Angina (ischemic heart disease)

- **History taking and physical exam:**
  - Paroxysmal chest “squeezing” or pressure, often accompanied by sensation of smothering and fear of impending death
  - Substernal chest pain often brought on by exertion (due to decreased supply and increased demand), poorly localized, nonpleuritics, exertional, could radiate to arm, lower jaw, back shoulder, short in duration, relieved with rest or nitrates, due to fixed coronary artery stenosis
  - Midsternal pain that may radiate to jaw, shoulders, arms, wrists, back of neck or some combination. Pain classically radiates to the left, but may radiate to right or bilaterally. Women tend to have right shoulder and back pain radiation more than men
  - Associated symptoms: *dyspnea, nausea, diaphoresis, numbness, fatigue*
  - Stable angina: exacerbated by physical activity and relieved by rest; usually lasts less than 3 minutes; pain relieved by sublingual NTG (can be repeated every 5 minutes up to 3 times – if doesn’t resolve, suspect UA, MI, or other)
  - Prinzmetal: vasospasm at rest, preservation of exercise capacity
  - Unstable angina: NSTEMI – suspected when pain is less responsive to NTG, lasts longer, and occurs at rest or with less exertion than previous episodes of angina – could last longer than 30 minutes – pain at rest = 90% occlusion
    - **Angina at rest (MC presentation)**
    - New onset angina symptoms
    - Increasing pattern of pain in previously stable patients
  - Chronic angina: caused by fixed stenosis (coronary plaque)
  - Look out for Levine sign

- **Diagnostic studies**
  - ECG: Horizontal / down-sloping STD; (ECG normal in 25-50% pt.) / non-specific t-wave changes (t-wave inversion), poor r wave progression
  - Physical stress test
    - Exercise stress test = most useful / cost effective – STD of 1mm = positive test – TEST OF CHOICE INITIALLY IN MOST PATIENTS WITH NORMAL RESTING ECG
    - Stress echo: localize regions of ischemia – dobutamine is used to provoke ischemia (increases myocardial oxygen demand)
  - Pharm. Stress test:
    - Radionuclide myocardial perfusion imaging (MPI): MC used to test in pts. with baseline ECG abnormalities – localizes the regions of ischemia (contraindicated in asthmatics) in pt. who can’t tolerate exercise
      - Adenosine / dipyridamole = pt. who can’t tolerate exercise
  - Other tests: myocardial perfusion scintigraphy, radionuclide angiography, stress echo, PET, SPECT, CTA, EBCT, cardiac MRI
  - **Coronary angiography (“cath”) is definitive diagnostic procedure but used selectively bc of cost and invasiveness – GOLD STANDARD**
    - Outlines coronary artery anatomy, determine location and extent of CAD indications

- **Diagnosis**

- **Health maintenance**
  - RF: CAD = MC, HTN, DM, increased age >65, tobacco use, family history CAD (CVD <55 in male relative, >65 for female relative), obesity, hyperhomocysteinemia, physical inactivity, *hyperlipidemia (decreased HDL, increased LDL), smoking*, increased alcohol intake, low intake fruits and vegetables
  - More women die of CVD than men every year
  - MetSyn: 3+ of the following: abdominal obesity, triglycerides >150, HDL <40 for men <50 for women, fasting glucose >110, HTN
➢ (“OBSE”): Obesity, BP elevated, Elevated TG, Suppressed HDL, Elevated glucose

- Clinical therapeutics
  - Preventive / rehab: exercise, weight reduction, diet low in fat and cholesterol, smoking cessation, aggressive control of diabetes, hypertension, hyperlipidemias
  - Sublingual NTG tabs or sprays / sublingual isosorbide dinitrate = primary pharmacotherapy
    - Increase oxygen and decrease coronary vasospasm – drug of choice for acute management of actue sx of anginal
      - s/e: headache, flushing, tolerance, hypotension, peripheral edema, tachyphylaxis p 24h (avoid with nitrate free period for 8 hours)
      - CI: SBP < 90, RV infarction, sildenafil / PDE-5 inhibitors
  - Long-acting nitrate should include daily 8-10 hour tx free interval to prevent drug tolerance
    - Adverse effects = headache, nausea, light-headed, hypotension
  - B-blockers prolong life in patients with coronary disease and are first line therapy for chronic angina
    - Increase prolonged filling times; reduce myocardial oxygen requirements during exercise / stress
    - ACE-I also useful (esp. pt with sx of HF)
    - CCB decrease cardiac muscle oxygen demand but are used only for pt. whom B-blockers contraindicated or maximized
      - Major role in preventing /terminating ischemia induced by coronary vasospasm by increasing coronary vasodilation, prolongs filling times
  - Platelet-inhibiting agents reduce possibility of infarction secondary to emboli and should be used in all pt. unless contraindication exists (aspirin / Plavix)
    - Prevent platelet activation / aggregation (decrease thromboxane A2)
  - Ranolazine prolongs exercise duration and time to angina and is useful for sx control
  - Classic regimen: daily aspirin, sublingual NTG prn pain, daily beta blocker, statin (if increased LDL)
  - Unstable angina:
    - Aspirin – prevents platelet activation / aggregation
    - Unfractionated heparin – binds to and potentiates antithrombin III’s ability to inactivate factor Xa, inactivates thrombin (factor IIa); give if hx of ACS, EKG changes, or positive cardiac markers
    - Low molecular weight heparin: binds to and potentiates antithrombin III’s ability inactivate factor Xa; give if hx of ACS, EKG changes, + cardiac markers; side effects: thrombocytopenia
    - ADP inhibitors (clopigidreg - plavix): useful in initial treatment of ACS in pt. with aspirin allergy; inhibits ADP-mediated platelet aggregation
    - GPIIb/IIIa inhibitors – inhibits final pathway for platelet aggregation
    - Factor Xa inhibitor: binds to and enhances antithrombin (no direct effect on thrombin)
    - Adjunct therapy:
      - B-blockers: lower myocardial O2 consumption + antiarrhythmic effects; CI with severe bradycardia,; hypotension, decompensated CHF, 2nd/3rd heart block, cocaine induced MI
      - Nitrates: decrease anginal sx but not mortality
      - Morphine: vasodilation = decrease preload, relieve pain, decrease anxiety
      - CCB (verapamil, diltiazem): use 3rd line if can’t tolerate B-blocker
        - Treatment of choice in vasospastic disorders (prinzmetals + cocaine use)
  - Scientific concepts (underlying pathways)
    - Insufficient oxygen supply to cardiac muscle, most commonly caused by atherosclerotic narrowing and less commonly by constriction of coronary arteries
    - Cocaine associated with myocardial ischemia and infarction secondary to vasospasm (usually younger patients)
    - Etiology: CAD = MC, coronary artery spasm, AS/Al, severe systemic or pulmonary HTN, hypertrophic cardiomyopathy
    - Pharm. Therapy = increase supply and decrease demand
**Arrhythmias**

- **Clinical significance** depends on how much it impairs cardiac output / how likely it is to deteriorate into more serious disturbance
- **Presentation ranges**: asymptomatic → hemodynamic instability, shock, death
- **Unstable**: chest pain, dyspnea, altered mental status, hypotension
- **Diagnostic studies**: ECG monitoring, event recording, measurement of HR variability, signal-averaged ECG, exercise stress testing, electrophysiologic testing, autonomic testing
- **Tx**: antiarrhythmic drugs
  - Stable = treated with medications; unstable = treat with electricity

### Supraventricular arrhythmias

- **Types**:
  - **Sinus brady**: <60 – well conditioned athletes / sinus node pathology with increased risk ectopic rhythm
    - Unstable – give vagolytic (atropine) or positive chronotropic (epinephrine, dopamine)
    - Transcutaneous / transvenous pacing indicated / may need permanent pacing
  - **Sinus tach**: >100 – fever, exercise, pain, emotion, shock, thyrotoxicosis, anemia, HF, drugs – causes symptoms when >150
    - Unstable – synchronized cardioversion
    - Medications: amiodarone, B-blocker (esmolol) / procainamide may be indicated
    - Regular narrow complex tachy usually represents AV nodal reentry tachy such as PSVT
      - 1. Stable pt. = Valsalva / carotid sinus massage 2. Rapid IV adenosine push 3. B-blocker / CCB can be used if adenosine doesn’t work
      - Sustained / recurrent PSVT → refer for catheter ablative therapy
  - **Atrial premature beats** – benign (no treatment in absence of symptoms)
  - **Paroxysmal supraventricular tachy**: most common of the paroxysmals, pt. complains of “racing heart”
  - **Afib (“irregularly irregular”)**: MC chronic arrhythmia – can lead to decrease in CO and MC cause of embolic cerebrovascular accidents – called “holiday heart” when caused by excessive alcohol use or withdrawal
    - Tx depends on presentation: 1. Maintain normal ventricular rate to decrease sx and restore sinus rhythm – cardiovert with 100-200J if unstable
    - Of stable pt.: anticoag (heparin / enoxaparin, warfarin, dabigatran) if >48hr hx and rate control 3-4 weeks before trying conversion
      - No thrombus and low risk thrombus formation can be treated with cardioversion once anticoagulation with heparin is established
    - Rate control in presence of HF: digoxin, amiodarone, dronedarone
    - No HF: metoprolol / esmolol or diltiazem / verapamil
  - **Atrial flutter**: usually in pt. with COPD, HF, septal defects, CAD
    - Cardioversion with 50J = primary option in pt. with hemodynamic or symptomatic instability
    - Stable: anticoagulants + rate control (beta-blocker)
    - Sustained / recurrent: refer for catheter ablative surgery after anticoag w/ warfarin
    - If antiarrhythmic therapy is chosen for chronic a flutter, dofetilide is primary choice
  - **Junctional rhythms**: normal hearts, myocarditis, CAD, dig toxicity
    - Clinical features: palpitations, angina, fatigue, HF sx, asymptomatic
    - Diagnostic studies: ECG
    - Tx options:
      - Significant brady / tachy: continuous cardiac monitoring, BP monitoring, pulse ox, IV, oxygen if hypoxic

### Ventricular Rhythms
Aka premature ventricular complexes (PVCs) – common and benign; occur with increasing frequency if myocardium is irritated by factors such as ischemia / electrolyte disturbance
- May be asymptomatic or aware of it
- V tach: 3+ consecutive ventricular premature beats; can be sustained or unsustained (both associated with electrolyte abnormalities); can be stable or unstable and can present without a pulse; frequent complication of acute MI / dilated cardiomyopathy
  - Can be asymptomatic or complain of palpitations, dizziness, syncope, sudden death
  - Tx:
    - With severe hypotension / loss of consciousness – synchronized cardioversion
    - Pulseless = immediate defibrillation + CPR
    - Pharm: amiodarone, lidocaine, procainamide (in that order)

Torsades de pointes (polymorphic V tach): QRS twists around baseline – can occur spontaneously or when pt. has hypokalemia / hypomagnesemia or from drugs that prolong the QT
- Tx: IV magnesium, correct electrolyte abnormalities, withdraw drugs
- Isoproterenol infusion and overdrive pacing may be indicated after initial therapy / permanent pacemaker if it’s recurrent

Long QT syndrome: congenital or acquired / associated with recurrent syncope, QT interval usually .5-.7sec long, ventricular arrhythmias, sudden death
- Tx: treat electrolyte abnormalities and discontinue drugs

Brugada syndrome: genetic – syncope, ventricular fib, sudden death, often during sleep (more common Asian men)

V fib: no effective pumping action exisits, without intervention = death

Diagnostic studies: ECG

Treatment:
- Ventricular premature beats / unsustained V tach without heart disease / electrolyte abnormalities are usually not treated – may be treated with B-blockers / CCB if pt. is symptomatic
- Those with identifiable site of arrhythmic origin benefit from radiofrequency ablation
- ICD may be indicated for recurrent sustained V tach with structural heart disease or without reversible cause for congenital long QT syndrome / brugada

