity
- Look for a **history of smoking** (#1 risk factor) or **cocaine abuse**
- Pain is cyclical (mostly **occur in morning hours**, no correlation to cardiac workload)
Diagnostics:

- EKG may show inverted U waves, ST-segment or T-wave abnormalities
- Cardiac enzymes: normal troponin, CK-MB
- Check Mg level, CBC, CMP, lipid panel

Treatment:

- Stress testing with myocardial perfusion imaging or coronary angiography
- Pharmacotherapy SL, topical, or IV nitrates (initial)
- Antiplatelet, thrombolytics, statins, BB
- Once diagnosis made—CCB and long-acting nitrates used for long-term prophylaxis (amlodipine)

Claudication

- Reduced arterial blood supply can’t meet metabolic demand of muscles utilized during walking
- Reduction in blood flow to leg muscles (mostly lower extremity but sometimes upper extremity), MC by atherosclerotic plaque; NOT due to blood clot / embolization, chronic limb ischemia
- Usually lower extremities
- r/f: smoking, diabetes, hypertension, hypercholesterolemia, advanced age, male gender, obesity, sedentary lifestyle, family history vascular dz, heart attack, stroke
- s/sx: pain in leg with walking, relieved within few minutes of rest, reproducible at the same walking distance each time; calf muscle atrophy, hair appendages die, dry scaly skin, prolonged cap refill
- dx: ABI <0.9; angiography = gold standard
  - normal 1.2-1; mild = 0.9-0.7; moderate = 0.7-0.4; severe/rest pain: <0.4
- tx: platelet inhibitors (cilastazol, aspirin, Plavix); treat lipids (statins); revascularization with PTA/stents; exercise (walk to point of claudication); **beta blockers = contraindicated in isolated PAD → worsens claudication!!**
  - Surgery: bypass in presence of rest pain and provides relief of sx in 80-90% of patients
    - Surgical graft bypass (fem-pop), angioplasty, endarterectomy, surgical patch angioplasty

Dyspnea on exertion

- Arrhythmia: afib, inappropriate sinus tachy, sick sinus syndrome / bradycardia
  - Hx: palpitations, syncope
  - PE: Irregular rhythm, pauses
  - Dx: ECG, event recorder, holter monitor, stress testing
- Myocardial: cardiomyopathies, coronary ischemia
  - Hx: dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, chest pain or tightness, prior coronary artery diseases or a fib
  - PE: edema, JVD, S3, displaced cardiac apical impulse, murmur, crackles
  - Dx: ECG, BNP, echo, stress testing, coronary angiography
- Restrictive: constrictive pericarditis, pericardial effusion/tamponade
  - Hx: chest pain, dyspnea
  - PE: paradoxical pulse
  - Dx: EKG showing low voltage QRS along with electric alternans; echo with increased pericardial fluid; XR shows water bottle heart
- Valvular: aortic insufficiency/stenosis, congenital heart disease, mitral valve insufficiency/stenosis
  - Hx: dyspnea on exertion
  - PE: murmur, JVD
  - Dx: echo

Peripheral Artery Disease

- MC from atherosclerosis and is significant independent r/f for cardiovascular and cerebrovascular morbidity/mortality
- Initially asymptomatic → claudication, ischemia, pain with exercise
- Acute arterial occlusion may be caused by thrombosis or embolism
- Thrombotic disease may also be result of trauma, hypovolemia, inflammatory arteritis, polycythemia, dehydration, repeated arterial punctures, hypercoagulable states
- Intermittent claudication: foot / lower leg pain with exercise relieved by rest; usually first symptom of PAD
- Exam: femoral / distal pulses weak or absent; skin changes (shiny, atrophic skin), dependent rubor, PAINFUL ULCER
- Caused by arterial occlusion: 6p’s: pain, paralysis, pallor, paresthesia, poikilothermia, pulselessness
- **Diagnosis:** Doppler ultrasound flow studies can be used to determine systolic pressures in the peripheral arteries; Dx: doppler ultrasound flow studies, ankle brachial index (<0.9)
  - Angiography = gold standard
- **Tx:** aggressive r/f modification – discontinue smoking, diabetes/HTN/hyperlipidemia must be controlled
  - Exercise, b-blocker, ACE-I, statin, platelet inhibitors (cilostazol, aspirin, clopidogrel), revascularization with PTA, bypass grafts, stenting, angioplasty, endarterectomy

**Syncope**

- **Definition:** sudden, brief loss of consciousness with loss of postural tone followed by spontaneous revival
- **Pathophysiology:** insufficient cerebral blood flow
  - Causes: decreased cardiac output: cardiac disorders that obstruct outflow/systolic dysfunction/diastolic dysfunction/arrhythmias
- **MC causes:** vasovagal, idiopathic
- **Presentation:** pt is motionless, limp, cool extremities, weak pulse, shallow breathing; sometimes muscle jerks
- **Near-syncope:** light-headed, sense of impending faint
- **Red flags:** syncope during exertion, multiple recurrences within a short time, heart murmur or other findings suggesting structural heart disease, older age, significant injury during syncope, family hx unexpected cardiac death/exertional/unexplained
  - Usually quick recovery time vs seizure -> longer recovery time
- **Testing:** EKG, pulse oximetry, sometimes echocardiography, sometimes tilt table testing, blood tests only if clinically indicated, CNS imagine rarely indicated
- **If recurrent / results in injury → do further eval**
- **Treatment:** identify and treat underlying cause

**Varicose Veins**

- **Definition:** dilated superficial veins in lower extremities; usually no obvious cause
- **r/f:** prior pregnancy, obesity, family history, prolonged sitting/standing, history of phlebitis
- **causes:** valvular incompetence
- **s/sx:** dilated, tortuous veins develop superficially in lower extremities (mostly great saphenous vein); may be asx or achy / fatigue; chronic distal edema, abnormal pigmentation, fibrosis, atrophy, skin ulceration
- **dx:** clinical, duplex ultrasound
- **tx:** graduated compression stocking, leg elevation, regular exercise, unna boots, skin grafts, radiofrequency / laser ablation, compression sclerotherapy, surgical stripping

**WPW:**

- catheter ablation of bypass tracts = treatment for pt with symptomatic arrhythmias; safer, cost-effective, just as successful as surgery

**Sick Sinus Syndrome**

Permanent pacemakers are the therapy of choice in patients with symptomatic bradyarrhythmias in sick sinus syndrome.

**Indications for CABG**

Class I indications for CABG from the American College of Cardiology (ACC) and the American Heart Association (AHA) are as follows

- Over 50% left main coronary artery stenosis
- Over 70% stenosis of the proximal left anterior descending (LAD) and proximal circumflex arteries
- Three-vessel disease in asymptomatic patients or those with mild or stable angina
- Three-vessel disease with proximal LAD stenosis in patients with poor left ventricular (LV) function
- One- or two-Vessel disease and a large area of viable myocardium in high-risk area in patients with stable angina
- Over 70% proximal LAD stenosis with either an ejection fraction (EF) below 50% or demonstrable ischemia on noninvasive testing

- **CABG is the treatment of choice in a diabetic with two or three vessel disease**

**Cardiac Functional Capacity**

- Functional capacity = indicator of postoperative cardiac complication risk
- assess at initial preoperative evaluation
- poor functional capacity → increased cardiac complications in noncardiac surgery
- expressed in metabolic equivalents (1 MET equals 3.5 mL O2 uptake/kg per minute, which is the resting oxygen uptake in the seated position)
- self-care, the ability to complete activities of daily living (ADLs), vacuuming, walking 2 mph, and writing are examples of poor functional capacity (less than 4 METs)
- The ability to walk up a flight of stairs, walk 4 mph, walking a golf course, doing yard work, and cycling are examples of moderate functional capacity (4-10 METs)
- Jogging, playing singles tennis, swimming, and skiing are examples of excellent functional capacity (greater than 10 METs).
- One specific indicator of increased risk of postoperative cardiopulmonary complications is the inability to climb two flights of stairs or walk four blocks.

ENDOCRINOLOGY

**Adrenal Carcinoma**
- Adrenal cortical cancer (ACC) = rare; less common benign masses (myelolipoma/lipoma/pseudocyst) – usually found incidentally
- Functional tumors: pheochromocytoma, aldosteronoma, cortisol-producing adenomas
- Dx: 24 hour catecholamines, MRI or CT of abdomen to visualize catecholamine secreting adrenal tumor
- Tx: complete adrenalectomy
  - Pre-op nonselective alpha blockade: phenoxybenzamine or phentolamine 7-14 days followed by beta blocker to control HTN → NO BETA BLOCKERS TO PREVENT UNOPPOSED ALPHA CONSTRICTION → LIFE THREATENING HTN
  - Open transabdominal approach for surgical resection → greater visualization
  - Laparoscopic techniques preferred for benign adrenal tumors → reduced blood loss / length of stay, quicker recovery but decreased visualization of tumor and adjacent structures

**Adrenal Crisis**
Acute phase of adrenal crisis is treated with IV saline and hydrocortisone.
To better mimic the normal physiologic response the baseline dose should be doubled for the duration of the illness. Doses should be increased 5-10 fold with major events such as surgery.