Conduction Disturbances

Sick sinus syndrome: physiologically inappropriate sinus brady, sinus pause, sinus arrest or episodes of alternating sinus tach + brady – usually found in elderly / infants who had heart surgery
- Can be caused by digitalis, CCBs, B-blockers, sympatholytic agents, antiarrhythmic drugs, aerosol propellant abuse
- Reversible if caused by digitalis, quinidine, B-blockers, aerosol propellants
- Usually asymptomatic, but may have syncope, dizziness, confusion, HF, palpitations, decreased exercise tolerance
- Most require permanent pacing if symptomatic

AV block: refractory conduction of impulses from atria to ventricles thorugh AV node / bundle of His and divided into first degree, second degree, and complete / third degree
- First degree: PR interval >.2 sec
  - usually asymptomatic; higher grade may produce weakness, fatigue, decreased exercise tolerance
  - no treatment
- Second degree: aka wenckebach - not all atrial beats conducted to ventricles
  - Mobitz 1: progressive lengthening PR interval with shortening RR interval until atrial impulse eventually doesn’t get conducted to ventricles (long, longer, drop now you have Wenckebach)
    - Permanent cardiac pacing is only treatment
  - Mobitz 2: intermittently nonconducted atrial beats – block within His bundle system – almost always secondary to organic disease involving infranodal system and may progress to complete heart block
    - Permanent cardiac pacing is only treatment
    - Temp transthoracic or transvenous pacing should be followed by permanent pacing
Third degree: complete heart block – complete dissociation between atria and ventricles caused by lesion distal to His bundle
  • Temp transthoracic or transvenous pacing should be followed by permanent pacing

Diagnostic studies: ECG changes

Chest pain

Key physical exam: vital signs + BP in both arms; complete cardiovascular exam (JVD, PMI, chest wall tenderness to palpation, heart sounds, pulses, edema); lung and abdominal exams; lower extremity exam (inspection for signs of DVT)

Cardiac

Myocardial infarction:
  ▪ Hx: hypertension, hyperlipidemia, smoking
  ▪ Sx: sudden onset substernal heavy chest pain, radiation to left arm, dyspnea, diaphoresis, nausea
  ▪ Labs: ECG, CPK-MB, troponin x3, CXR, CBC, electrolytes, helical CT, echo, cardiac catheterization

Angina: ECG changes
  ▪ Sx: retrosternal squeezing pain that lasts for 2 minutes and occurs with exercise; relieved with rest; not related to food intake
  ▪ Labs: ECG, CPK-MB, CXR, CBC, electrolytes, exercise stress test (easiest / most affordable), upper endoscopy / pH monitoring, cardiac catheterization (coronary angiography = gold standard)

Aortic dissection
  ▪ Hx: uncontrolled hypertension
  ▪ Sx: sudden onset severe chest pain that radiates to back
  ▪ Labs: TTE, ECG, CPK-MB, troponin, CXR, CBC, amylase, lipase, CTA (chest with contrast), MRI/MRA (aorta), aortic angiography, upper endoscopy

Pericarditis
  ▪ Hx: viral infection
  ▪ Sx: retrosternal stabbing, chest pain that improves when leaning forward, worsens with deep inspiration
  ▪ Labs: ECG, CPK-MB, troponin, CXR, echo, CBC, upper endoscopy, ESR

CHF
  ▪ Sx: cough exacerbated by lying down at night and improved by propping with pillows, exertional dyspnea
  ▪ Labs: CBC, CXR, ECG, echo, PFTs, BNP, CT-chest

Lung cancer
  ▪ Hx: heavy smoker
  ▪ sx: 6 mo worsening cough, hemoptysis, dyspnea, weakness, weight loss
  ▪ labs: CBC, sputum gram stain, culture, cytology, CXR, CT-chest

asthma:
  ▪ sx: SOB, cough, wheezing worse in cold air
  ▪ labs: CBC, CXR, peak flow mausrement, PFTs

Pulmonary

Costochondritis
  ▪ Hx: viral infection
  ▪ Sx: stabbing chest pain that worsens with deep inspiration, relieved by aspirin
  ▪ Exam: chest wall tenderness
  ▪ Labs: ECG, CXR, CPK-MB, troponin, CBC

Pneumonia
  ▪ Hx: heavy smoker + COPD
  ▪ sx: 1 week pleuritic chest pain, fever, chills, cough with purulent yellow sputum
Pulmonary embolism
- Hx: recent immobilization (e.g. surgery)
- Sx: acute onset SOB at rest and pleuritic chest pain, tachycardia, hypotension, tachypnea, mild fever
- Labs: d-dimer, CTA – chest with IV contrast, CXR, ECG, ABG, CPK-MB, troponin, CBC, electrolytes, BUN/Cr, glucose, doppler U/S (legs)

Pneumothorax

COPD exacerbation (bronchitis)
- Hx: COPD, smoker
- Sx: increased sypnea and sputum production
- Labs: CBC, CXR, ABG, PFTs, sputum gram stain and cluture, CT-chest, echo

TB
- Hx: contact with TB pts, healthcare workers, traveling
- Sx: worsening cough of 6 weeks, weight loss, fatigue, night sweats, fever
- Labs: CBC, PPD/quantiferon-TB gold, sputum gram stain, acid fast stain and culture, CXR, CT-chest, bronchoscopy, HIV antibody, lymph node biopsy

Pulmonary edema:
- Sx: worsening dyspnea of 6 hours + cough with pink, frothy sputum
- Labs: CXR, ECG, CBC, ABG, PFTs, BNP

GI
- GERD
  - Sx: retrosternal burning sensation that occurs after heavy meals and when lying down; relieved by antacids
  - Labs: ECG, barium swallow, upper endoscopy, esophageal pH monitoring, H. pylori stool antigen

Other
- Sickle cell disease – acute chest syndrome
  - Hx: African American pt. with hx of sickle cell disease
  - Sx: acute onset severe chest pain with hx of sickle cell disease
  - Labs: CBC with retic count and peripheral smear, LDH, ABG, d-dimer, CXR, CPK-MB, troponin, ECG, CTA – chest with IV contrast

History taking and physical exam:
- Definition: Inability of heart to pump sufficient blood to meet the metabolic demands of the body at normal filling pressures
- MC cause is coronary artery disease
- Left-sided failure: increased pulmonary venous pressure from fluid backing up into lungs
  - exertional pulmonary vascular congestion leading to exertional dyspnea plus cough, fatigue, orthopnea, paroxysmal nocturnal dyspnea, basilar rales, gallops, and exercise intolerance
  - dyspnea = MC symptom (initially exertional ⇒ orthopnea) + paroxysmal nocturnal dyspnea, pulmonary congestion / edema (rales, ronchia, chronic nonproductive cough (commonly missed), pink frothy sputum (surfactant), wheezing due to airway edema, pleural effusion
  - CHF = MC cause of transudative pleural effusion
  - Physical exam: HTN, tachypnea, Cheyne Stoke’s breathing (deeper faster breathing with gradual decrease and periods of apnea), cyanosis, S3 (systolic), S4 (diastolic)
  - Increased adrenergic activation: dusky, pale skin, diaphoresis, sinus tach, cool extremities (due to poor perfusion / peripheral arterial vasoconstriction), fatigue, AMS
- MC cause L sided is CAD and HTN
- **Right-sided failure** - due to increased systemic venous pressure → signs of systemic fluid retention; causes systemic vascular congestion
  - Peripheral edema (pitting edema in legs), JVD (increased JVP), GI / hepatic congestions: anorexia, n/v due to edema of GI tract, hepatosplenomegaly, RUQ tenderness, hepatojugular reflex
  - Distended neck veins, tender or nontender hepatic congestion, decreased appetite/nausea, dependent pitting edema; most frequently caused by left-sided heart failure; predominant features = peripheral edema and hepatomegaly
  - MC cause R sided is L sided

- Systolic: decreased EF associated with **S3 gallop (S3 = filling of dilated ventricle) → MC form of CHF**
- Diastolic: normal/increased EF associated with S4 gallop (S4 = atrial contraction into stiff ventricle); HTN
  - High v.low output:
    - High: anemia, thyrotoxicosis, beriberi, paget’s dz of bone
    - Low: HTN, CAD

- Cardiac signs: parasternal lift; enlarged, displaced, or hyperdynamic apical impulse; diminished first heart sound; S3 gallop; S4 heard in diastolic failure
- Sympathetic activity produces pallor and cold, clammy skin
- Nocturia = common symptom
- Hypotension and narrow pulse pressure are frequently present; dependent on etiology of HF, BP can be low, elevated, normal
- Four classes of HF defined by degree of limitation of daily activity
  - I: no limitation of physical activity; ordinary physical activity does not cause undue fatigue, dyspnea, or anginal pain
  - II: slight limitation of physical activity; ordinary physical activity results in symptoms
  - III: marked limitation of physical activity; comfortable at rest, less than ordinary activity causes symptoms
  - IV: unable to engage in any physical activity without discomfort; symptoms may be present even at rest

- Diagnostic studies:
  - CXR (especially useful for CHF): kerley b lines, butter fly (batwing pattern), cephalization of vessels, peribronchial cuffing, perihilar congestion, cardiomegaly, bilateral or right sided pulmonary effusions, perivascular or interstitial edema, venous dilation and cephalization, alveolar fluid, pleural effusions → pulmonary edema
  - Anemia, renal insufficiency, hyperkalemia, hyponatremia, elevated liver enzymes (on diuretics may develop hypokalemia)
  - ECG: non-specific changes (low voltage), underlying arrhythmia, intraventricular conduction defects, left ventricular hypertrophy, non-specific repolarization changes, new/old MI

- **ECHOCARDIOGRAPHY:** most useful imaging study bc can assess size and function of chambers, valve abnormalities, pericardial effusion, shunting, segmental wall abnormalities; used to determine and monitor ejection fraction → KEY DIAGNOSTIC AND PROGNOSTIC INDICATOR
  - Ejection fraction most important determinant in prognosis
    - Normal EF = 55-60 (<35 = increased mortality → cardio defib placed)
    - Systolic: decreased EF, thin ventricular walls, dilated LV chamber, +S3
    - Diastolic: normal/increased EF, thick ventricular walls, small LV chamber, +S4
  - BNP (SERUM B-TYPE NATRIURETIC PEPTIDE) or N-terminal pro-BNP is usually elevated; cardiac markers (CK-MB and troponins) should also be tested to eval for new MI
    - May identify CHF as cause for dyspnea in ER; indicated severity and prognosis
    - Ventricles release B-type natriuretic peptides during volume overload in attempt to reverse process (causing decreased RAAS activation, decreased TBFV, increased NA excretion)
      - >100 = CHF is likely
  - Stress imaging / radionuclide angiography may be indicated to assess cause / severity
Cardiac cath: indicated if atherosclerosis is suspected – can visualize the ventricle and determine CO

Older pt. should undergo thyroid function testing; iron studies indicated in suspected cases of HF caused by hemochromatosis

Health Maintenance:
- Key management is recognize and treat reversible causes of JF
- Preventative and rehab nonpharm: aerobic exercise, low-sodium diet, tobacco cessation, alcohol cessation, stress reduction
- NA restriction <2g/day; fluid restriction <2L/day, exercise, smoking cessation