**Fatigue**

### Causes of subacute and chronic fatigue

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<thead>
<tr>
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**Endocrinologic/metabolic**
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<td>Generally normal exam</td>
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<td>None</td>
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<td>Polymyalgia rheumatica</td>
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<td>Decreased range of motion of shoulders, neck, and hips</td>
<td>Erythrocyte sedimentation rate</td>
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<td>Generally normal exam</td>
<td>Screening test (eg, PHQ-2, PHQ-9)</td>
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<td>Generalized nervousness, panic attacks, phobias</td>
<td>Tachycardia, muscle tension</td>
<td>Screening test (eg, GAD-7)</td>
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<tr>
<td>Somatization disorder</td>
<td>Multiple chronic constitutional and localized complaints</td>
<td>Generally normal exam</td>
<td>Screening test (eg, SSS-8)</td>
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<tr>
<td>Medication toxicity</td>
<td>Variable</td>
<td>Generally normal exam</td>
<td>None</td>
</tr>
<tr>
<td>Substance use</td>
<td>Variable</td>
<td>Generally normal exam</td>
<td>None</td>
</tr>
</tbody>
</table>

![Flowchart Diagram]
Heat / Cold Intolerance

- Heat intolerance:
  - Hyperthyroidism (thyrotoxicosis): nervousness, weight loss, increased appetite, heat intolerance, sweating, muscular weakness/fatigue, increased bowel frequency, polyuria, menstrual irregularities, infertility
    - s/sx: goiter, tachycardia, afib, warm moist skin, thyroid thrill/bruit; exophthalmos
    - labs: TSH low/absent, increased T3, T4
    - indications for surgery: very large goiter, low RAI uptake, suspicion of malignancy, ophthalmopathy, tx of pregnant pt or children, women who want to become pregnant within 1 year of treatment, pt with amiodarone-induced hyperthyroidism
      - subtotal thyroidectomy: nearly all thyroid removed but parathyroid and recurrent laryngeal nerves spared
      - complete reserved for those with graves ophthalmopathy
  - The tremor that occurs with hyperthyroidism is typically a high frequency, low amplitude tremor that is present with action.
  - Cold intolerance: myxedema, pituitary tumor

- Cold intolerance: myxedema, pituitary tumor
  - Hypothyroid: increased TSH, low free T4
  - Pituitary tumor:
    - decreased TSH → cold intolerance, weight gain, fatigue, coarse hair, myxedema
    - decreased ACTH causes hypocortisolism resulting in fatigue, slow return to health after minor illness and orthostatic hypotension
  - hypoparathyroid = common complication of thyroidectomy

- most patients with Ca levels <12 = asymptomatic
- s/sx: nausea, vomiting, loss of appetite, weakness, fatigue, constipation, confused, lethargic, polyuria with renal failure, cardiac arrhythmias
- labs: ALWAYS increased PTH
- primary: increased PTH (parathyroid adenoma)
- secondary: increased PTH by physiologic response to hypocalcemia or vitamin D deficiency (CKD is MC cause of secondary hyperparathyroidism)
- s/sx: Bones, Stones, Groans and Psychic Moans
  - bone loss from increased PTH and Ca absorption = pain in bones
  - renal loss of calcium and phosphate = kidney stones
  - increase GI absorption of calcium and abdominal cramps = groans
  - irritability, psychosis, depression = moans
- dx: usually found incidentally; increased Ca is MC metabolic abnormality; increased PTH from bone, GI tract, kidneys
  - labs: increased calcium, increased PTH, increased calcium, decreased phosphorus
    - urine: hyperphosphaturia, hypercalciuria
- tx: remove the parathyroid adenoma → subtotal or total
  - supplement with calcium and Vitamin D
  - with super high calcium → treat with IV fluids, Lasix, calcitonin
  - may need to treat osteoporosis with bisphosphonates

<table>
<thead>
<tr>
<th>Common Cause</th>
<th>Serum Ca</th>
<th>Serum Phos</th>
<th>PTH</th>
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<tr>
<td>adenoma</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>parathyroidectomy</td>
<td>↓</td>
<td>↑</td>
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### Summary Table

- **Hyperparathyroidism**
  - Serum Ca: ↑
  - Serum Phos: ↓
  - PTH: ↑
  - Common Cause: adenoma

- **Hypoparathyroidism**
  - Serum Ca: ↓
  - Serum Phos: ↑
  - PTH: ↓
  - Common Cause: parathyroidectomy
Ectopic PTH ↑ ↓ ↓ malignancy

Vit D malabsorption ↓ ↓ ↑ celiac disease, other GI disease

↓ D with no phosphate excretion from the kidney. ↓ ↑ ↑ renal failure, pseudo hypoparathyroidism

Palpitations
- Ddx: anxiety, electrolyte abnormalities (hypokalemia, hypomagnesemia), exercise, hyperthyroidism, ischemic heart disease, ingestion of stimulant drugs (coca, amphetamines, caffeine), medications (digoxin, beta-blockers, hydralazine, diuretics, minoxidil), pheochromocytoma, hypoglycemia in DM1, mitral valve prolapse, afib, WPW, sick sinus syndrome
- Structural heart disease (CAD, cardiomyopathy, valve disease)
  - ECG, echo, exercise stress test
  - Labs: CMP, CBC, TSH, urine toxicology
  - Tx based on diagnosis

Pheochromocytoma
- Definition: catecholamine secreting adrenal tumor that secretes norepinephrine and epinephrine → HTN, palpitations, HA, sweating
- Over 90% benign
- Sx: 5Ps → pressure (increased BP), pain (HA), perspiration, palpitations (tachycardia), pallor
- Dx: 24 hour plasma fractionated metanephrines catecholamines
  - MRI or CT to visualize tumor
- Tx: complete adrenalectomy
  - Preoperative nonselective alpha-blockade: phenoxybenzamine or phentolamine 7-14 days followed by beta blockers to control HTN → NO BETA BLOCKERS TO PREVENT UNOPPOSED ALPHA CONSTRICION → LIFE THREATENING HTN
  - Laparoscopic adrenalectomy, long-term monitoring

Thyroid Carcinoma
- MC risk factor = radiation exposure
- Papillary carcinoma = MC (80%); PAPILLARY = POPULAR; MC in females 40-60yo
- May or may not feel palpable thyroid nodule/mass
- Solitary thyroid nodule: must be >1cm to be palpated; thyroid adenoma is MC benign nodules
- Dx: ultrasound; lesions >1cm should be biopsied; smaller ones can be followed and re-evaluated if they grow
  - High risk on US: microcalcifications, hypoechogenicity, solid nodule, irregular nodule margins, chaotic intramodular vasculature, nodule more tall than wide
Thyroid uptake scan: cancerous = cold (doesn’t take up iodine from radioactive thyroid scan); non-cancerous = hot (takes up iodine)

- COLD = CANCER
  - If cold nodule → fine needle aspiration

**Tx:** depends on type – complete or partial removal of thyroid with chemotherapy and external beam radiation for anaplastic thyroid cancer

### Thyroid Nodule

**Evaluation of thyroid nodule:**
- Palpable nodule: childhood irradiation to head and neck confers 25 fold increase in thyroid cancer
- Confirm by ultrasound: microcalcifications, hypoechoegenicity, a solid nodule, irregular nodule margina, chaotic intramodular vasculature, nodule that is more tall than wide
- Thyroid uptake scan: cancerous lesions doesn’t make hormone and will not take up iodine (cold nodule)
- Non-cancerous will take up iodine at either normal rate or quicker rate (hot nodule)
- Fine needle aspiration: surgical resection if indicated
- **Dx:** start with ultrasound
- **Tx:** based on ultrasound guided fine-needle aspiration
  - Malignant = thyroidectomy
  - Benign: follow up in 6 mo
  - Bad specimen = repeat in 1-4 weeks

### Tremors

- Rest tremors: parkinson’s disease, wilson’s disease, essential tremor
- Postural and action (terminal) tremors: physiologic tremor, stress, fatigue, anxiety, emotion
  - Endocrine: hypoglycemia, thyrotoxicosis, pheochromocytoma, adrenocorticosteroids
  - Drugs/toxins: b-agonists, dopamine agonists, amphetamines, lithium, TCAs, neuroleptics, theophylline, caffeine, valproic acid, alcohol withdrawal, mercury, lead, arsenic, others
  - Charcot-marie tooth syndrome
  - Cerebellar tumor
- Kinetic (intention) tremor: disease of cerebellar outflow, MS, trauma, tumor, vascular dz, Wilson’s, drugs, toxins
- Misc. rhythmical movement disorders: psychogenic tremor, orthostatic tremor, rhythmical movements in dystonia, asterixis, clonus, nystagmus

### Dermatology

#### Basal Cell Carcinoma

- **MC type of skin cancer**
- *s/sx:* raised pearly “rolled borders,” telangiectasia, central ulcer (rodent ulcer)
- **MC presentation:** papule/nodule that may have scab or erosion + pearly papule, erythematous patch >6mm, non-healing ulcer in sun-exposed area (face, trunk, lower legs)
- Risk factor: fair-skinned individuals with hx of sun exposure
- **Dx:** shave/punch biopsy
- **Tx:** depends on type, size, location
  - Most treated surgically → photodynamic therapy, tissue scraping, electrosurgery, mohs, wide local incision

#### Burns

- **Rule of 9’s:** head, each arm, chest, abdomen, each anterior leg, each posterior leg, upper back, lower back all = 9%
  - genitals =1%
  - palm = 1% (palmar method)
- **degree involvement:**
  - 1st degree (sunburn): skin blanches with pressure, skin may be tender
  - 2nd degree (partial): skin red and blistered, skin is very tender
  - 3rd degree (full thickness): burned skin is tough / leathery; NON-TENDER
• 4th degree: into bone and muscle

• **Minor burns**: <10TBSA in adults, <5% in young/old; <2% full thickness → must NOT involve face, hands, perineum, feet, cross major joints / circumferential

• **Major burns**: >25% TBSA in adults, >20% in young/old, >10% full thickness burn → burns involve face, hands, perineum, feet, cross major joints / circumferential

• **Tx**: monitor ABCs (airway, breathing, circulation, disability, environment/exposure), fluid replacement, topical antibiotic
  - Cleanse with mild soap / water, don’t apply ice directly
  - Irrigate chemical burns with running water for 20 minutes
  - Topical antibiotic cream applied to superficial burns
  - Fingers / toes should be wrapped individually
  - Escharotomy indicated for eschar restricting airway, ventilation, causingishemia
  - DO NOT ASPIRATE BULLAE but leave in place until rupture on own then debride

• Children with >10% total body surface area and adults >15% burns need formal fluid resuscitation
  - 4mL x % body surface burned x weight in kg → ½ in 1st 8 hours and other ½ in remaining 16