Clinical Therapeutics:
- Initial therapy: early initiation ACE-I (decrease left ventricular wall stress and slow myocardial remodeling and fibrosis; decrease preload / afterload, decrease aldosterone production; potentiates other vasodilators; increases exercise tolerance) AND a diuretic
  - Vasodilators: (decrease afterload)
    - ACE-I = first line treatment: decrease mortality, decrease rehospitalization, directly reverses pathology by decreasing renin and sympathetic stimulation, decreases remodeling
      - s/e: first dose hypotension, azotemia / renal insufficiency, hyperkalemia, cough and angioedema due to increased bradykinin
      - c/i: hypotension, pregnancy
    - If ACE-I / b-blocker not tolerated, ARB can be used (blocks effects of ang. II) – losartan, valsartan, candesartan, irbesartan
    - B-blocker may also be useful to improve ejection fraction, reduce LV dilation, reduce incidence of dysrhythmia
      - Decrease morality (increase EF, reduce ventricular size) – don’t use in decompensated CHF
  - Hydralazine + nitrates combined: if can’t tolerate ACEI or BB (not as good as ACE-I), vasodilator with decreased mortality; hydralazine = safe in pregnancy; good in African-americans; NTG decreases preload + hydralazine decreases afterload; s/e = dizziness, headache, tachyphylaxis (8th nitrate-free period to prevent)
  - Aldosterone receptor antagonists helpful in combination with ARB and B-adrenergic antagonists
  - Decrease preload:
    - Loop diuretics: most effective tx for sx for pts with mild-moderate CHF, INHIBIT WATER TRANSPORT ACROSS LOOP OF HENLE = INCREASED EXCRETION OF WATER, CL, NA, K, S/E: HYPOKALEMIA, CALCEMIA,NATREMIA, HYPERGLYCEMIA, HYPERURICEMIA, SULFA SALLERGIES
    - Potassium sparing diuretics: weak diuretic, most useful in combo with loop diuretics, decrease morality in HF< s/e: hyperkalemia, gynecomastia, CI: renal failure
    - HCTZ: s/e: hyponatremia, hyperuricemia, hyperglycemia
  - Positive inotropes:
    - Sympathomimetics (digoxin): used sort term in severe acute CH, good relief in pt with afib, decreased hospitalization but no mortality benefit with digoxin; inotropic = prolong intracellular calcium/contraction via N/K pump inhibition; negative chronotropic / dromotrope – slow conduction velocity
      - s/e: narrow TI – arrhythmias, seizures, dizziness, anorexia, n/v/d, double/blurry vision green/yellow hallows around lights, gynecomastia
      - dig toxicity: digitalis effect on ECG, downsloping sagging ST segment ,junctional rhythms, hypokalemia worsens toxicity, give antidote digoxin immune fab.
  - As symptoms progress: right sided HF with fluid retention, sodium retention, edema may develop
    - Use thiazide / loop diuretic
  - CCB (amlodipine) used to treat associated with angina or HTN
  - Ivabradine: slows progression (inhibits If channel in the sinus node)
  - ICDs indicated when EF <35% / biventricular pacing when QRS is prolonged
  - Coronary revascularization indicated with reversible ischemia
**LMNOP: Lasix, morphine, nitrates (venodilators), oxygen, position (place upright to decrease venous return) – Lasix removes fluids helping symptoms, morphine and nitrates reduce preload (reduce heart strain)**

- Diastolic heart failure: b-blockers, ACE-I, calcium channel blockers (heart rate and bp control and relief of ischemia diuretics for volume overload)
  - helpful for diastolic heart failure (unlike systolic failure where it is generally not helpful and even harmful)
- meds that decrease mortality: ACE-I, ARB’s, B-blockers, nitrates + hydralazine, spironoloactone
  - CCB not used in systolic HF except with angiong + HF or normal EF
- HF outpatient regimen: **ACE + diuretic initially ⇒ +/- add b-blockers**
- In severe cases refractory to therapy, mechanical support with ventricular device (implantable cardioverter defibrillator in pts with EF <35%) or intra-aortic balloon pump may be used as bridge to cardiac transplant

**Scientific Concepts:**

- Initial insult leads to increased afterload, increased preload, decreased contractility
- Acute decompensated heart failure with worsening of baseline symptoms characterized by pulmonary congestion (worsening dyspnea, rales, pink frothy sputum etc), sympathetic activation or CXR findings of congestion
- Characterized by abnormal retention of water and sodium resulting in venous congestion ⇒ dyspnea / edema
- Pathologic changes in one or more of the following: myocardial contractility, structural integrity of valves, preload or afterload of the ventricle and heart rate
- May be right sided, left sided, both / associated with systolic and/or diastolic cardiac dysfunction

**Coronary Artery Disease**

- atherosclerosis is a chronic inflammatory response to lipid accumulation in the arterial wall
- Initially clinically silent intimal plaques (could be there for years/decades)
- Fissuring or erosion of atherosclerotic plaques (exposes junk underneath) triggers thrombus formation (accumulates over seconds/minutes causing acute ischemia of end organ)
- Impact of atherosclerosis: ~90% of cases of MI/CAD
  - **CAD is #1 killer** in USA and worldwide ⇒ Death rates ↓ yearly since 1968 – MC cause of cardiovascular death and disability
  - Impact of atherosclerosis: majority of heart failure, ~60% of strokes (from atherosclerosis), ~1/3 of all cases of dementia (vascular dementia)
  - affects heart, brain, peripheral vasculature esp in legs (not in arms)

**History and physical exam:**

- risk Factors - Framingham Heart Study: Identified major CV risk factors: smoking, diabetes, dyslipidemia (↑ LDL, ↓ HDL), hypertension, family history of early CAD, men <55, women <65
- men = 4x more often than women; by age 70, ratio is 1:1
- non-modifiable: male gender, age
- modifiable: hypertriglyceridemia, lipoprotein-a (AKA “Lp little a”) – subset of cholesterol, hyperfibrinogenemia, C-reactive protein (CRP), physical inactivity
- Depends on location of vessels – cerebral occlusions (neuro deficits), renal artery blockage (kidney failure), coronary blockage (myocardial ischemia, angina / infarction)

**Diagnostic studies:**

- Markers of inflammation: high-sensitivity CRP (hsCRP) (elevated)

**Health Maintenance: Major strategies = control risk factors**
- **Smoking cessation**
- ▼ LDL (dietary & pharmacologic)
- Management of blood pressure
- Hyperglycemic control
- Improving low HDL levels (exercise)
- ▼ hypertriglyceridemia
- Obesity reduction: encourage BMI <25kg/m² and waist circumference <40 (<35 in women), regular aerobic exercise
- Diet: low-saturated fat, low trans fat, low cholesterol, high in fiber, rich in veg, fruit, whole grain
- Physical inactivity (start exercising!)

  - **Clinical Therapeutics:**
    - “Primary prevention” = platelet inhibitors (Aspirin, etc.)
      - Anti-platelet agents are CORNERSTONE of coronary disease – they are cheap and available!
    - “Secondary prevention”
      - Platelet inhibitors (Aspirin, etc.)
      - β-blockers
      - ACE inhibitors or angiotensin II antagonists
      - Statins: reduce atherosclerotic lesions, inhibit their progression, improve endothelial function, both primary & secondary prevention in high risk patients
    - Nitroglycerin: used in symptomatic patients
      - reduces SYMPTOMS only by causing coronary vasodilatation - mimics endogenous nitric oxide

  - **Scientific Concepts:**
    - LDL moves from blood into intima ▸ accumulate under endothelial cell layer
    - LDL particles taken up by macrophages & become foam cells ▸ fatty streak
    - Fibroblasts form collagen cap over lipid pool
    - Stable lesion ▸ thickens (possibly restricting flow)
    - Unstable lesion ▸ fissures develop in endothelium exposing (very thrombogenic) lipids to circulation, triggering thrombus formation and acute complete/partial occlusion of vessel
    - Atherosclerosis Timeline
      - Foam cells are macrophages that gobble up lipids in the wall; it then dies off and stays there and becomes a foam cell; when it dies it releases cytokines that attract more macrophages to the area
        - happens by like age 10^8
      - Fibrous plaque forms over lipid core
        - Complete clot – ST elevation MI
        - Incomplete clot – unstable angina or non-ST elevation MI
Vulnerable plaque is easy to rupture; thick plaque is stable.

- Adhesion, activation, aggregation, propagation of clot, platelet adherence
- High magnification of a thrombus showing a mixture of RBCs & platelets incorporated into fibrin meshwork

**Endocarditis**

- Occurs through direct inoculation at surgery, in injection drug users, or late-stage HIV disease
- Infection of endothelium / valves secondary to colonization during transient / persistent bacteremia
- **Mitral = MC valve involved; M>A>T>P**

**History and physical exam:**
- Fever (80-90% - including FUO), ECG conduction abnormalities, anorexia, weight loss
- Peripheral manifestations:
  - Janeway lesions: painless erythematous macules on palms/soles (emboli/immune)
  - Roth spots (retinal hemorrhage with pale center)
  - Osler nodes: tender nodules on pads of digits
  - Splinter hemorrhages of proximal nail bed, clubbing, hepatosplenomegaly, petechiae
  - Septic emboli: CNS, kidneys, spleen, joints

**Diagnostic studies:**
- **Blood cultures (before ABX initiation) – 3 sets at least 1 hour apart**
- EKG: at regular intervals to assess for new conduction abnormalities – prone to arrhythmias
- Echo: obtain TTE first; consider TEE if TTE nondiagnostic / increased suspicion
  - TEE = more sensitive than TTE
- Labs: CBC → leukocytosis, anemia (normocytic, normochromic), increased ESR/RF
- **Diagnosis: 2 major OR 1 major + 3 minor OR 5 minor (80% accuracy)**
- Modified duke criteria:
  - Major:
    - 1. Sustained bacteremia (2 positive blood cultures)
    - 2. Endocardial involvement: a. **positive echo** showing vegetations / abscess OR b. clearly established new valvular regurg (AR/MR)
Minor:
• 1. Predisposing condition (IVDU, indwelling cath)
• 2. Fever (>38C / 100.4F)
• 3. Vascular / embolic phenomena: janeway lesions, septic arterial or pulmonary embolic, ICH
• 4. Immunologic phenomena: osler’s nodes, roth spots, positive RF, acute glomerulonephritis
• 5. Positive blood culture not meeting major criteria
• 6. Positive echo not meeting major criteria (ex. Worsening murmur)

Health maintenance:
Endocarditis prophylaxis:
- Cardiac conditions: prosthetic (artificial) heart valves, heart repairs using prosthetic material (not including stents), prior hx endocarditis, congenital heart disease
- Dental: manipulation of gums, roots of the teeth, oral mucosa perforation
- Respiratory: surgery on respiratory mucosa, rigid bronchoscopy
- Procedures involving infected skin / MSK tissues (including abscess I&D)
- Regimens: amoxicillin 2g 30-60m before (clindamycin 600mg if penicillin allergy)
- ***prophylaxis no longer routinely recommended for GI / GU / valvular heart dz pt
- Good oral hygiene recommended to reduce temporary episodes of bacteremia

Clinical Therapeutics:
- Medical management: obtain culture data first then start abx promptly after (abx may be delayed to obtain blood culture data) then adjust abx based on organism and sensitivities – duration of therapy = 4-6 weeks
  - Suggested empiric therapy:
    - native valve subacute bacterial endocarditis:
      - penicillin/ampicillin + gentamicin
      - vancomycin in IVDU
    - prosthetic valve:
      - vanco + gentamicin + rifampin (for s.aureus)
      - fungal: amphotericin B, caspofungin if severe + valve surgery / replacement (treat 6-8 weeks)

Indications for surgery:
- Refractory CHF, persistent or refractory infection, invasive infection, prosthetic valve, recurrent systemic emboli, fungal infection
- When recovered, pt. typically receives life-long fluconazole