• Urine output should be 0.5mL per kg weight per hour

**Cellulitis**

- definition: acute bacterial skin/skin structure infection of dermis and subcutaneous tissue
- caused by **S. aureus**, **group A strep**, or children with H.flu/strep pneumo
- s/sx: pain, erythema, warmth, swelling; flat margins, not well demarcated
- tx: antibiotics asap + culture (then follow up in 48 hours)
  - oral for mild: cephalexin or dicloxacillin
  - suspected MRSA: start empiric and culture
    - clindamycin, Bactrim, doxycycline, IV vanco/Linezolid
  - cat bite – augmentin or doxy if PCN allergy
  - puncture wound (cover for pseudomonas): ciprofloxacin
  - modify with known pathogens, underlying conditions/diseases, animal bite exposure

**Discharge**

- breast/nipple discharge
  - mastitis/breast abscess: nipple discharge, redness, warmth
  - breast CA: nipple discharge, breast discomfort, breast mass
  - gynecomastia: nipple discharge, breast enlargement, overweight
  - inflammatory breast cancer: nipple discharge, breast enlargement, redness
  - hypogonadism: nipple discharge, sexual dysfunctional, reduced sex drive

- GU:
  - Vaginal infection: yeast, BV, trichomoniasis, HPV, herpes
  - Cervix: cervicitis
  - Foreign body: tampon
  - STI: chlamydia / gonorrhea
  - Vaginal medicines / douching
  - Menopause: discharge, absence of menstruation, anxiety

- Other skin conditions: abscess = red, painful, +/- discharge

**Postoperative Drug Eruptions**

- Drug-induced exanthems: MC cutaneous reactions to drugs, responsible for ~90% of all drug rashes (abx, sulfonamides)
- Ddx: SJS, early TEN, erythema multiforme, viral exanthems, drug rash
- Dx: skin biopsy: shows necrotic epithelium; often obvious on physical exam
- Tx: stop all offending medications!!!
  - Early admission to burn unit, manage fluid/electorlytes/nutrition, airway stability, eye care
  - Ophthalmology/dermatology consult
  - IVIG
  - Steroids used to be treatment of choice but now ? increased risk sepsis

**Melanoma**
• Definition: tumor from malignant transmoration of melanocytic cells → mostly skin but can’t be on any tissue (ocular, GI, GU, lymph, leptominentes)
• MC sites: back for men, calves for women
• Likes to metastasize and spread to any sit on body – **MC tumor responsible for metastasis to the heart**
• r/f → HARRMM: hx prior melanoma, age >50yo, absence of regular dermatologist, mole changes, male gender
• Dx: BIOPSY! → excisional is preferred - with doubt biopsy full depth of dermis and extend slightly beyond edges of lesion
  - Microstaging is important part of staging / clinical management
  - Physical exam: ABCDE (asymmetry, border irregularity, color varied, diameter increasing or >6mm, elevation raised –
    doesn’t have to meet all criteria to be suspicious
• Prognosis most strongly associated with **depth** of the lesion
• Tx: excision and wide margins (stage I-III), but for stage IV systemic tx with chemotherapy
• Prevention: avoid sunburns, use sunscreen, avoid tanning beds

**Pressure Ulcers**

• Soft tissue injury from pressure between external surface + bony prominence
• Pt usually >65yo with hip fracture / ICU care; sacrum and hip = MC sites
• r/f: age >65, impaired circulation, immobilization, undernutrition, incontinence
• Dx: observation
  - Stage 1: erythema of localized area, non-blanching over bony surface (stays red)
  - Stage 2: partial loss dermal layer; pink ulceration
  - Stage 3: full dermal loss often showing subcutaneous tissue / fat
  - Stage 4: full thickness ulceration exposing bone, tendon, muscle → osteomyelitis may be present
• Tx: manage based on stage of ulcer
  - Stage 1: aggressive preventive measure, thin film dressings for protection
  - Stage 2: occlusive dressing to maintain healing, hydrocolloids, transparent films
  - Stage 3/4: debridement of necrotic tissue; exudative ulcers benefit from absorptive dressings; dry ulcers need occlusive
dressing to maintain moisture
  - Debridement: incisional with scalpel when extensive and dry; mechanical with wet-dry dressings
    - Surgical closure may be necessary; vacuum-assisted closure; negative pressure; remove necrotic debris;
    negative pressure wound therapy shows efficacy
• Prevention: reposition q2hrs

**Rash**

• Difficult to diagnose, esp if generalized
• Dx:
  - Abx side effects – fever, skin rashes, AMS
  - zinc deficiency – perioral pustular rash
  - paget disease: pruritus, well-demarcated, erythematous, eczematous rash
  - herpes zoster: classic vesicular lesions; dermalomatous distribution
  - herpes simples: clear vesicles on erythematous base, crusting
  - systemic rheumatoid disease (still’s): multiple (>5) joint involvement, fever, LAD, hepatosplenomegaly, rash,
    subcutaneous nodules, pericarditis
  - fat emboli from long bone fracture: respiratory insufficiency, coagulopathy, encephalopathy, upper body petechial rash
• tx: observe / treat empirically, diagnostic testing, refer to dermatology for workup

**Redness / Erythema**

• Causes: infection, massage, electrical treatment, acne medication, allergies, exercise, solar radiation (sunburn),
  photosensitization, cutaneous radiation syndrome, mercury toxicity, blister agents, niacin administration, waxing/tweezing,
radiotheraphy treatment
• Dx: ulcer, eczema, stasis / contact dermatitis, drug allergy, cellulitis / MRSA, chemical burns, angioedema / urticaria,
  venous insufficiency, herpes zoster, scarlet fever, tinea infections, psoriasis, acne, polycythemia vera, paronychia, osteomyelitis,
  abscess, autonomic hyperreflexia, lymphadenitis, carbuncle, furuncle, neutropenia

**Osteomyelitis**

• Infection of bone → spreads via spinal arteries
• Bones of vertebral spine = MC affected
• May originate in urinary tract and IV drug use can carry it to the spine
• frequently caused by pseudomonas (puncture wound to foot) – treat with ciprofloxacin
• caused by breakdown of coagulation factors → decreased fibrinogen / platelet count, prolonged PT and PTT, and presence of firing split products → bleeding/oozing from multiple sites

DIC

• caused by breakdown of coagulation factors → decreased fibrinogen / platelet count, prolonged PT and PTT, and presence of firing split products → bleeding/oozing from multiple sites
• dx: thrombocytopenia, high PTT, high PT, high d-dimer, decreased plasma fibrinogen
• tx: correct underlying cause
  o replace platelets, coagulation factors, fibrinogen to control severe bleeding
  o heparin in pt with slowly evolving DIC who at risk for DVT

Squamous cell carcinoma

• malignant epithelial tumor arising from epidermal keratinocytes → enlarging hyperkeratotic macule, scaly or crusted lumps
• usually from pre-existing actinic keratosis; much faster growing than BCC
• s/sx: erythematous, indurated, scaly/ulcerated papules on sun exposed skin (face, lips, ears, hands, forearms, lower legs – often tender/painful – size varies
• exam: basal cell = telangiectasia with central ulceration and rolled border vs scaly papules of SCC
• dx: biopsy
• tx: surgical excision +/- mohs, radiotherapy, cryotherapy, electrodessication and curettage
• good prognosis with immediate treatment

Urticaria

• pruritus and urticaria frequently occur during paranesesia period
• causes: anesthetic agent, widespread use of potent opioids, latex, abx, LMWH, cold compresses, underlying systemic disease
• dx: clinical
• tx: identify and stop offending agent – treat/monitor anaphylaxis urgently; antihistamines / steroids may be needed

NEUROLOGY

Change in Speech

• aphasia: inability to comprehend / form language due to damage of specific brain regions
  o usually caused by postop CVA (trouble speaking / numbness and face drooping / one arm weakness) OR head trauma
  o other causes: MS, intracerebral hemorrhage, migraine headache, carotid disease, recurrent laryngeal nerve injury (thyroidectomy), apraxia of speech
• dysarthria: motor speech disorder resulting from neurological injury of motor component or moto-speech system
  o causes:
    ▪ toxic/metabolic: wilson’s, hypoxic encephalopathy (drowning), central pontin myelinolysis
    ▪ degenerative: parkinsonism, ALS, MS, huntington’s, niemann-pick disease, friedreich’s ataxia
    ▪ others: TBI, thrombotic/embolic stroke, brain tumor, cerebral palsy, Guillain-barre, hypothermia, lyme disease, stroke, intracranial hypertension (pseudotumor cerebri), tay-sachs disease
• perioperative
  o vision loss = very rare
    ▪ ischemic optic neuropathy: caused by ischemia / poor circulation to short posterior ciliary blood vessels that supply the front, anterior, portion of optic nerve → painless abrupt reduction in vision and optic disc pallor / swelling
    ▪ visual changes: range from transient blurring of vision to irreversible blindness
      ▪ transient: usually from ocular ointments, excessive drying of cornea, corneal trauma
      ▪ complete / partial: surgical trauma, embolic events, acute anemia, hypotension
• postop
  o ocular injury / vision loss
- injury: MC = corneal abrasion (+/- associated with vision loss)
- vision loss: MC = central retinal artery occlusion, ischemic optic neuropathy, cerebral vision loss

- other causes: sickle cell, syphilis, Bechet disease, local anesthetic toxicity, general anesthesia, orbital fracture, cataract, optic neuritis, polyarteritis nodosa, myocardial infarction, migraine headache, cardiac valvular disease, hypotension, internal carotid disease, vasculitis, collagen vascular disease, sleep apnea, anemia, diabetes mellitus, herpes zoster infection, acute nagle closure glaucoma, retrobulbar hemoatoma, pituitary apoplexia, glycine induced vision loss
- r/f: long duration prone position, excessive blood loss, hypotension, anemia, hypoxia, excessive fluid replacement, use of vasoconstricting agents, elevated venous pressure, head positioning, patient-specific vascular susceptibility
- dx:
  - complete history and eye exam (pupils and opthalmoscopy, color vision and amsler grid, visual field testing)
  - labs: ESR (temporal arteritis), CRP, fasting blood glucose, VDLR, fluorescent treponemal antibody absorption, ANA