Scientific Concepts:
- ~50% cases involve nonalbicans candida spp. And are resistant to treatment → cause lg. vegetations
- Enterococci = MC in men 50 y with hx of GI/GU procedures
- HACEK: haemophilus, actinobacillus, cardiobacterium, elkenella, klingella → assoc. with lg. vegetations
  1. Acute bacterial endocarditis: infection of normal valves with virulent organism (s. aureus)
  2. Subacute Bacterial Endocarditis: indolent infection of abnormal valves with less virulent organism (s. viridans) – oral flora = source of infection
  3. Endocarditis w IVDA: MRSA (s.aureus), pseudomonas, candida – esp. HIV patients
  4. Prosthetic Valve endocarditis: early (after 60 days) – staph epidermis = MC, p 60 days resembles subacute

Hyperlipidemia
Excess of lipids (including cholesterol and triglycerides) in the blood
Hypercholesterolemia; hypothyroidism, pregnancy, kidney failure
Hypertriglyceridemia: DM, ETOH, obesity, steroids, estrogen

History and Physical exam:
- Most pt. are asymptomatic
- May develop xanthomas (especially in eyelids, achilles tendon) → usually indicate genetic cause
  - Nearly 2/3 with xanththelasmas (eyelids) have normal lipid profiles
- Hypertriglyceridemia may cause pancreatitis
- Severe hypercholesterolemia may develop premature arcus senilis; lipemia retinalis (cream-colored retinal vessels) with triglyceride levels >2,000

Diagnostic Studies:
Evidence of CVD / CHD = fasting complete lipid profile
- Total cholesterol alone, total and HDL cholesterol, or LDL and HDL cholesterol levels
- Without cardiac risk factors: measurement of total cholesterol
- USPSTF recommends screening for pt. with no evidence of CVD and no other risk factors should begin at 35 y/o; national cholesterol education program recommends screening all adults at age 20 y/o regardless of risk factors
- LDL goals
  - Pts with CAD or equivalent
    - Start drugs >130, LDL goal: <100, optimal level <70
  - No CAD but >2 RF
    - Start drugs >160, LDL goal <130
  - Everyone else:
    - Start drugs >190, LDL goal <160

Diagnosis / Screening:
- AHA: adults 20–79 who are free from CVD assess risk factors every 4-6 years to calculates 10 year CVD risk
- USPSTF: higher risk = initiate screening at age 25 for males and 35 females (>1 RF: HTN, smoking, family hx); low risk = initiate screening at age 35 males, 45 females

Health Maintenance:
- Goal of lipid levels:
  - Total cholesterol<200, HDL >45, triglycerides <150

Therapeutic Indications:
- Isolated increase LDL: statins, bile acid sequestrants
- Isolated increase TG: fibrates, niacin
- to increase HDL: niacin, fibrates
- type II DM: fibrates, statins (niacin may cause hyperglycemia so use caution in pt. with DM)

Clinical Therapeutics:
- 1. LIFESTYLE CHANGES!!!! weight reduction, increased exercise + diet: restriction of cholesterol and carbohydrates, decrease trans fatty acid, Mediterranean diet reduces LDL cholesterol without reducing HDLs; increase antioxidants from fruits and vegetables, soluble fiber may decrease LDL, CAD prophylaxis with aspirin 81 mg daily recommended; smoking cessation
- lipid lowering agents: plaque stabilization, reversal of endothelial dysfunction, decreased thrombogenicity, may cause the regression of atherosclerosis
- statins:
  - best drug to decrease LDL
  - decrease LDL (20-40%), increase HDL, decrease TG
  - inhibits rate limiting step in hepatic cholesterol synthesis (HMGcoA reductase inhibitor);
  - increase LDL receptors (removes LDL From blood)
  - shown to decrease cardiovascular complications
  - atorvastatin, lovastatin, simvastatin, pravastatin
  - s/e: myositis, myalgias, rhabdo, hepatitis
  - c/i: used in tetracyclines and abx (higher evidence si/e)
  - best given at bed time (when cholesterol synthesis is maximal)
- ** new lipid guidelines 2013 – statins recommended by 10 year and lifetime risk calculator instead of strict numbers – r/f = age, race, smoking, blood pressure, blood cholesterol levels, diabetes mellitus
  - Type 1 or 2 pt. with DM between ages 40-75
  - Pt without CVD ages 40-75 but have 7.5% higher risk for having heart attack or stroke in 10 years
  - People >21 y with LDL levels >190
  - People with CVD

- nicotinic acids / niacin (vitamin B3)
  - best drug to increase HDL
- 2nd best drug to decrease TG
- Shown to decrease cardiovascular complications
- s/e: flushing, headache, warm sensation, pruritis 80% - aspirin / ibuprofen prior to dosing may decrease flushing
- s/e: hyperuricemia – may precipitate gout, hyperglycemia
- decrease LDL (25%), increase HDL (20%), decrease triglycerides (20-50%)
  - **fibrates**: minimal decrease LDL, increase HDL (10-15%), **decrease triglycerides (60%)**
    - **best drug to decrease triglycerides**
    - inhibit peripheral lipolysis and reduce hepatic / TG production
    - s/e: increased LFT’s, myositis, myalgias esp with concomitant statin use; increased bile lithogenicity (gallstones)
    - c/i: severe hepatobiliary dz or renal dz
    - gemfibrozil, fenofibrate
  - **bile acid sequestrants**:
    - decreased LDL (increased with statins), increase HDL (3%), may increase TG (use in pts with normal TG)
    - binds bile acids in intestine blocking enterohepatic reabsorption of bile salts
    - most useful in combine with statin or niacin
    - ONLY MEDS SAFE IN PREGNANCY
    - Used for pruritis associated with biliary obstruction
    - s/e: GI – n/v, bloating, crampy abdominal pain, increased LFTs; increased triglycerides
    - may impair absorption of digoxin, warfarin, fat soluble vitamins (give those meds 1 hour before or 4 hours after BAS)
    - cheolstyramine, colestipol, colesevelam
  - ezetimibe: blocks intestinal absorption of dietary and biliary cholesterol by blocking cholesterol transporter and may be used as monotherapy or in combination with statin
    - lowers LDL; may be used with statins
    - s/e: increased LFT’s especially with statin use

### Hypertension

#### History and Physical Exam:
- goals: identify CV risk factors, reveal secondary causes HTN, assess for end organ damage
  - **fundoscopic exam**: papilledema = advanced stage of malignany HTN = more prognostic than isolated BP measurement
  - skin: uremic appearance, striae (cushing’s),
  - neck: carotid bruits, JVD
  - cardiopulm: loud cmpomponent of S2, S4
  - abdomen: phenochromocytoma, polycystic kidneys, bruits over renal artery (stenosis), dilation of aorta, truncal obesity
  - arterial pulses: decreased or absent femoral pulse (peripheral vascular disease) and BP greater in upper extremities suggests aortic coarctation; edema
- complications:
  - cardiovascular: coronary artery disease, heart failure, MI, LV hypertrophy, dissection, aneurysms, PVD
  - neurologic: TIA, stroke, ruptured aneurysms, encephalopathy
  - nephropathy: renal stenosis, sclerosis – HTN 2nd MC cause of end stage renal dz in US
  - optic: retinal hemorrhage, blindness, retinopathy

#### Diagnostic Studies:
- 2 separate blood pressure readings on 2 different visits

#### Diagnosis:
- elevated BP >2 reading on >2 different visits
- systolic >140 or diastolic >90
isolated systolic shows greater risk for CVD than isolated diastolic in pts >50y

- normal: <120/<80
- pre-HTN: 120-139/80-89
- stage 1: 140-159/90-99
- stage 2: >160/>100

Health Maintenance:
- goal <130/
- bp goals in pt. with CKD: 140/90 (instead of 130/80)
- bp goal in pt. >60: 150/90
- choosing initial meds in uncomplicated HTN in non-african American pt.: any 1 of the 4 drugs from following classes: thiazide-type diuretics, ACE-I, ARB, CCB (departure from just HCTZ diuretics as initial drug)
- LVH most important prognostic factor

Clinical Therapeutics:
- nonpharm: DASH diet, salt restriction, weight loss, exercise, moderate ETOH, pt. education
- medications if:
  - SBP >140 or DBP > 90 (<60yo)
  - SBP >150, DBP >90 (>60)
  - SBP >140 or DBP >90 (CKD)
  - SBP >140 or DBP >90 (DM)
- 1st line therapy:
  - ACE-I (-pril)
    - Cardioprotective, synergistic effect when used with thiazides (decrease preload/ afterload); renoprotective
    - Indications: HTN (esp with h/o DM, nephropathy, CHF, s/p MI)
    - s/e: 1st dose hypotension, azotemia / renal insufficiency (esp if Cr >3), hyperkalemia, cough and angioedema (increased bradykinin), hyperuricemia
    - CI: pregnancy
    - NOT IN AFRICAN AMERICANS (use thiazides / CCB instead)
    - Include in CKD
  - ARB (-sartan)
    - consider in pt not able to tolerate beta blockers / ACEI in addition to ACEI
    - CI: pregnancy
    - NOT IN AFRICAN AMERICANS (use thiazides / CCB instead)
    - Include in CKD
  - CCB
    - Dihydropyridines (nifedipine, amlodipine) – potent vasodilators (little or no effect on cardiac contractility or conduction) – MC used in HTN
    - Non-dihydro (verapamil, diltiazem): affect cardiac contractility and conduction – used with afib
    - Ind: HTN, angina, raynauds
    - s/e: headache, dizziness, lightheadedness, flushing, peripheral edema – constipation with verapamil
    - CI: pts taking beta blockers / 2nd and 3rd degree HB
  - Diuretics (thiazide)
    - HCTZ:
      - tx of choice as initial therapy in uncomplicated HTN; cardioprotective
      - decrease blood volume / blood pressure by decreasing Na/H2O retention
      - MOA: prevent kidney Na/water reabsorption at distal diluting tubule
      - s/e: hyponatremia, hypokalemia, hyperuricemia (gout), hyperglycemia (DM)
    - loops (furosemide): use in HTN, CHF, edema, mild renal dz
      - inhibit water transport across loop of Henle = increased excretion Na, Cl, K
      - strongest class diuretics
• s/e: volume depletion, hypokalemia, hyperuricemia, hypochloremia metabolic alkalosis, hyperglycemia,
• CI: sulfa allergy
  ▪ Potassium sparing (spironolactone, amiloride, eplerenone):
    • Inhibit aldosterone mediated Na/H2O absorpption
    • s/e: hyperkalemia, gynecomastia with spironolactone
    • CI: renal failure, hypnatiremia
○ Beta blockers (no longer used first line)
    ▪ HTN (h/o MI / tachy); angina, acute MI, HF, pheochromocytoma, migrains, essential tremor
    ▪ MOA: blocks adrenergic renin release
    ▪ s/e: fatigue, depression, impotence, may mask tachy sx of hypoglycemia in DM (use caution with DM pt); caution if hypotensive or HR <50
    ▪ CI: 2nd/3rd degree heart block, decompensated heart failure; nonselective agents CI
    ▪ Cardioselective (B1): atenolol, metoprolol, esmolol
    ▪ Nonselective (B1, B2): propranolol
    ▪ Both alpha and B1,2: labetalol, carvedilol
○ Alpha 1 blockers (prazosin, terazosin, doxazosin): not used first line
    ▪ drug of choice for pts with HTN and BPH
    ▪ s/e: 1st dose syncope, dizziness, headache
○ Systolic heart failure: ACE-I or ARB, beta blocker, diuretic, aldosterone agonist
○ Post-MI: ACE-I, bb, ARB, aldo antagonist
○ CKD: ACEI-I and/or ARB
○ Angina: beta blocker, CCB
○ Afib: BB, non-dihyd CCB
○ Aflutter: BB, non-dihyd CCB
○ BPH: alpha blocker
○ Essential tremor: BB (non cardioselective)
○ Hyperthyroid: BB
○ Migraine: BB, CCB
○ Osteoporosis: thiazide diuretic
○ Raynauds: dihydropyrd CCB
○ Contraindications:
  ▪ Angioedema: ACE-I
  ▪ Bronchospastic dz: beta blocker
  ▪ Depression: reserpine
  ▪ Liver dz: methylodopa
  ▪ Pregnancy: ACE-I, ARB, renin hinhibitor
  ▪ Heart block: BB, nondihyd CCB
○ adverse effects:
  ▪ depression: BB, central alpha-2 agonist
  ▪ gout: diuretic (use CCBs instead)
  ▪ hyperkalemia: aldo antagonist, ACE-I, ARB, renin inhibitor
  ▪ hyponatremia: thiazide diuretic
  ▪ renovascular dz: ACE-I, ARB, renin inhibitor
Scientific Concepts:
  ▪ pathogenesis:
    ▪ increase sympathetic activity
    ▪ increased angiotensin II activity and mineralocorticoid excess
    ▪ increased mineralocorticoid activity (Na and water retention)
  ▪ primary essential: 95% HTN due to idiopathic etiology
  ▪ secondary: 5% - due to underlying, identifiable, and correctable cause; suspect if refractory to antihypertensives or sever BP
renal (4%) – renovascular MC cause of secondary