**Epidural Hematoma**

- Transient loss of consciousness from injury, then lucid period followed by increasing drowsiness, headache, and unilateral (contralateral) weakness.
- Blood in the space between dura and skull
- Cause: traumatic intracranial hemorrhage after temporal bone skull fracture → tearing of middle meningeal artery
- Dx: non-con head CT → lenticular, unilateral convexity (lens-shape) usually in temporal region – usually doesn’t cross suture lines but may cross midline
- Tx:
  - surgical craniotomy
  - medical to decreased intracerebral pressuer: mannitol, steroids/ventricular shunt, hyperventilate

**Motor and Sensory Loss**

**Spinal Cord Injuries**

**Anterior cord syndrome:**
- Loss of pain and temperature below the level with preserved joint position/vibration sense

**Central cord syndrome:**
- Loss of pain and temperature sensation at the level of the lesion, where the spinothalamic fibers cross the cord, with other modalities preserved (dissociated sensory loss)

**Complete cord transection:**
- Rostral zone of spared sensory levels (reduced sensation caudally, no sensation in levels below injury); urinary retention and bladder distention

**Brown-Sequard syndrome (hemisection of the cord):**
- Loss of joint position sense and vibration sense on the same side as lesion and pain and temperature on the opposite side a few levels below the lesion
- Lesion of half-ipsilateral cervical cord lesion
- Contralateral sensory findings: pain and temperature loss

**Neuropathies**

- Distal sensory polyneuropathy
  - Stocking-glove: sensory loss affecting distal lower and upper extremities
- Axonal: MC due to diabetes mellitus, alcohol, vitamin B12 deficiency, HIV, lyme, uremia, chemotherapy, vasculitides, amyloidosis
• Other sensory neuropathies: sjogren’s, guillain-barre, chemotherapy induced (esp platinum drugs), vitamin B6 toxicity

**Subarachnoid Hemorrhage**

• Sudden, severe (often excruciating) HA → “worse headache of my life”
• s/sx: sudden, transient LOC (~50% of pt), blood pressure rises, fever, confusion, stupor, coma, nuchal rigidity (meningeal irritation)
• herald bleed (~40% of pt) → less severe but atypical headache with focal neurologic signs – usually happens 1-3 weeks before severe SAH
• causes:
  o ruptured cerebral arterial aneurysm or AVM → bleeding into CSF in subarachnoid space
  o ruptured saccular (berry) aneurysm = ~75% nontraumatic cases with mortality rate of 50% - during 5th/6th decade of life
    ▪ r/f: smoking, HTN, hypercholesterolemia, heavy alcohol use
• dx: non-con head CT = initial
  o LP if CT is unrevealing / negative with high clinical suspicion
    ▪ Elevated opening pressure, grossly blood fluid in all four tubes
  o Cerebral angiography to evaluate entire vasculature
  o EEG may show side/site of hemorrhage
• Tx: manage hypertension, surgery to clip/wrap aneurysm, remove/embolize AVM by intra-arterial catheter

**Subdural Hematoma**

• Caused by head injury (fall, MVA, assault) → tearing of blood vessels
• Usually elderly pt with hx of multiple falls + neuro sx (chronic)
• Acute: sx within 48 hours of injury
• Subacute: 3-14 days
• Chronic: 2+ weeks (common in alcoholics / elderly)
• Dx: non-con head CT → crescent shaped, concave hyperdensity that can extend across suture line
• Tx: depends on severity
  o Small + mild sx: no tx other than observation; repeat imaging to monitor
  o Severe: surgery to reduce pressure in brain
    ▪ Burr hole trephination: hole drilled in skull and blood suctioned
    ▪ craniotomy: larger section of skull removed for better access to hematoma to recuce pressure
    ▪ craniectomy: section of skull removed for extended period of time to allow injured brain to expand / swell without permanent damage (rarely used)

**Pituitary Adenoma**

• located at sella turcica → applies pressure on optic chiasm → diminished temporal vision / bitemporal hemianopsia
• lactotrophy adenomas (prolacintomas) → hypersecretion of prolacting → amenorrhea, galactorrhea, headache
• dx: MRI, CT, eval hormone levels
• tx: 1st line = dopamine agonists (cabergoline / bromocriptine); estrogen for women with hypogonadism
  o if medical fail → consider transsphenoidal resection

**Vascular Disorders**

• carotid artery narrow (stenosis) caused by atherosclerotic plaque build up → necrotic core with weak fibrous cap → disruption of cap → thrombosis / embolization
• r/f: male gender, family hx, hyperlipidemia, smoking, HTN, diabetes
• associated conditions: PAD, coronary artery disease
• sx: transient visual disturbance (amaurosis fugax), unilateral muscle weakness or paresthesia, dizziness, tinnitus, aphasia
• physical exam: carotid bruit, motor / sensory defects
• dx:
  o duplex doppler UA = best initial test to determine level of stenosis
    ▪ >50% = moderate, >70% = severe
  o Angiography is US unavailable / unclear
• Screening: not recommended for asymptomatic pt → optimize therapy for underlying dz (HTN, hyperlipidemia)
• Tx: smoking cessation, antiplatelet therapy (aspirin / Plavix), statins for all pt
  o Operative: carotid artery revascularation for stenosis >70%
    ▪ Carotid endarterectomy (CEA) = 1st line; carotid artery stenting if can't tolerate CEA

**Transient Ischemic Attack**
• Definition: transient episode of neurologic dysfunction without acute infarction
• Low flow TIA: from stenotic atherosclerotic lesion at internal carotid artery origin → usually short lived (minutes) and recurrent
  o Sx: hand, arm, face numbness, weakness; aphasic syndromes when dominant hemisphere effected
• Dx: CT indicated for all; also MRA, carotid duplex ultrasound, transcranial doppler ultrasound
• Tx: resolve symptomatic carotid atherosclerotic dz
  o CEA (carotid endarterectomy) for symptomatic carotid stenosis 70-99% with life expectancy 5 yrs
    ▪ **Low dose aspirin should be started prior to procedure and continued for at least three months after CEA is done**
  o Recently symptomatic 70-99% stenosis → carotid artery stenting preferred if: carotid lesion not suitable for surgery, radiation-induced stenosis, cardiac/pulmonary/other disease at risk with anesthesia
• Symptomatic stenosis <50% → medical management (antithrombotic meds)
Bladder Carcinoma

- Transitional cell: MC type; 3x more common men than women
- sx: PAINLESS HEMATURIA IN A SMOKER
- dx: cystoscopy with biopsy = gold standard
- tx: endoscopic resection with cystoscopy q3mo
  - recurrent / multiple lesion treated with intravesical chemotherapy

Chronic Renal Failure (chronic kidney disease)

- Renal failure or insufficiency (stages 2-5)
- **Stage 1**: normal GFR (≥ 90 mL/min/1.73 m2) plus either persistent albuminuria or known structural or hereditary renal disease
- **Stage 2**: mild GFR 60 to 89 mL/min/1.73 m2
- **Stage 3**: moderate GFR 30 to 59 mL/min/1.73 m2
- **Stage 4**: severe GFR 15 to 29 mL/min/1.73 m2 (symptomatic stage)
- **Stage 5**: kidney failure GFR < 15 mL/min/1.73 m2
- Risk factors include: **Diabetes, hypertension**
- Independent risk factor for cardiovascular disease

Symptoms

- Abnormally elevated serum creatinine for 3+ months
- Abnormal GFR <60 mL/min for 3+ months
- Persistent proteinuria or abnormalities on renal imaging, even if GFR normal

Labs

- U/A, spot urine sample for albumin or protein: Cr ratio, SCr level
- **Broad waxy casts**
- GFR estimation GFR <60 mL/min
- Anemia, hyponatremia, hyperkalemia, hyperphosphatemia, hypocalcemia, hypermagnesemia, hyperuricemia
- **Metabolic acidosis** with high anion gap
- Renal ultrasound: symmetrically small echogenic kidneys (<8.5 cm)

Prevention:

- Aggressive glucose and BP control, low salt diet
- **Target BP**: <130/80
- Avoid nephrotoxic agents
- Dose adjust meds
- Refer for stage 3+ (GFR <30)
- ACE/ARBs

Diet and Medication

- **Protein restriction to 0.6-0.8 g/kg/day**
- Sodium: 2 g/d
- K+ restriction if hyperkalemic
- Phosp: 800-1000 mg/d
- **Kidney transplant with dialysis CKD stage 5 (GFR 5-10 mL/min)**

Prognosis:

- 80% with CKD die, from CVD mostly before getting dialysis

Dialysis

- Indications for dialysis:
• Uremic symptoms: pericarditis, encephalopathy, GI complications (anorexia, nausea, vomiting), azotemia
  • GFR <10
  • Fluid overload unresponsive to diuresis
  • Refractory hyperkalemia
  • Surgery: successful AV fistula for hemodialysis requires large vein (>5mm) close to the skin for at least 20cm
  • Cephalic vein = ideal (radial artery to cephalic vein is classic dialysis access fistula) → autogenous graft
  • No vein available → prosthetic graft (poor patency rate and higher potential for infection than autogenous graft)
  • All veins used require dilation and “arterialization” of the wall → takes at least 6 weeks prior to cannulization
  • Have to have flow rate of at least 300cc/min
  • MRI for eval and f/u of peripheral AV malformations; CTA also gives good information
• Types:
  • Hemodialysis (HD): acquired through AV fistula / prosthetic graft → 3x/week treatment (3-5 hr session each)
  • Peritoneal dialysis (PD): peritoneal membrane = dialyzer → instilled into peritoneal cavity through indwelling catheter
    ▪ Water / solutes move across capillary bed between visceral / parietal layers into dialysate – dialysate then drained and fresh dialysate instilled creating exchange
    ▪ Complications: peritonitis (nausea, vomiting, abdominal pain, diarrhea, constipation, fever)
    ▪ Can be done at home overnight