- renal artery stenosis: abdominal bruits, fibromuscular dysplasia MC cause renal artery stenosis in young pts.; atherosclerosis in elderly
- endocrine: primary hyperaldosteronism (hypokalemia, low plasma renin), pheochromocytoma
- coarctation of aorta, sleep apnea, ETOH, oral contraceptives, Sudafed, COX-2 inhibitor

Hypertriglyceridemia

History and Physical Exam
- DM, ETOH, obesity, steroids, estrogen

Diagnostic Studies
- lipid panel

Diagnosis
- >150

Clinical Therapeutics
- fibrates: minimal decrease LDL, increase HDL (10-15%), decrease triglycerides (60%)
  - best drug to decrease triglycerides
  - inhibit peripheral lipolysis and reduce hepatic / TG production
  - s/e: increased LFT’s, myositis, myalgias esp with concomitant statin use; increased bile lithogenicity (gallstones)
  - c/i: severe heptobiliary dz or renal dz
  - gemfibrozil, fenofibrate
- bile acid sequestrants:
  - decreased LDL (increased with statins), increase HDL (3%), may increase TG (use in pts with normal TG)
  - binds bile acids in intestine blocking enterohepatic reabsorption of bile salts
  - most useful in combination with statin or niacin
  - ONLY MEDS SAFE IN PREGNANCY
  - Used for pruritis associated with biliary obstruction
  - s/e: GI – n/v, bloating, crampy abdominal pain, increased LFTs; increased triglycerides
  - may impair absorption of digoxin, warfarin, fat soluble vitamins (give those meds 1 hour before or 4 hours after BAS)
  - cholestyramine, colestipol, colesevelam

Health Maintenance
- triglycerides <150

Peripheral Vascular Disease:
- atherosclerotic dz of the lower extremities (and vessels outside the heart and brain)
- sx: intermittent claudication = MC presentation; reproducible pain / discomfort in lower extremity brought on by exercise with exercise + relieved with rest; erectile dysf(n)
  - aortic bifurcation / common iliac = buttock, hip ground claudicatos
  - Lerche syndrome: claudication, impotence, decreased femoral pulses
  - femoral artery: thigh / upper caul claudication (MC)
  - popliteal artery: lower calf claudication
- signs: weak or absent distal pulses, arterial bruits, loss of hair, shiny atrophic skin, pallor with dependent rubor
  - dependent rubor, coolness of skin = more advanced disease
- severe disease: numbness, tingling, ulcerations, ischemia, gangrene
- 6ps caused by acute arterial embolism: pain, pulselessness, pallor, parestehsias, poiklothermia, paralysis
  - resting leg pain = limb threatened by ischemia (advanced disease)
- Diagnostics: arteriography = gold standard (clinically only done if revascularization is planned); doppler ultrasonography; ankle-brachial index <0.9 (normal = 1.1-2)
- Management: risk factor modification: discontinue tobacco, control diabetes, hypertension hyperlipidemia
Medications: B-blocker, ACE-I, statins
  o Platelet inhibitors:
    ▪ cilostazol = mainstay of treatment (helpful for intermittent claudication)
    ▪ aspirin
    ▪ clopidogrel (Plavix)
  o supportive: foot care, exercise: fixed distance walking to point of claudication, resting and continuing until symptoms occur 1hour/day
  o amputation if severe/gangrene
  o acute arterial occlusion: heparin for embolisms; thrombolytics if thrombus; embolectomy

Interventions: exercise program, antiplatelet therapy

Varicose Veins:
  o Sx: asymptomatic; aching and fatigue
  o Signs: dilated, tortuous veins; greater saphenous = MC; flat, reticular veins; telangiectasia; spider veins
  o Severe disease: chronic distal edema, abnormal pigmentation, fibrosis, atrophy, skin ulceration
  o Diagnostics: duplex ultrasonography
  o Management: weight loss, control risk factors; graduated compression stockings
  o Interventions: exercise programs, elevation, radiofrequency or laser ablation, compression, sclerotherapy, surgical stripping

Phlebitis:
  o Sx: superficial = dull pain, erythema; deep = swelling, heat, redness
  o Signs: superficial = erythema, tenderness, induration; deep = heat, edema, homan’s sign (calf pain w foot dorsiflexion)
  o Severe disease: vascular compromise
  o Diagnostics: duplex ultrasonography, venography, D-dimer
  o Management: superficial: bed rest, local heat, elevation, NSIADs; deep = anticoagulation (prevention is key!!)
  o Interventions: surgery

Chronic Venous Insufficiency:
  o Symptoms: progressive edema, itching, dull pain, ulcerations
  o Signs: shiny, thin, atrophic skin
  o Severe disease: ulceration (stasis ulcer, dermatitis)
  o Diagnostics: clinical; duplex ultrasonography
  o Management: prevention, elevation, avoid extended standing or sitting, compression hose
  o Interventions: wet compresses, compression boots or stockings, skin grafting

Valvular Disease
- **Tricuspid regurg:**
  - Location: LLSB, holosystolic
  - Radiation: right sternum and xiphoid area
  - JVP
- **Pulmonic Stenosis:**
  - Location: 2nd-3rd LICS; midsystolic crescendo-decrescendo
  - Radiation: left shoulder and neck
  - Early pulmonic ejection sound common
- **Tx for tricuspid and pulmonic:** sodium restriction, diuretic therapy, arterial vasodilators for pulmonary HTN; surgical repair, valvuloplasty, replacement (preferred)
PULMONOLOGY

**Asthma**

**Definition:** Obstruction of airflow, bronchial hyperreactivity, inflammation of the airway – disease of chronic inflammation → airway narrowing + increased mucus production

**History and Physical:**
- **Strongest predisposing factor = atopy; atopic triad = wheeze, eczema, seasonal rhinitis**
- **Triggers:** allergens, exercise, respiratory tract infections, GI disorders, drugs (BB, ACE-I, aspirin, NSAIDs), stress (emotional / weather change)
- Intermittent cough, chest tightness, breathlessness, wheezing (1/3 children = no wheezing)
- Asymptomatic periods

**Diagnostic Studies:**
- Airflow obstruction indicated by decreased ratio of forced expiratory volume in 1 second to forced vital capacity (FEV1/FVC <75%); <12% increase FEV1 after bronchodilatory therapy is supportive of diagnosis
- ABG may be normal in mild cases but hypoxemic / hypercapnic in severe cases with PaO2 <60mmHg and PaCO2 >40mmHg (rarely obtained)
- CXR may show hyperinflation (only indicated if pneumonia / other disorder is expected)
- Handheld peak expiratory flow meters estimate variability, quantify severity of attacks – good for those with chronic disease to self-monitor
- Histamine / methacholine challenge to establish diagnosis when spirometry is nondiagnostic; FEV1 decrease >20% = diagnostic

**Clinical Therapeutics:**
- **Long term:** corticosteroids, cromolyn, nedocromil, long-acting bronchodilators, leukotriene modifiers, theophylline
- **Short term:** short acting inhaled B2 agonist, ipratropium bromide, systemic corticosteroids
- Inhaled corticosteroids = more effective anti-inflammatory meds for management of chronic asthma

**Health Maintenance:**
- Minimize chronic symptoms, prevent recurrent exacerbations, minimize need for urgent care visits; maintain near normal pulm function; adequate hydration; avoid triggers

<table>
<thead>
<tr>
<th>Components of Severity</th>
<th>Classification of Asthma Severity</th>
<th>≥12 years of age</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td>Intermittent</td>
<td>Mild</td>
</tr>
<tr>
<td>≥2 days/week</td>
<td>&gt;2 days/week but not daily</td>
<td>Daily</td>
</tr>
<tr>
<td>Nighttime awakenings</td>
<td>≤2/day/night</td>
<td>3–4/day</td>
</tr>
<tr>
<td>β-blocker use for symptom control (not prevention of EIB)</td>
<td>≤2/day/week</td>
<td>&gt;2 days/week but not more than 1x on any day</td>
</tr>
<tr>
<td>Interference with normal activity</td>
<td>None</td>
<td>Minor limitation</td>
</tr>
<tr>
<td>Normal FEV1/FVC</td>
<td>85%</td>
<td>20–39%</td>
</tr>
<tr>
<td>Lung function</td>
<td>Normal FEV1 between exacerbations</td>
<td>FEV1 &gt;80% predicted</td>
</tr>
<tr>
<td></td>
<td>FEV1 &gt;80% predicted</td>
<td>FEV1/FVC normal</td>
</tr>
<tr>
<td>Risk</td>
<td>Exacerbations requiring oral systemic corticosteroids</td>
<td>0–1/year (see note)</td>
</tr>
</tbody>
</table>

**Recommended Step for Initiating Treatment**

(See figure 4-5 for treatment steps.)