Dysuria

• After short-term single catheterization, rate of UTI = 1-5% (higher in pregnant, elderly, debilitated pt)
• Indwelling catheter → bacterial colonization, esp in women (95% after 5 days)
• Antimicrobial therapy before and after surgery to prevent bacteremia

Cystitis: Infection of the bladder and is characterized by dysuria without urethral discharge. E. coli (most common)
• sx: Dysuria, urgency, frequency, hematuria, new onset incontinence (in toilet-trained children), abdominal or suprapubic pain, absence of fever, chills, or flank pain, change in urine color/odor
• Diagnosis:
  ▪ Urine dipstick → nitrite, leukocyte esterase
  ▪ Urinalysis: pyuria, bacteriuria +/- hematuria +/- nitrites
  ▪ CBC: leukocytosis
  ▪ Blood cultures: obtain in febrile patients; consider in complicated UTI
  ▪ Urine culture (gold standard): only obtain if symptomatic
    ▪ > 100 k CFU/mL (women)
    ▪ > 1000 CFU/mL men or cath patients
    ▪ → takes 24 h to obtain results
• Treatment:
  ▪ Treat with Nitrofurantoin (not over age 65), Bactrim, or Fosfomycin
  ▪ Ciprofloxacin is reserved for complicated cases
  ▪ Postcoital UTI: single-dose TMP-SMX or cephalexin may reduce the frequency of UTI in sexually active women

Lower UTI in pregnancy
• Nitrofurantoin (Macrobid): 100 mg PO BID × 7 days
• Cephalexin (Keflex): 500 mg PO BID × 7 days
Interstitial cystitis: Symptoms relieved with voiding. Diagnosis of exclusion.
  - **Hunner's ulcer** on cystoscopy

Pyelonephritis: Dysuria + fever + flank Pain + nausea and vomiting + CVA tenderness
  - **Organism: E. coli**
  - **Dx:** Urinalysis → Bacteria and WBC casts
  - **Treatment:**
    - Outpatient: ciprofloxacin/levofloxacin +/- ceftriaxone IM
    - Inpatient: Ciprofloxacin/levofloxacin or imipenem for more severe disease

Recurrent UTI - Two uncomplicated UTI in 6 months OR 3 + uncomplicated UTIs in the previous year
  - Relapse: recurrence of UTI **within 2 wk of treatment** caused by the **same organism**
  - Refinement: recurrent UTI caused by **different bacteria**; more common than relapse
  - **Dx:** Urine culture (clean-catch midstream or straight cath)
  - **Empiric treatment**
    - Repeat culture in 1-2 Wks
    - Prophylaxis × 6 mo → once daily low-dose Bactrim, nitrofurantoin, cephalxin or ciprofloxacin
    - + Postcoital dose
    - + Contraception, topical estrogen (postmenopausal women 3x weekly), postcoital voiding, liberal fluid intake, cranberry tablets

Urethritis: Inflammation of the **urethra** caused by **infectious** or **noninfectious causes** (trauma, foreign body)
  - Most common in males age 20-24
  - **C. trachomatis, N. gonorrhoeae,** ureaplasma urealyticum, trichomonas vaginalis, Mycoplasma genitalium, HSV
  - s/sx: Urethral discharge, dysuria, itching - usually without frequency of urination
  - **Diagnosis:** **nucleic acid amplification test (NAAT)** of first voided urine
    - Gram stain of anterior urethral specimen: >5 WBC
    - Urinalysis w/ culture: (+) leuk-esterase
    - Prostate examination, to rule out bacterial prostatitis and cystitis
  - **Tx:** If no gonococci treat with azithromycin or doxycycline
    - If gonococci detected treat for both Neisseria and Chlamydia **(Ceftriaxone 250 mg IM × 1, Doxy 100 mg PO BID × 7 or Azithro 1 g PO × 1)**
  - Recurrence - Flagyl or tinidazole + azithromycin

Epididymitis: Epididymitis is characterized by dysuria, unilateral scrotal pain, and swelling
  - **The pathogen is based on patient's age and risk factors**
    - **men < 35 chlamydia and gonorrhea** → treat with doxycycline 100mg PO BID for 10 days PLUS ceftriaxone 250 mg IM × 1
      - Refer sexual partner(s) for evaluation and treatment if contact within 60 days of the onset of symptoms
    - **men > 35 E.coli** → **Levofloxacin (Levaquin) 500 mg/day PO for 10 days** / Ofloxacin 300 mg PO BID for 10 days
    - **Prehn's sign** = relief with elevation is a classic sign

Prostatitis Sudden onset of fever, chills, and low back pain combined with urinary frequency, urgency, and dysuria
  - **Men < 35: Chlamydia and Gonorrhea** - ceftriaxone and azithromycin (or Doxycycline)
  - **E coli** in men > 35 - treat with fluoroquinolones or Bactrim x 1 month
  - **Chronic prostatitis** - treat with fluoroquinolones or Bactrim x 6-12 weeks
  - If you suspect acute prostatitis do **not massage the prostate this can lead to sepsis**

Enlarged Prostate
  - Nocturia
  - Treat with alpha 1 adrenergic antagonist – Flomax

Swelling caused by fluid collection in spaces surrounding tissue/organs
  - **Peripheral:** lower legs / hands
  - Ascites: abdomen
  - Chest: pulmonary edema (lungs); pleural effusion (space surrounding lungs)
  - Lymphedema: surgical removal of lymph nodes for cancer tx (breast CA = MC) → swelling of limb / skin thickening on affected side

**Edema**
- Angioedema: reactions to medications / some inherited disorders → fluid leaks out of blood vessels into surrounding tissue
- Drugs: can be side effect (oral diabetes, anti-hypertensives, non-prescription pain relievers, estrogens)
  - **calcium channel blockers can do this**
- Infection: peritonitis
- Kidney disease → swelling to lower legs / around eyes
  - Nephrotic syndrome: peripheral / periorbital edema; sodium retention / hypoalbuminemia → fluid retention
    - Proteinuria >3.5g/day
  - Heart failure: swelling of legs, abdomen, lungs → shortness of breath
  - Cirrhosis: obstruct blood flow through liver → ascites / peripheral edema in lower legs
- MC causes: chronic venous insufficiency; complication of DVT
- Sx: “heavy legs”, itching, pain, hyperpigmentation, stasis dermatitis, thick brawny skin, increased abdomen size, trouble breathing
- Dx:
  - U/S = initial test of choice
  - Low suspicion – d-dimer
  - Color duplex ultrasound, ABI
  - Urine dipstick to r/o nephrotic syndrome
- Tx: treat underlying cause
  - Leg edema → compression stockings, elevate legs above heart 30 min 3-4x/day
  - Sodium restriction
  - With chronic without volume overload avoid diuretics
    - May enhance sodium retention through increased secretion of renin and angiotensin → AKI / oliguria
- Fluid and Electrolyte Disorders
- IV or orders re-evaluated every 4-6 hours on first day of surgery and q24hrs thereafter
- 2000-2500 5% dextrose daily = general rule
- Don’t add potassium during first 24 hours after surgery (due to increased amounts in circulation from stress and increased aldosterone activity)
- Fluid loss through NG tube <500 can be replaced by increasing infusion
  - 20mEq of potassium added to every liter of fluid if the patient has good urine flow, demonstrated deficiency
- Postop calcium in pt with thyroid / parathyroid surgery → monitor and replace
- Nephrolithiasis
- s/sx: colicky flank, groin pain, hematuria, N/V, CVA tenderness
- types:
  - calcium oxalate (most common; 75-85%): stones = radiopaque → AVOID GRAPEFRUIT JUICE (make stones worse)
  - struvite (10-15%) → stone radiopaque → chronic UTI; need chronic abx as tx
  - uric acid: stones = radiolucent; 5-8%
  - cystine: <1%; stones = radiolucent; think young boys with kidney stones
- dx:
  - noncon CT of abdomen and pelvis = gold standard → doesn’t show type of stone so give pt strainer to catch it from urine
  - urinalysis will often show microscopic hematuria
- Tx:
  - <5mm: 80% chance spontaneous passage
  - 5-10mm: 20% chance passage → may require elective lithotripsy
  - >10: not likely to pass → ureteral stent or percutaneous nephrostomy = gold standard
  - Extracorporeal shock wave lithotripsy in pt with stones >6mm or intractable pain
- Orthostatic Hypotension
- Orthostatic = postural hypotension → excessive fall in BP when an upright position is assumed
- Definition: drop of >20mm systolic bp, 10mm diastolic bp, or both → 2-5 minutes after change from supine to standing
- Dx: measure BP and heart rate after 5 min of supine and 1 and 3 min after standing (or sitting if can’t stand)
  - Hypotension without compensatory increase in HR suggests autonomic impairment; marked increase to >100bpm or by >30 beats/min suggests hypovolemia
  - ECG, electrolytes and glucose routinely checked
  - Tilt table testing when autonomic dysfunction suspected
- Tx: identify cause and treat accordingly → physical measure to reduce venous pooling, increased Na intake, sometimes steroid / midodrine
  - Causes: autonomic dysfunction, medications, postprandial (from insulin response to high carb meals / blood pooling in GI tract – worse with alcohol intake), vagal / carotid sinus hypersensitivity, hypovolemia, adrenal insufficiency
  - Drugs: alcohol, alpha blockers, anti-depressants, antihypertensives, antiparkinsonism, antipsychotic, beta-blocker, diuretic, muscle relaxers, narcotic analgesic, phosphodiesterase inhibitors, sedatives / hypnotic drugs

Renal Cell Carcinoma
- Renal clear cell = most common type (80%); transitional cell = second most common
- r/f: smoking
- s/sx: hematuria = most common presenting symptom (~60%) flank pain + abdominal pain = ~30%, palpable abdominal / renal mass
- dx: ultrasound or CT then biopsy = gold standard
- tx: surgery with radical nephrectomy = curative

Hypovolemic Shock
- decrease in circulating blood volume in intravascular system → decrease CVP pressure (amount of blood in right ventricle)
- less blood = decreased bp
- body compensates by increased pulse rate

Renal Vascular Disease
- renal artery stenosis: narrowing of one or both renal arteries → most often caused by atherosclerosis / fibromuscular dysplasia
- narrowing of renal artery can impede blood flow to target kidney → renovascular hypertension
- dx:
  - renal artery bruit on auscultation
  - HTN resistant to 3+ drugs
  - If on ACE-i and pt develops acute renal failure / sharp rise BUN:Cr, think renal artery stenosis
  - Ultrasound = initial imaging
  - Renal arteriography = gold standard
- Tx: stenting of renal arteries
  - Percutaneous transluminal angioplasty (PTA) + stent placement with surgical bypass of stenotic segment
  - Extensively infarcted kidney must be removed if revascularization not expected to result in functional recovery

Testicular Carcinoma
- MC solid tumor in young men ages 15-40 (average 32 yo); 5 year survival = 90%
- Seminoma = MC type (60%)
- r/f: history of cryptorchidism
- s/sx: firm, painless, nontender testicular mass
- dx:
  - tumor markersBHCG (+) and afp (+) in non seminoma germ cell tumors
  - scrotal ultrasound
  - other radiologic studies to look for mets (common to belly, brain, lung)
- tx:
  - orchiectomy +/- chemotherapy / radiation depending on cell type
  - orchiectomy and radiation are the most effective combination
  - seminomatous tumors = radiosensitive and can be treated with radiation therapy
  - nonseminomatous = radioresistant
  - AFP can be used to identify early relapse / monitor

Urinary Retention
- Causes:
  - Obstructive: urethral stricture, bladder calculi, neoplasm, foreign body
  - Neurogenic: MS, Parkinson, CVA, postop retention
    - Post-op urinary retention (POUR) → common after spinal / epidural anesthesia → prolonged blockage of parasym pathetic fibers that innervate the bladder with resultant urinary retention / need for urinary bladder catheter
  - Traumatic: urethral, bladder, spinal cord injury
  - Extra urinary: fecal impaction, AAA, rectal / retroperitoneal mass
Infectious: local abscess, cystitis, genital herpes, zoster

Acute urinary retention: can’t void despite full bladder
  - r/f: male gender, prostatic enlargement, epidural, spinal or prolonged anesthesia; antihistamine / narcotic use; pelvic / perineal procedures
  - sx: suprapubic discomfort with urgency / inability to void (8 hours after surgery / catheter removal), pain, vomiting, palpable bladder on exam, hypotension, bradycardia, cardia dysrhythmias
  - complication: infection, ischemia, long term bladder dysfunction
  - dx: bladder u/s 500mL of urine; post-void residual >500mL; urine culture; CBC if suspected infection
  - tx: immediate sterile catheterization for 24h then void trial; identify and treat underlying cause

chronic urinary retention: painless, develops gradually, fullness sensation, overflow incontinence, suprapubic dullness, rounded midlines mass
  - dx: postvoid residual bladder volume by cath or u/s; abdominal US or CT to identify masses, stones, hydronephrosis
  - tx: immediate sterile catheterization for 24h then void trial; identify and treat underlying cause

Detrusor sphincter dyssynergia: usually from neurological pathway (SCI / MS)
  - urethral sphincter muscle dyssynergically contracts during voiding causing flow to be interrupted and bladder pressure to rise
  - sx: daytime and nighttime wetting, hx of UTI / bladder infections; associated constipation / encopresis
  - PE: palpable bladder, bladder residual volume upon placement of foley catheter
  - Dx: post-void residual volume >150mL
  - Tx: botulinum A toxin injections; surgical incision of bladder neck (can cause incontinence)

Wilms Tumor
  - Nephroblastoma (wilms tumor); MC solid renal tumor of childhood (5% childhood cancers younger than 15)
  - Arise from kidney and seen in otherwise healthy children <4yo
  - r/f: family history, horseshoe kidney
  - associated conditions: WAGR syndrome, aniridia, genitourinary anomalies
  - s/sx: asymptomatic abdominal mass noticed by parent increasing in size; gross hematuria (rare), microscopic hematuria
  - PE: smooth/firm, well-defined mass that doesn’t cross the midline
  - ***never palpate abdomen of child with Wilms’ tumor → increases risk of rupturing encapsulated tumor → cancer cells spread to other areas of the body
  - Dx: ultrasound and CT of abdomen followed by biopsy or resection; CXR to evaluate for mets
  - Tx: surgical resection and chemo (most cases = curable)

HEMATOLOGY

Normal = Hgb 13.5 in men, 12 in women; Hct 39% in men, 37% in women

Microcytic → chronic disease, iron deficiency
  - Iron deficiency: MC anemia in the US
    - Causes: Gl bleed, PICA
    - s/s: nail spooning, weakness, fatigue, bruising/gum bleeding, sore tongue, peripheral neuropathy, balance problems, depression, dementia, glossitis, loss of vibratory / fine touch
    - labs: decreased serum iron, ferritin, transferring
      - microcytic (MCV < 80), hypochromic (decreased MCHC), low H&H
      - low ferritin <15, high TIBC, target cells
    - tx: Lifelong IM B12: 1-3 ug/d (animal products, fortified cereal) for pernicious anemia
      - IV Cyanocobalamin 1 mg IM daily × 7 d, then weekly × 4 wk, then monthly for life
      - PO B12 1-2 mg PO daily for vegans and bariatric surgery
      - Takes years to deplete B12 stores
  - Chronic disease: often coexistent with iron deficiency
    - Dx: presence of chronic infection, inflammation, cancer; can be microcytic or normocytic
      - Labs: low EPO, decreased serum iron and TIBC, normal to 3x increase serum ferritin
      - Peripheral smear: normocytic, normochromic (cancer, CKD) to microcytic hypochromic (TB, RA), decreased serum EPO levels in anemia of renal failure, increased ESR
    - Tx: Erythropoetin 50-150 U/kg IV 3 × weekly; treat the underlying disease

Lead poisoning → basophilic stippling → treatment = EDTA

Hemolytic anemias: G6PD, hereditary spherocytosis, sickle cell anemia, thalassemia
  - General:
    - CBC: increased reticulocyte count (hallmark)
- Sx: dark urine, back pain, jaundice
- Iron studies: increased serum iron, ferritin and transferrin, decreased TIBD, H&H <2x standard deviation of normal, total indirect bilirubin increased, haptoglobin decreased
- Normal = Hgb 13.5 in men, 12 in women; Hct 39% in men, 37% in women
- Peripheral smear: microcytic, normochromic anemia, spherocytes on bone marrow biopsy

- G6pd:
  - *After infection or medication* (oxidative stress) → Depletion of reduced glutathione = oxidation of Hgb = precipitation of Hgb → Heinz bodies + Bite cells → RBC destruction
  - Most commonly seen in *tropical geographic areas* prevalent for malaria (Africa, China, and Mediterranean regions)
  - Flare triggers: *Fava beans, antimalarials, sulfonamides*
  - s/sx: *mild jaundice, mild-moderate anemia, malaise, abdominal discomfort in LUQ, splenomegaly*
  - peripheral smear: *bite cells, heinz bodies*
  - tx: Avoid potentially harmful drugs, monitor infection; Acute—blood transfusion

- Hereditary Spherocytosis:
  - Inherited dysfunction / deficiency in one of the erythrocyte membrane proteins → spherocytic erythrocytes destroyed in the spleen; autosomal dominant
  - s/sx: mild jaundice, mild-moderate anemia, malaise, abdominal discomfort in LUQ, splenomegaly
  - dx: decreased MCV, increased retic count, LDH, indirect bilirubin, stool urobilinogen, low Hgb
  - peripheral smear = spherocytes, increased osmotic fragility, coombs negative
  - tx: *Splenectomy* → Indicated even when anemia compensated and asymptomatic; Delay operation until children are age 6; Cholecystectomy if gallstones present
  - Complications: *Hypoplastic crises*: follow acute viral illness → profound anemia, headache, nausea, pancytopenia, hypoplastic marrow, pigmented gallstones

- Sickle Cell: mutation in B-globin gene that changes sixth amino acid from glutamic acid to valine
  - s/sx: acute pain few hours to 2 weeks, tenderness, fever, tachycardia, anxiety
  - complications: pulmonary hypertension, ESRD, hand-foot syndrome, priapism → permanent impotence
  - labs: very high retic count + pain in African American male
    - hemoglobin S on electrophoresis
    - blood smear: sickled RBCs, Howell-jolly bodies, target cells
  - tx:
    - crisis: vigorous hydration, aggressive pain medication (morphine), nasal O2
    - severe sx: hydroxyurea (mainstay)
  - health maintenance: regular slit lamp exams (retinopathy), abx for splenectomy pt undergoing dental / invasive procedures, vigorous oral rehydration during anticipated periods of extreme exercise, exposure to hot/cold, emotional stress, infection
    - pneumococcal and Hib vaccines in early life

- *Thalassemia:*
  - B-thalassemia: only 2 B-globin genes on chromosome 11, but 4 alpha-globin genes on chromosome 16
  - Disrupted ratio between alpha and beta chains, changes stability of Hgb → hemolysis
  - s/sx: asymptomatic, mild anemia, jaundice, hepatosplenomegaly
  - labs: microcytic hypochromic, normal to increased serum iron, ferriting, transferring saturation, normal TIBC
  - peripheral smear: target cells, basophilic stippling
  - tx:
    - Symptomatic - Cardiovascular instability, continued/excessive blood loss—transfuse
    - Asymptomatic: *PO iron repletion (200 mg per day)* and B12 - only if concomitant iron deficiency anemia
    - An allogeneic bone marrow transplant
    - Folic acid 5 mg daily
    - Deferoxamine (iron chelator)
    - Splenectomy for splenomegaly
    - Poor prognostic factors: Chronic infection, liver or heart failure, die in 20-