<table>
<thead>
<tr>
<th>Step 1</th>
<th>Step 2</th>
<th>Step 3</th>
<th>Step 4 or 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>In 2–6 weeks, evaluate level of asthma control that is achieved and adjust therapy accordingly.</td>
<td>Consider severity and interval since last exacerbation. Frequency and severity may fluctuate over time for patients in any severity category. Relative annual risk of exacerbations may be related to FEV1.</td>
<td>and consider short course of oral systemic corticosteroids.</td>
<td></td>
</tr>
</tbody>
</table>
**Bronchitis**

**Definition:** inflammation of the airways (trachea, bronchi, bronchioles)

**History and Physical:**
- **Cough (without or without sputum for 1-3 weeks)**, dyspnea, fever, sore throat, headache, myalgias, substernal discomfort, expiratory rhonchi or wheezes
- Sputum color not predictive of bacterial involvement
- No signs of tachypnea and tachycardia

**Diagnostic Studies:**
- No lab eval
- CXR negative (if differentiating with pneumonia)

**Diagnosis:**

**Clinical Therapeutics:**
- Supportive measures – hydration, expectorants, analgesics, B2 agonists, antitussives (dextromethorphan, guaifenesin, dextromethorphan/pseudoephedrine, bromhexine)
- DO NOT USE NONPRESCRIPTIN COUGH AND COLD PRODUCTS IN KIDS <2 YEARS OLD
- For acute exacerbations of chronic bronchitis (more likely bacterial), empiric first line tx is second generation cephalosporin; second line treatment = second generation macrolide or Bactrim
- Abx indicated for: elderly pt., those with underlying cardiopulmonary diseases, and cough >7-10 days, immunocompromised
- Acute exacerbations in healthy adults = no empiric abx
- Abx no statistical benefit in healthy patients

**Health Maintenance:**

**Scientific Concepts:**
- >90% caused by viruses (adenovirus)
- With chronic lung disease, causes include H.flu, S.pneumo, M. catarrhalis

**COPD**

**Definition:** progressive, largely irreversible airflow obstruction due to 1. Loss of elastic recoil 2. Increasing airways resistance
- Includes emphysema and chronic bronchitis – both usually coexist with one being more dominant
History and Physical:
- Risk factors: cigarette smoking / exposure = most important risk, alpha 1 antitrypsin deficiency = genetic and linked to COPD in pt. <40y/o (protects elastin in lungs from damage by WBCs)
- Emphysema:
  - DOE = hallmark symptom
  - Hyperinflation of lungs + hyperresonance to percussion, decreased / absent breath sounds, decreased fremitus, barrel chest (increased AP diameter), quiet chest, pursed lip breathing
  - ABG / labs: respiratory alkalosis
  - Matched V/Q defects, mild hypoxemia, normal CO2
  - Cachectic with pursed lip breathing → “pink puffers”
- Chronic Bronchitis:
  - Productive cough hallmark sx
  - Rales (crackles), rhonchi, wheezing, signs of cor pulmonale (peripheral edema, cyanosis)
  - Respiratory acidosis (increased Hct/RBC bc chronic hypoxia stimulates erythropoiesis)
  - Severe V/Q mismatch, severe hypoxemia, hypercapnia
  - Obese and cyanotic = blue bloaters

Diagnostic Studies:
- PFTs / spirometry = gold standard diagnosis COPD
  - FEV1 = important factor of prognosis and mortality (<1L = increased mortality)
  - Obstruction: decreased FEV1, decreased FVC, decreased FEV1/FVC;
  - Hyperinflation: increased lung volumes: increased RV, TLC, RV/TLC, increased FRC (functional residual capacity)
- CXR/CT scan
  - Emphysema: hyperinflation: flat diaphragm, increased AP diameter, decreased vascular markings; bullae
  - Chronic bronchitis: increased AP diameter, increased vascular markings, enlarged right heart border
- ECG: cor pulmonale: RVH, RAE, RAD, R-sided heart failure (due to longstanding pulmonary hypertension), MAT, hypertension

Diagnosis:

Clinical Therapeutics:
- SMOKING CESSATION = SINGLE MOST IMPORTANT STEP
- 1. Bronchodilators: combo therapy b2 agonist + anticholinergic = greater response than used alone – tx of choice in stable COPD with resp. symptoms
  - 1. Anticholinergics: tiotropium (Spiriva) inhaled long acting; ipratropium (Atrovent)
    - Ipratropium preferred over short acting B2 agonist in COPD
    - s/e: dry mouth, thirst, blurred vision, urinary retention, difficulty swallowing, mydriasis
    - CI: glaucoma, BPH
  - 2. B2 agonist: albuterol, terbutaline, salmeterol (long acting)
    - s/e: B1 cross reactivity, tachycardia/arrhythmias, muscle tremor, CNS stimulation
    - CI: severe CAD; caution in pt. with DM (hyperglycemia), hyperthyroid
  - 3. Theophylline: only used in refractory cases bc narrow therapeutic index – monitor serum levels to prevent nausea, palpitations, arrhythmias, seizures from toxic levels; higher doses needed in smokers
- 2. Corticosteroids: inhaled corticosteroids not considered monotherapy
  - s/e: osteoporosis, thrush
- 3. Oxygen: only medical therapy proven to decrease mortality (decreases pulmonary hypertension / cor pulmonale by decreasing hypoxia mediated pulmonary vasoconstriction)
  - Ind: use if cor pulmonale / O2 sat <88%

Health Maintenance:
- Control triggers: pollutants, bronchospasm, cardiopulm disease, meds (decongestants, B blockers, sedative)
- Infections: bronchitis and pneumonia from virus: s.pneumo, h.flu, m. catarrhalis
- Prevention of exacerbations: SMOKING CESSATION
- Vaccinations: pneumococcal and influenza q fall
- Pulmonary rehab: improves quality of life, dyspnea, and exercise intolerance
- Surgery: A. lung reduction surgery – improves dyspnea by removing damaged lung B. lung transplant
o Azithromycin has anti-inflammatory properties in the lung

Scientific Concepts:
- A. emphysema abnormal, permanent enlargement of terminal airspaces
  - Smoking: chronic inflammation decreases protective enzymes and increases damaging enzymes → alveolar capillary and alveolar wall destruction from decreased gas exchange surface area → loss of elastic recoil & increased compliance → increased airway obstruction (air trapping)
- B. chronic bronchitis: productive cough >3 mos x2y consecutively – increased airway resistance leading to airway obstruction – mucous plugging + mucociliary escalator destruction → pt. prone to microbial infections

<table>
<thead>
<tr>
<th>Stage</th>
<th>PFT% predicted</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>I: mild</td>
<td>FEV1 &gt; 80%</td>
<td>Bronchodilators prn short acting / decrease risk factors</td>
</tr>
<tr>
<td>II: moderate</td>
<td>FEV1 50-80%</td>
<td>Above + long acting dilator</td>
</tr>
<tr>
<td>III: severe</td>
<td>FEV1 30-50%</td>
<td>Above + pulm rehab; steroids if increased exacerbations</td>
</tr>
<tr>
<td>IV: very severe</td>
<td>Cor pulmonale, right heart failure, resp failure, FEV1 &lt;30%</td>
<td>Above + O2 therapy</td>
</tr>
</tbody>
</table>

Lung Cancer
Small cell carcinoma (15%) + non-small cell lung carcinoma (85%) + other (<1%)

History and Physical:
- Risk factors: tobacco, second hand smoke, occupational exposure, indoor air quality, genetic factors, hormonal factors, oncogenic viruses
- Lung cancer in non-smokers = more likely to be adenocarcinoma
- Multiple hit hypothesis: irritants + carcinogens + time + mutations + time (oncogenes, tumor suppressor genes, growth factors, epigenetics)
  - Normal epithelium → squamous metaplasia → dysplasia → carcinoma in situ → invasive carcinoma
  - Majority are symptomatic at presentation: cough, anorexia, weight loss, fatigue, anemia, fever, pain
  - >80% have 3+ symptoms
- Pulmonary lesion manifestations: cough, hemoptysis, dyspnea, chest pain → postobstructive pneumonia
- Intrathoracic spread: lymphnodes, dysphagia, hoarseness (recurrent laryngeal nerve paralysis), phrenic nerve paralys, Pancoast tumor, horner syndrome (ipsilateral miosis, ptosis, anhidrosis), invasion of brachial plexus, superior vena cava syndrome (facial / upper extremity swelling), pleural effusion, pericardial effusion, lymphangitic spread,
- Extrathoracic spread: hilar / mediastinal lymph nodes, pleura, opposite lung, liver, adrenal gland, bone, CNS
- Paraneoplastic syndromes: endocrine syndromes (hypercalcemia, SIADS, ectopic ACTH secretion) / neurologic syndrome (disease of neuromuscular junction – strength improves with repetitive stimulation), digital clubbing, hypercoagulable states

Types
- **Small cell lung carcinoma** = strongly associated with cigarettes, occurs central, 2/3 present at “extensive stage”, poorly differentiated neuroendocrine tumors
  - ACTH → cushing syndrome
  - SIADH → hyponatremia
- Squamous cell: usually central, but can be peripheral; highly associated smoking
- **Adenocarcinoma**
  - Bronchioloalveolar cell carcinoma (BAC) – subtype of adenocarcinoma, most common in non-smokers; very slow growing: mucinous, non-mucinous, mixed
- **Neuroendocrine tumors:**
  - Typical carcinoid + atypical = younger, non-smoker, less aggressive
    - Carcinoid syndrome = flushing
  - Large cell neuroendocrine + small cell = smokers, more aggressive
- **Solitary Pulmonary Nodule**
Asymptomatic lesion <3cm diameter surrounded by normal lung parenchyma; no symptoms – incidental finding
10-70% = malignant; ground glass density + irregular borders
Unchanged in size >2 years = likely benign
Increased malignancy: advanced age, hx smoking, hx prior malignancy, lesions characteristics / size
Get a CT and evaluate the lesion
- 8-20 mm = increased chance malignancy (get a PET scan / bx)
- 30-100 days doubling time growth rate = more likely malignant
- Margin characteristics – smooth borders suggests benign; lobulated suggest malignancy; corona radiata suggest malignancy, halo sign = depends
- Attenuation (density characteristics) – solid lesions more likely benign/ ground glass / part solid more likely malignant
- Calcification >200 hounsfield units
  - Diffuse, popcorn, central usually benign
  - Location: malignant nodules can be found in any lobe but upper lobes increased probability malignancy
Eval: PET scan + biopsy

Diagnostic Studies:
- Requires tissue sample, fiberoptic bronchoscopy, transthoracic needle aspiration, mediastinoscopy, thoracotomy, video assisted thoracoscopic surgery
- CXR and CT can detect suspicious lesions, but don’t provide specific diagnosis
- PET scan
- Combined PET + CT: improve preop staging

Diagnosis:

Clinical Therapeutics:
- Depends on staging
- Basic modalities: surgery, chemo, radiation
  - Surgery
    - Stage 1 and 2: surgery = primary treatment; candidacy depends on pulm function (<40% precludes)
    - Stage 1 non-surgical = radiotherapy
    - Cure unlikely without resection
  - Chemo:
    - Adjuvant: antineoplastic drugs following surgery or radiation therapy
    - Neoadjuvant: before surgery / radiation – used with stage IIA or IIB
    - Palliative: improve quality of life and symptoms
  - Radiation: not indicated for stage I and II
  - SCLC:
    - Initial response rates to chemo = excellent
    - Remissions tend to be short lived
    - 2 year survival is 20-40% in limited stage disease and 5% in extensive disease

Health Maintenance:

Scientific Concepts:
- Almost all lung cancers = carcinomas arising from epithelial tissue lining respiratory tract

History and Physical:
- CAP = s. pneumo
  - Pneumonia acquired outside of the hospital / not in nursing home OR ambulatory prior to admission who develops pneumonia 48 hours of initial admission to hospital
- Hospital acquired = gram negative rods: pseudomonas
  - Pneumonia occurring >48 hrs after hospital admission
- Immunocompromised: hospital acquired
- Aspiration: outpt = aneurobes; inpt = GNR and s.aureus
- Typical (s.pneumo)
  - Lobar pneumonia on XR
  - Sudden onset fever, productive cough and prurulent sputum, pleuritic chest pain, rigors assoc with s. pneumo especially, tachycardia, tachypnea
  - p/e: bronchial breath sounds, dillness to percursssion, increased tactile fremitus, egophony
- Atypical (mycoplasma)
  - Diffuse patchy infiltrates on XR
  - Low grad fever, dry non productive cough, extrapulum sx: myalgias, malaise, sore throat, headache, n/v/d
  - p/e: often normal
  - p/e: dullness with percussion, increased fremitus, bronchial / egophony breath sounds

Diagnostics:
- CXR / CT scan: CXR resolution lags behind clinical improvement for weeks
  - Abscess formation = s. aureus
  - Upper lobe (RUL) with bulging fissure and cavitations → klebsiella
- Sputum: gram stain / culture
  - Rusty / blood tinged = strep pneumo
  - Currant jelly = klebsiella
  - Green = h.flu / pseudomonas
  - Foul smelling = anaerobes