- **Macrocytic → B12 and folic acid deficiency**
  - **B12 deficiency**
- Autoimmune destruction of gastric parietal cells → atrophic gastritis → lack of intrinsic factor production (required for absorption in small intestine); cofactor for DNA synthesis, brain/nervous system function, RBC formation
- Causes/rf: chronic alcoholism, vegetarianism, celiac, crohn’s, gastric bypass surgery, parasites
- Pernicious anemia = most common cause
- s/s: weakness, fatigue, bruising/gum bleeding, sore tongue, peripheral neuropathy, balance problems, depression, dementia, glossitis, loss of vibratory and fine touch
- labs: B12 decreased (can be false positive with folate deficiency); increased homocysteine, increased MMA, hypersegmented neutrophils
- tx: Lifelong IM B12: 1-3 ug/d (animal products, fortified cereal) for pernicious anemia
  - IV Cyanocobalamin 1 mg IM daily × 7 d, then weekly × 4 wk, then monthly for life
  - PO B12 1-2 mg PO daily for vegans and bariatric surgery
  - Years to deplete

**Folate deficiency:** cofactor for DNA synthesis, alcoholics / malnourished have smaller stores
- r/f: decreased intake, increased requirement (pregnant), sickle cell anemia, thalassemia, sprue, crohn’s, drugs
- s/sx: looks like B12 but no neurologic symptoms; neural tube defects (spina bifida)
- labs: increased homocysteine, low serum folic acid, RBC folic acid <150 (diagnostic), macro-ovalocytes and hypersegmented PMNs (pathognomonic)
- tx: PO folic acid 1-5 mg/d (first line)
  - avoid ETOH and folic acid antagonists (Bactrim, phenytoin, sulfasalazine)
  - Green leafy vegetables, yeast, legumes, fruits, animal proteins
  - Prophylactic folic acid— pregnant/lactating women, contemplating pregnancy, sickle cell patient

**Normocytic**
- Hemolytic anemias: G6PD, hereditary spherocytosis, sickle cell anemia, thalassemia
- CBC: increased reticulocyte count (hallmark)
- Sn: dark urine, back pain, jaundice
- Iron studies: increased serum iron, ferritin and transferrin, decreased TIBD, H&H <2x standard deviation of normal, total indirect bilirubin increased, haptoglobin decreased
  - Normal = Hgb 13.5 in men, 12 in women; Hct 39% in men, 37% in women
  - Peripheral smear: microcytic, normochronic anemia, spherocytes on bone marrow biopsy
- Autoimmune Hemolytic Anemia: ab bind to pt own erythrocytes → hemolysis
- Types:
  - Warm (90%): immunoglobulin IgG binds preferentially to red cells at 37C
  - Cold: immunoglobulin IgG binds preferentially to red cells < 37C
- Sn: anemia, pallor, dyspnea, acrocyanosis, livedo reticularis, mild splenomegaly
- Labs: (+) direct coombs test, increased retic, increased LDH, decreased haptoglobin, increased indirect bilirubin
- Tx: depends on disease → rituximab, steroids, cyclosporine, IVIG

**Microcytic = TICS → thalasemia, iron deficiency, chronic disease, sideroblastic**
**Normocytic = ABCD → acute blood loss, bone marrow failure/infiltration, chronic disease, destruction (hemolytic)**
**Macrocytic = FATRBC → fetus (pregnancy), alcohol, thyroid (hypothyroid), reticulocytosis, B12/folate deficiency, cirrhosis**

**Easy Bruising / Bleeding**
- Bruising without history of trauma or bruising after minor trauma not caused by anything else
- HIT, ITP, TTP, HUS, hemophilia, DIC, von willebrands
- Meds: NSAIDs, anticoagulation, antiplatelets, steroids, antidepressants (fluoxetine, sertraline, paroxetine), antibiotics (penicillins, cephalosporins)
- Nutrition:
  - Protein malnutrition: less developed countries
  - Vitamin C: severely malnourished, chronically ill + alcohol abuse, institutionalized
  - Vitamin K: bacterial overgrowth, celiac, chronic pancreatitis, IBD, alcohol abuse
- Family history: males with X-linked disorder (factor VIII or factor IX deficiency) / von Willebrand factor deficiency
- Ddx in post-op setting
  - Surgical bleeding → from major vein / artery missed during surgery
  - Medications: aspirin, clopidogrel, heparin, warfarin, antiplatelets/anticoagulation agents
  - Inherited coagulation disorders: von Willebrand, hemophilia A or B, heavy menses
  - VWD: most common heritable bleeding disorder; autosomal dominant → deficiency in VWF
    - s/sx: easy bruising, skin bleeding, prolonged bleeding, hx of epistaxis, heavy menstrual bleeding
dx: CBC, PT, aPTT< plasma VWG antigen, plasma VWF activity, factor VIII activity
labs: low VWF antigen, low VWF activity, low factor VIII
tx: desmopressin (DDAVP) or replacement with VWF concentration → heme consult

- Liver disease: reduced clotting factors
- Renal failure: uremia impairs platelet function
- DIC: sepsis, malignancy, childbirth complications → bleeding and microthrombi – diffuse bleeding from wounds and surgical sites, hematemesis, digital cyanosis, renal insufficiency, stroke

PULMONOLOGY

**Hemoptysis**
- MC causes: bronchitis (50%), tumor mass (20%), tuberculosis (8%)
- Definition: expectoration of blood – can be massive or blood tinged sputum
- MC cause = bleeding from bronchial artery (bronchitis, bronchiectasis, bronchogenic carcinoma)
- PE: examination of sputum at bedside, signs of respiratory distress (tachypnea, tachycardia, accessory muscle use, cyanosis), auscultation heart and lungs, examination of extremities for edema
- Labs: H&H, WBC for infection
- Dx: CXR = initial diagnostic study
- r/f lung malignancy: age, smoking, longer duration of hemoptysis
- tx: if <30mL → likely benign infectious with no r/f + normal XCR → manage with observation

**Lung Carcinoma**
2 major categories:
- **small cell lung CA** → 15% of cases (VERY BAD) – MC type of lung cancer that’s metastatic at the time of → worse prognosis
  - highly aggressive and almost always occurs in smokers – rapidly growing; ~80% have mets at time of diagnosis
  - rarely amenable to surgery
  - mediastinal mass / lymph nodes on one side
  - associated with smoking (99% smokers)
  - associated with ACTH and ADH → hyponatremia and hypercalcemia
  - tx: CAN’T BE TREATED WITH SURGERY – chemo only
- **non small cell** → 85% of cases – squamous cell, adenocarcinoma, large cell carcinoma
  - grows slower / more amenable to surgery
  - adenocarcinoma (35-50%) – non-smoker with incidental finding (small peripheral lesion)
  - squamous cell (25-35%): more likely smoker with hemoptysis, central bronchus solitary tumor + abnormal CXR showing large central solitary tumor
    - presentation: smoking history, central mass, hilar adenopathy, mediastinal widening
  - large cell: fast doubling rates; only ~5% respond to surgery
  - tx: CAN be treated with surgery
    - type 1-2 = surgery; stage 3 – chemo then surgery; 4 = palliative
- dx: CXR can help with diagnosis but bronchoscopy + biopsy for central lesions or fine needle transthoracic aspiration = gold
- associated syndromes:
  - superior vena cava syndrome – tumor pushes on SVC → facial / arm swelling
  - pancoasts syndrome: shoulder pain + horner syndrome (ptosis, myosis, anhidrosis) + bony destruction
  - paraneoplastic syndrome: high Ca, SIADH, anemia, DVT, Cushing’s

**Pleural Effusion**
- pathologic accumulation of fluid in pleural space (often association with pneumonia)
- transudative: TRANSIENT → fluid due to hydrostatic pressure (cirrhosis, CHF, nephrotic syndrome)
  - MC causes are heart failure, cirrhosis with ascites, hypoalbuminemia (nephrotic syndrome)
  - Right sided think transudative
- Exudative: infection, malignancy, immune
  - MC causes are pneumonia, cancer, pulmonary embolism, TB
  - Light’s criteria: if at least one of the following present, fluid = exudative
    - Basically increased protein and increased LDH = exudative
    - Pleural fluid protein/serum protein ration >0.5
    - Pleural fluid LDH/serum LDH ratio >0.6
• Pleural fluid LDH > 2/3 upper limit of lab normal serum LDH
  ▫ Left sided is likely exudative
• Dx: imaging (CXR) to confirm presence of fluid and pleural fluid analysis to help determine cause
  ▫ Lateral decubitus XR, chest CT, ultrasound if unclear whether XR density represents fluid / parenchymal infiltrates or whether suspected fluid loculated / free-flowing
• PE: decreased tactile fremitus, dullness to percussion
• Tx: thoracocentesis (drainage / indwelling catheter)

Post-Op Pneumonia

• 3rd MC complication for all procedures
• Prolongs the length of stay by a mean of 7 - 9 days
• Hospital-acquired pneumonia (pneumonia developing 48 - 72 h after admission)
• Ventilator-associated pneumonia (VAP, pneumonia developing 48 - 72 h after endotracheal intubation) occurring in the postsurgical patient
• The most important pathogens are
  ▪ Pseudomonas aeruginosa → more common with increasing length of hospital stay
  ▪ Methicillin-sensitive Staphylococcus aureus + strep pneumo + H. flu → develops within 4-7 days
  ▪ Methicillin-resistant S. aureus (MRSA) → more common with longer stay
• Risk factors: antibiotic treatment, high gastric pH (due to stress ulcer prophylaxis or therapy with H2 blockers or proton pump inhibitors), and coexisting cardiac, pulmonary, hepatic, or renal insufficiency
• Major risk factors for postoperative pneumonia are: age > 70, abdominal or thoracic surgery, functional debilitation
• Dx: Chest x-ray or chest computed tomography
  ▪ Sometimes bronchoscopy or blood culture
• Treatment includes empirically chosen antibiotics active against resistant organisms: Piperacillin/tazobactam, cefepime, levofloxacin, Imipenem, Meropenem
  ▪ In treatment settings where MRSA rates are > 20%, vancomycin or linezolid should be added

Pneumothorax

• Accumulation of air in pleural space
• Symptoms: acute onset ipsilateral chest pain / dyspnea, decreased tactile fremitus, deviated trachea, hyperresonance, diminished breath sounds, unilateral chest expansion
• Can be spontaneous (primary) → absence of underlying disease (tall, thin males ages 10-30 / marfan’s = greatest risk)
• Secondary: occurs with underlying disease – asthma, COPD, cystic fibrosis, interstitial lung disease
• Traumatic: penetrating / blunt trauma
  ▪ Tension from penetrating trauma, CPR, mechanical ventilation → mediastinal shift / tracheal deviation to contralateral side and impaired ventilation → cardio compromise → MEDICAL EMERGENCY
  ▪ If suspected, large bore needle inserted through chest wall to allow air to move out of the chest for decompression
• Dx: CXR reveals pleural air; ABG reveals hypoxemia
• Tx:
  ▪ Small (<15% diameter of hemithorax) – resolve spontaneously without need for chest tube placement
  ▪ Large (>15%) / symptomatic – chest tube placement with serial CXR q24 hrs until resolved

Hemothorax:

• Bleeding into the pleural space
• Large volume: immediate insertion of large bore thoracostomy tube → drain existing blood, quantity amount, reduce risk fibrothorax, permit apposition of pleural surfaces in attempt to reduce hemorrhage

Pulmonary Embolism

• arises from thrombi in systemic venous circulation or right side of heart, from tumors that invade venous circulation or other
• >90% from clots in deep veins of lower extremities (homan’s sign – dorsiflexion foot = calf pain)
• r/f: virchow’s triad – hypercoaguable state, trauma, venostasis (surgery, cancer, oral contraceptives, pregnancy, smoking)
• dx: spiral CT = initial method of identifying
  ▪ ABG = respiratory alkalosis secondary to hyperventilation
- EKG: S1Q3T3 (rare); non-specific ST wave changes
- CXR: Westermark sign or Hampton hump (triangular or rounded pleural base infiltrate adjacent to hilum)
- VQ scan: perfusion defects with normal ventilation (normal VQ rules out PE; abnormal – non-specific)
- D-dimer
- Pulmonary angiography = gold standard definitive

**Tx:** heparin = anticoagulant of choice or warfarin (INR 2-3)
- Duration: minimum of warfarin 3 months with reversible risk factor
- Unprovoked: warfarin recommended for at least 6 months then reevaluate
- 2 episodes unprovoked, long term with warfarin

**Shortness of Breath**
- MANY CAUSES – some to do with underlying disease; others not (exercise, altitude, tight clothing, prolonged bed rest, sedentary lifestyle)
- Disease: asthma, COPD, pneumonia, CHF, acid reflux, pneumothorax, PE, foreign body aspiration, interstitial lung disease, obesity, pulmonary hypertension, sarcoidosis, tuberculosis, anemia, cardiomyopathy, pericarditis, epiglottitis, generalized anxiety, myasthenia gravis, fractured rib, sudden blood loss

**Bronchospasm**
- Common post-op complication
- Causes: exacerbation of chronic lung condition, aspiration, histamine release from medications, trachea stimulation from secretions, suctioning, endotracheal intubation
- s/sx: dyspnea, wheezing, chest tightness, tachypnea, hypercapnia
- Tx: treat underlying cause → short acting beta-2 agonist (albuterol) + bronchodilator = 1st line therapy for asthmatic

**Weight Loss / Fatigue**
- MANY CAUSES – some to do with underlying disease; others not (dieting, exercise, malnutrition, lack of access to food, lack of sleep, heavy exertion, jetlag, large meal, aging)
- Disease: COPD, chronic fatigue syndrome, sarcoidosis, interstitial lung disease, pulmonary hypertension, tuberculosis, HIV, sleep apnea, RA, hyperthyroidism, T1DM, T2DM, clinical depression / seasonal affective disorder, anorexia, insomnia, anemia, anxiety

**OBSTETRICS / GYNECOLOGY**

**Adenopathy**
- Enlargement of lymph nodes due to gynecologic infections, malignancy, inflammation
- Most commonly seen adenopathy in breast disease is axillary adenopathy (85%)
- Other adenopathies seen: internal mammary, parasternal, supraclavicular

**Benign Breast Disease**
- Fibroadenoma = most common benign breast condition in young adolescent women (10-20%)
  - Young, reproductive age women, associated with cyclic history, breast pain resolves with menses
  - s/sx: painless, firm solitary (rubbery feeling) well-defined mobile mass; grows slowly over time and doesn’t wax/wane with menses; no axillary involvement / nipple discharge
  - dx: ultrasound / mammogram + fine-needle biopsy or excision biopsy
    - rare, but malignant neoplasms have occasionally been found in fibroadenoma
- Fibrocystic breast disease = 2nd most common
  - Lumps develop in breasts that come and go
  - No increased risk breast cancer
  - s/sx: painful, swollen, lumpy breasts bilaterally, intermittent to constant pain; well-circumscribed rubbery lumps, round, discrete, and relatively moveable
  - may increase or decrease in size with menstrual hormonal changes
  - ovarian hormones are causative agent; usually resolves with start of menstrual cycle
  - dx: breast cyst aspiration + U/S and/or mammogram; straw colored fluid with no blood
  - tx: no definitive cure; bra with extra support for pain relief; caffeine may worsen condition; monthly breast exams one week after period
    - NSAIDs, heat/ice, supportive bra, decrease caffeine/fat/chocolate
    - OCPs with low estrogenic activity and potent progestin
    - Most resolve spontaneously; +/- fine needle aspiration for removal of fluid
Breast Carcinoma

- MC cancer and 2nd MC cause of death in adult women
- Rf: 70% pt diagnosed have no risk factors
  - First degree relative with hx breast CA, obesity, ETOC, >65yo, late >17 or early <12 menarche, early menopause, increased estrogen exposure (postmenopausal HRT), BRCA1 and BRCA 2 inherited genetic mutation (lifetime risk 40-80%) but only 5-10% of women diagnosed have BRCA genes
- Late stage s/sx: bone pain, nipple retraction, breast pain, edema of arm, dimpling of skin from lymphatic obstruction (peau d’orange = poor diagnosis)
- Tumor types:
  - Infiltrating intraductal carcinoma (IIC) = most common – 80% → painless, stony hard unilateral mass. Begins as ductal carcinoma in situ
  - Infiltrating lobular = 10% → frequently bilateral
  - Inflammatory breast cancer (2%): red, swollen, warm, itchy breast often with nipple retraction and peau d’orange (NO LUMP)
  - Paget’s disease of the nipple (1%) – chronic eczematous itchy, scaling rash on the nipples and areola
  - Tumors may be estrogen receptor positive (75%), progesterone receptor positive (65%) as well as HER2 positive (25%)
- Screening:
  - Ages 40-44 have the choice to start annual mammograms (optional)
  - 45-54 should get mammograms every year
  - Women 55 and older q2years
  - ACOG recommends annual mammograms beginning at age 40 and every other year aged 50-74
  - USPSTF task force found inadequate evidence to recommend for or against screening age >75 yrs
- Dx:
  - Aspirate if cystic, mammography, U/S, breast biopsy = definitive
- Tx: segmental mastectomy (lumpectomy) followed by breast irradiation in all patients with adjunctive chemotherapy in women with positive nodes is appropriate therapy and just as effective as modified radical mastectomy for stage I and II and tumor <4cm
  - Tamoxifen in tumors that are ER-positive
  - Aromatase inhibitors in ER positive postmenopausal women
  - Monoclonal AB treatment in pt with HER2 positivity

Nipple Discharge

- MC causes of nipple discharge in non-lactating breast: duct ectasia (MC), intraductal papilloma, carcinoma
- Premenopausal women – most noticeable before menstruation and often due to fibrocystic condition – may be green/brown in color
- Milky discharge from multiple ducts more likely hyperprolactinemia
- Dx: mammogram and ultrasound
  - Serum prolactin levels, TSH (hypothyroidism)
- Tx: depends on cause – usually benign but can get proximal duct excision (tx and dx)

Pain

- Mastitis: superficial infection of breast; usually occurs during breastfeeding
- Abscess: often progression of mastitis (nursing = staph aureus)

Skin Changes

- Nonlactating: mixed infection
- Paget’s disease of the breast: scaling rash / dermatitis of the nipple caused by invasion of skin by cells from ductal carcinoma
- During pregnancy:
  - Melasma (aka chloasma = mask of pregnancy): hyperpigmentation of the face; occurs in ~75% pregnancy women
  - Also in women taking OCPs
- Vascular changes: estrogen causes vascular distention and instability and proliferation of blood vessels in pregnancy
  - Spider angioma – appear in second to fifth month of pregnancy as red lesions with branches extending out from central puncta
  - Varicosities: saphenous, vulvar, hemorrhoidal varicosities increased during pregnancy
- Striae gravidarum: stretch marks = common
- Pruritus may be physiologic, related to flare of pre-existent disorder, or pregnancy related
  - Chlorpheniramine = 1st generation antihistamine of choice for use during pregnancy
- Hirsutism: most common on face, but also can be on arms, legs, back, suprapubic
- Nails grow faster during gestation – changes that may occur include development of transverse grooves, subungual keratosis, distal onycholysis, melanichia
- Androgenic alopecia: rare, late in pregnancy, hair in frontoparietal area recedes in mild form of androgenic alopecia
- Vaginal: bluish/purplish coloration of vagina (chadwick sign) and cervix (goodell sign) = early changes in pregnancy
  - Blue appearance related to increased blood flow