Clinical Therapeutics:
- CAP outpatient: macrolide or doxy (FQ only if co-morbid conditions / recent abx use)
- CAP inpatient: B lactam + macrolide OR broad spectrum FQ
- CAP ICU: B lactam + macrolide OR B lactam + broad spectrum FQ
- HAP (pseudomonas): anti-pseudomonal B lactam _ anti pseudomonal aminoglycoside or FQ
- Aspiration: clindamycin or augmentin +/- metronidazole

Health Maintenance:
- PCV 13: used in childhood vaccination
- PPV23: polyclaval pneumococcal vaccine used in adults
  - Age >65 = revaccinated every 5 years
  - 2-64 with chronic disease every 5 years

Scientific Concepts:
  **Bacterial**
  - Microaspiration of oropharyngeal secretions = MC route of infection
  - S. pneumo = MC cause of CAP (65%)
  - H. flu = 2nd MC cause CAP and with underlying pulmonary disease: COPD, bronchiectasis, cystic fibrosis
  - Mycoplasma pneumoniae: MC cause atypical (lacks cell wall) / walking pneumonia
  - Legionella – cooling towers, AC, contaminated water (aquatic environment, not person to person); anorexia, nausea, vomiting, diarrhea, increased LFTs, hyponatremia
  - Staph aureus: often see post viral illnesses x. flu
  - Klebsielle: severe illness in alcoholics, associated with cavitary lesions
  - Aneurobes: aspiration pneumonia especially RLL
  - Psudomonas aeruginosa: immunocompromised

  **Viral**
  - RSV and parainfluenza = MC cause viral in small infants / children
  - Influenza = MC cause in adults

  **Fungal**
  - Pneumocystis jiroveci (PCP) – compromised host (fatigue, dry cough, dyspnea on exertion – O2 desaturation with ambulation, pleuritic chest pain)
  - Histoplasma capsulatrum: Mississippi and ohio river valley
  - Coccidioides: southwest US in desert areas
Sleep Disorders

**History and Physical:**

- **Insomnia** = difficulty falling and staying asleep, intermittent wakefulness during the night, early morning awakenings, or some combination – depression + anxiety → persistent insomnia
  - Depression: fragmented sleep, decreased total sleep time, quicker onset REM, shift of REM to earlier in the night
  - Manic: decreased total sleep time; shortened REM and increased REM activity
- Hypersomnia: excessive daytime sleepiness = more severe problem – sleep apnea, narcolepsy, nocturnal myoclonus
- Parasomnia = abnormal behavior during sleep) – sleep terrors, nightmares, sleepwalking, enuresis
  - Nightmares occur during REM
  - Sleep terrors occur during stage 3 and 4 delta sleep
  - Sleepwalking associated with stage 3 or 4 delta sleep in first third of night
  - Childhood enuresis: within 3-4 hours of bedtime but not limited to certain stage of sleep
- History: depression, alcohol abuse, heavy smoking (>1 pack/day), inappropriate use of sedatives / stimulants, medical history uremia, asthma, hypothyroid
- Sleep apnea = obese, middle aged, older men with hypertension and associated CHF
- Narcolepsy: sudden, brief sleep attacks, cataplexy, sleep paralysis, hypnagogic hallucinations, which may precede sleep

**Diagnostics:**

- Polysomnography (sleep studies) assess EEG activity, heart rates, respiratory movement, oxygen saturation
- Thyroid studies

**Therapeutics:**

- Insomnia:
  - De-emphasis and reassurance with sleep hygiene education: avoid alcohol, caffeine, nicotine, exercise prior to bed; establish regular sleep hours; relaxation techniques; avoid prolonged daytime naps
  - Avoid medications; antihistamines may be effective for milder problems
  - Rapidly acting hypnotics can be used for short periods if necessary
- Sleep apnea: weight reduction, CPAP
- Narcolepsy: administration of stimulants (dextroamphetamine and modafinil)
- Nocturnal myoclonus: clonazepam
- Sleep terror / sleepwalking: benzos

**Scientific Concepts:**

- Sleep = REM (dreaming) and nREM (stages 1-4, 3 & 4 = delta sleep)

Tobacco Use / Dependence

**History / Physical Exam:**

**Diagnostics:**

**Therapeutics:**

**Scientific Concepts:**

Tuberculosis

Chronic infection with mycobacterium tuberculosis → granuloma formation

**History / Physical Exam:**

- Cough = MC sx: dry → productive without hemoptysis usually over 3 weeks
  - Hemoptysis in advanced stages
- Fever, drenching night sweats, anorexia, weight loss, pleuritic chest pain, dyspnea, hemoptysis, post-tussive rales
- Chronically ill, malnourished
- p/e: signs of consolidations
- Extra-pulmonary: can affect any organ
  - vertebral = pott’s disease
  - lymph nodes = scrofula
TB meningitis
Other: pericarditis, peritonitis, joints, kidney, adrenal, cutaneous

Diagnostics:
- Acid-fast smear and sputum culture x3 days – 3 negative smears = noninfectious
  - **AFB cultures gold standard**
- Radiography – indicated to excluded active TB and sued as yearly screening in pt with known positive PPD to rule/out active TB
  - Primary TB: homogenous infiltrates, hilar node enlargement, segmental atelectasis, cavitations
    - CONTAGIOUS
  - Middle / lower lobe
  - Reactive: fibrocavitary apical dz, nodules, infiltrates, posterior and apical segment of right upper lobe
    - Reactivation of latent TB with waning immune defenses – Us localized in apex / upper lobes with cavitory lesions ➔ CONTAGIOUS
    - HIV 7-10% yearly chance for reactivation of LTBI
- Miliary: millet-seed like nodular lesions
- Ghon complex (calcified primary focus) and ranke (calcified primary focus and calcified hilar lymph node) complex represent healed primary infection
- Latent: PPD positive 2-4 weeks after infection – NOT CONTAGIOUS
- TST (skin test) identifies individuals infected but doesn’t differentiate active vs latent infection
  - >5mm in HIV person, recent contact with active TB, evidence ofTB on CXR, immunosuppressed / steroids
  - >10mm: recent immigrants, HIV neg IVDU, healthcare personnel, children <4yo, DM, silicosis, CKD
  - >15mm: everyone else; no risk factors
- Definitive diagnosis requires identification of m. tuberculosis from culture (6-8 weeks to grow) or DNA or RNA amplification techniques (1-2 days)
- Biopsy revealing caseating granuloma (necrotizing)
- False negative: anergy (HIV, sarcoidosis), faulty application, acute non-TB infections, malignancy
- False positive: improper reading, cross reaction with atypical, c/n 2-10 years of BCG vaccination
- Booster effect: infected person’s immune system forgets about TB until years later when testing reminds the immune system – next PPD = positive bc of initial infection (years ago) NOT bc recently converted – confirmed by 2 step PPD

Therapeutics:
- Latent TB: IH for 9 months or RIF for 4 months or RIF and PZA for 2 months
  - Treat for LTBI only after active TB is ruled out
- Active TB: INH/RIF/PZA/EMB for 2 months followed by 4 months multidrug tx based on culture and sensitivity
  - Require combo chemo for 6-9 months
  - With HIV require therapy for at least 1 year
- Drug-resistant: expert advice sought
- INH 6-12 mo for prophylaxis in pt who have tested negative in the past but are now positive with known or unknown exposure (recent converters)
- **Pts no longer considered infectious 2 weeks after initiations of therapy**
- s/e:
  - INH: hepatitis, peripheral neuropathy – give B6 alongside it
  - RIF: thrombocytopenia, hepatitis, flu syndromes, orange body fluid
  - PZA: hepatitis and hyperuricemia (caution in gout / liver dz), Gl sx, arthritis, photosensitive rash
  - EMB: optic neuritis (red-green vision), peripheral neuropathy
  - Streptomycin: ototoxicity, nephrotoxicity

Health Maintenance:
- Confirmed or suspected cases reported to public health agencies
- Active dz = isolated until minimum 2 weeks tx
- BCG vaccine can be administered to TB negative person in settings with high risk for intense prolonged exposure - not recommended in the US
- Children, adolescents, immunocompromised in close contact with person with active TB should be offered tx until TST is negative 12 weeks after exposure
Scientific Concepts:
- Inhalation of airborne droplets → mycobacterium reaches alveoli and are ingested by alveolar macrophages
  - If Mtb remains viable in macrophage, bypassing its defense or escape = active infection
- Caused by inhaling organisms within aerosol droplets expelled during coughing by someone with active dz
- Most exposed people mount immune response sufficient to prevent → clinical illness, 10% infected develop dz (primary TB) and 5% fail to contain primary infection and progress to active TB
- 95% won’t be symptomatic (latent) - can’t spread the dz – usually in apices of lungs

GASTROINTESTINAL / NUTRITIONAL

**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

**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, periumbilical / 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 / extension (raise leg vs resistance)
  - Mcburney’s point tenderness: the point 1/3 the distance from ASIS and navel

Diagnostics:
- Abdominal US is preferred initial imagine
- 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

**Bowel Obstruction**

**Definition:**
- Most small bowel 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 is may cause SBO)
- Lg. 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

**Cholecystitis**

**Definition:** gall bladder (cystic duct) obstruction by stone → 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)

**Diagnostics:**
- **US = initial test of choice:** thickened GB <3mm, distented 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 (heptominodiacetic acid): gold standard** – positive HIDA = nonvisualization of gallbladder in cholecystitis
- 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 spincter of oddi spasm)
- Acute alcalculus cholecysititis: MC occur in seriously ill pt.
- Chronic cholecystitis: assoc with gallstones; strawberry GB (interior of GB resembles strawberry secondary to cholesterol submucosal aggregation) \(\rightarrow\) porcelain GB (premalignant condition)

**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 dissol
- **Cholecystectomy** in symptomatic pt (usually laproscopic)

**Complications:**
- **Choledocholithiasis:** gallstones in biliary tree \(\rightarrow\) +/- biliary colic or jaundice
  - **Tx:** stone extraction via ERCP
- **Cholangitis:** biliary tract infection secondary to obstruction by gallstone
  - **Dx:** ERCP
  - **Charcot’s triad:** fever/chills, RUQ pain, jaundice
  - **Reynold’s pentad:** shock + AMS
  - **Tx:** abs (PCN + aminoglycoside), decompression of biliary tree via ERCP stone extraction

**Cirrhosis**

**Definition:** irreversible fibrosis and nodular regeneration throughout the liver; secondary to chronic liver dz; nodules cause increased portal pressure; macro nodules associated ith higher risk hepatocellular carcinoma; in US >45% are alcohol related cases (others = hep B / hep C)

**History and Physical Exam:**
- ETOH = MC cause; others: chronic viral hepatitis (especially HCV, HBV, HDV); nonalcoholic fatty liver dz (obesity, DM, hypertriglyceridemia), hemochromatosis, autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis, drug toxicity
- **Weakness, fatigue, weight loss, muscle cramps, anorexia,** n/v/anorexia, menstrual changes (amenorrhea), impotence, loss of libido, gynecomastia, abdominal pain, hepatomegaly
- **Skin manifestations:** spider angioma (upper body), caput medusa, muscle wasting, bleeds, hepatosplenomegaly, palmar erythema, jaundice, dupuytren’s contractures
- **Late stage disease:** ascites, pleural effusions, peripheral edema, echymoses esophageal varices (due to portal HTN; may progress to ESLD), signs of hepatic encephalopathy (**confusion and lethargy, increased ammonia, fetor hepaticus**), asterixis, tremor, dysarthria, delirium, coma
- Spontaneous bacterial peritonitis: fever, chills, worsening ascites, abdominal pain \(-\) may lead to diarrhea / renal failure
- **Physical:** ascites (abdominal distention by fluid due to decreased oncotic pressure and decreased hepatic protein production), hepatosplenomegaly, gynecomastia (liver can’t metabolize estrogen)

**Diagnostics:**
- Lab values minimally abnormal until late in dz
- Anemia = common
- Mild elevations AST and alk phos, increased gamma-globulin, decreased albumin, abnormal coagulation studies
- US to determine liver size and eval for hepatocellular carcinoma
- Liver biopsy
US, CT, MRI can confirm the size and number of nodules and is helpful in guiding biopsy

Therapeutics:
- **Alcohol abstinence!!!**
- Treatment of viral causes = required
- Salt restriction and bed rest may be sufficient for ascites
- **Encephalopathy**: lactulose → neomycin (s/e = diarrhea); protein restriction
- **Ascites**: Na restriction, paracentesis
- **Pruritus**: cholestryamine
- Spironolactone 100mg daily → diuretic
- Liver transplant indicated in selected pt. = definitive management
- Spontaneous bacterial peritonitis treated with abx

Health Maintenance:
- Cirrhotic pt must be screened every 6 mo for hepatic carcinoma regardless of successful treatment of the underlying cause

**Colorectal Cancer**

Definition:

History and Physical:
- r/f: age >50y (peaks 65y), US/Crohns dz, polyps, familial adenomatous polyps (genetic mutation of APC gene – 100% develop colon cancer by age 40); **diet** (low fiber, high red/processed meat, animal fat); smoking, ETOH, AA, UC, HNPCC / Lynch syndrome (autosomal dominant → 3% colorectal cancers)
- those with familiar polyposis have virtually 100% risk developing dz
- **peutz-jehgers**: age 20y autosomal dominant, polyposis, mucocutaneous hyperpigmentation (lips, buccal, hands)
- slow growing so sx appear late in dz: iron deficiency anemia, rectal bleeding, abdominal pain, change in bowel habits, occult bleeding, intestinal obstruction
  - right sided (proximal) lesions tend to bleed and cause diarrhea
  - left side (distal) tend to cause bowel obstruction and present later; change in stool diameter
- fatigue and weakness if chronic blood loss → anemia
- change in stool size and shape may be noted + frank blood in stool

Prognosis:
- good in early dz
- when cancer involves only mucosa (dukes A or stage I), 5y survival rate >90%
- penetration through wall or involvement regional lymph nodes (dukes B / stage II), 5y survival 70-80%
- mets (dukes C / stage II with lymph nodes) and dukes D / stage IV (distant mets), 5y survival 5%

Diagnostics:
- occult blood in stool can be early marker
- CEA may be used to monitor although not detect colon cancer
- Sigmoidoscopy, colonoscopy, barium eneme may all be used to visualize suspected colonic masses;
- CXR CT used to detect mets
- colonoscopy with biopsy
- barium enema (apple core lesion classic)
- increased CEA
- CBC: anemia

Therapeutics:
- 5FU mainstay of chemotherapy
- **Surgical resection**
- Chemo in stage III or higher
- Radiation may be used for rectal tumors
- Monitor CEA with treatment

Health Maintenance:
- **Screening**:
  - Average risk: annually at 50y; colonoscopy q10y or flex sig q5y; up to 75y
  - 1st degree relative >60y: annually at 40; colonoscopy q10y
1st degree relative <60y: annually at 40 (or 10y before age relative diagnosed); colonoscopy q5y

**Scientific Concepts:**
- Progression of adenomatous polyp into malignancy (adenocarcinoma) usually occurs within 10-20 y
- 3rd leading cause in cancer deaths (after lung and skin)
- 90% = >50yo

**Colonic Polyps**

**Definition:**
- Common in industrialized world and can be benign or malignant
- Removal can reduce occurrence of colon cancer
- Inherited syndromes → genetic disposition of nearly 100% contraction
  - Up to 5% found with these syndromes: familial adenomatous polyposis, hamartomatous polyposis, peutz-jeghers syndrome, familial juvenile polyposis, PTEN multiple hamartoma syndrome

**History and Physical:**
- Generally asymptomatic; constipation, flatulence, rectal bleeding may occur
- Bleeding polyps → iron deficiency anemia
- Pseudopolyps/inflammatory: due to IBD (ex UC/Crohn’s)
- Not considered cancerous
- Hyperplastic = low risk for malignancy; 90% of all polyps
- Adenomatous: 10% of polyps; average is 10-20y before becoming cancerous (Esp >1cm)
  - Tubulous adenoma: non-pedunculated (MC of the 3 types)
  - Tubulovillous: mixture
  - Villous: highest risk of becoming cancerous; tends to be sessile

**Diagnostics:**
- Heme positive stool
- Barium enema, flexible sigmoidoscopy, coloscopy can detect them
- Histologic eval to determine dysplasia: hyperplastic polyps = lowest risk dysplasia; tubular have increased risk; villous = highest risk malignancy
- Family members of those with familial polyposis syndrome should be evaluated every 1-2 years beginning at 10-12 years of age – elective colectomy may be option for high risk individuals

**Therapeutics:**
- Depends on size and histology of polyps
  - Larger and dysplastic: removed with frequent follow up
  - Single distal hyperplastic polyp: same follow up as someone with tubular polyps (5 year follow up)
  - Multiple hyperplastic polyps, hyperplastic polyps at sites rather than distal or tubular polyps = 5y f/u
  - Villous: follow up colonoscopy at 3 years – surveillance = more frequent

**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 / kaopectate): 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
    - CI: 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 (benztropine)
    - Neuroleptic malignant syndrome: life threatening diroder – mental status changes, extreme muscle rigidity, tremor, fever, autonomic instability (tachycardia)
      - give dopamine agonist for reversal – bromocriptine / levodopa / carbidopa
  - Supportive therapy is sufficient for most patients with viral or bacterial diarrhea
  - Abx may be indicated with severe diarrhea / systemic symptoms (shigella, campylobacter, c. diff)
    - Metronidazole, oral vanco, fidaxomicin are abx of choice for abx induced C. diff colitis
  - Tx of the underlying cause = required for noninfectious diarrhea
  - Bismuth loperamide (immodium)

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

**Constipation**

**Definition:**
- Normal bowel function ranges from three stools/day to three stools/week
- Decrease in stool volume and increase in stool firmness accompanied by straining
- >50yo with new onset constipation should undergo further investigation to detect the underlying cause

**History and Physical:**

**Diagnostics:**

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

**Health Maintenance:**
- 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
- If treatable underlying cause is found, constipation will resolve with treatment of the disease process

**Scientific Concepts:**

**Esophagitis**

**Definition:**

**History and Physical:**
- GERD = MC cause
Infections in immunocompromised = 2nd MC (candida, CMV, HSV)

Also caused by: radiation therapy, medication or corrosive ingestion, eosinophilic (assoc with food allergies, atopic disease)

r/f: pregnancy, smoking, obesity, ETOH, chocolate, spicy foods, meds (NSAIDS, beta blockers, CCB)

odynophagia (painful swallowing), dysphagia (difficulty swallowing)

Symptoms resemble GERD

Diagnostics:
- upper endoscopy, double contrast esophogram

Therapeutics:
- treat underlying cause

Gastritis

Definition:
- gastritis = superficial inflammation/irritation of stomach mucosa with mucosal injury
- gastropathy = mucosal injury without evidence of inflammation
- protective factors: mucus, bicarb, mucosal blood flow, prostaglandins, alkaline state, hydrophobic layer, epithelial renewal
- imbalance protective factors → inflammation

History and Physical:
- imbalance between increased aggressive and decreased protective mechanisms
  - h. pylori = MC cause gastritis (gram negative spiral shaped bacillus)
    - causes type B gastritis – antrum and body of stomach
    - tolerates acidity of normal stomach and associated with peptic ulcer, gastric adenocarcinoma, gastric lymphoma
  - NSAIDs / aspirin: 2nd MC cause – disrupts mucosal protective barrier by prostaglandin inhibition (prostaglandins responsible for protecting gastric mucosa)
  - Acute stress: in critically ill patients
  - alcohol
  - MC = asymptomatic

If symptomatic → upper GI bleed, epigastric pain, nausea, vomiting, anorexia, dyspepsia, abdominal pain

Diagnostics:
- Endoscopy with biopsy = gold standard
  - Reveals severity / presence of H. pylori
    - Urea breath test also used for h. pylori
  - thick, edematous erosions <0.5cm; H pylori testing
- specific testing for underlying conditions (vitamin B12, CBC for pernicious anemia

Therapeutics:
- remove causative factor / treat underlying cause
- H. pylori positive: clarithromycin + amoxycillin + PPI (CAP); metronidazole if allergic to PCN
- H. pylori negative: PPI, antacids/H2RA, sucralfate
- Pharmacologic prophylaxis for pt high risk for stress related gastritis (IV proton pump inhibitors)

Gastroenteritis

Definition:
- MC form of salmonella infection
- Incubation period 8-48 hrs after ingestion of contaminated food / drink

History / Physical Exam:
- n/v/fever, abdominal cramping, blood diarrhea 3-5 days

Diagnostics:
Therapeutics:
- stool culture
  - self-limited
  - treat symptomatically
  - specific tx with Bactrim, ampicillin, cipro required for severely ill / malnourished pt. with sickle cell or pt who develop bacteremia

Definition:
- transient relaxation of LES \(\rightarrow\) gastric acid reflux \(\rightarrow\) 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 lung causes lung irritation
- halitosis, cough, hiccapping, 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
Gastrointestinal Bleeding

**Definition:** UGIB is classified as any blood loss from a gastrointestinal source above the ligament of Treitz.

**History and Physical:**
- Detailed review of NSAIDs, antiplatelet drugs, aspirin, anticoagulants, alcohol use
- Hematemesis (bright red emesis or coffee-ground emesis), hematochezia, or melena. Patients can also present with symptoms secondary to blood loss, such as syncopal episodes, fatigue, and weakness. UGIB can be acute, occult, or obscure
- Melena, hematemesis, coffee ground emesis, hematochezia (BRBPR = brisk UGIB)
- Syncope, orthostatic htn if severe
- Look for evidence of chronic liver dz (palmar erythema, spider angiomas, gynecomastia, jaundice, ascites) → variceal bleeding
- Cause: PUD = MC, majority is secondary to secondary ulcers, NSAIDs, H. pylori, stress related mucosal dz
- Other causes: erosive esophagitis, duodenitis, varices, mallory Weiss tear, vascular malformations
- UGIB = 75% all acute GI bleeding
- Aspirin + P2Y12 inhibitors and vitamin K antagonist – risk = 2-3x higher

**Diagnostics:**
- CBC
- Low MCV
- Elevated BUN/Cr
- Check coags
- Endoscopy within 24 hours admission or 12 hours for those high risk

**Therapeutics:**
- 2 lg bore IVs, IV fluids, protect airway
- Transfusion to keep crit about 20% or 30% in high risk pt.
- PPI for nonvariceal – 80mg bolus PPI + continuous infusion for 72hrs
- Use ocreotide when variceal bleed suspected – IV bolus 20-50mcg followed by continuous infusion
- If bleeding vessel: thermal coagulation, local injection epinephrine, clips
- Management of the patient presenting with UGIB should always follow a step-wise approach. The first step is to assess the hemodynamic status and initiate resuscitative efforts as needed (including fluids and blood transfusions). Patients should be risk stratified based on their initial presentation, hemodynamic status, comorbidities, age, and initial laboratory tests. There are several scoring systems available, with the most commonly used being the Rockall and Blatchford scores. Upper endoscopy should be offered within 24 hours to help diagnose the source of bleeding and help further guide management if needed
- Prevent recurrence of bleeding. If the patient is found to have *H. pylori*, eradication should be a target. If NSAIDs were likely the cause of the bleeding, they should be stopped, and if absolutely needed, alternative agents such as COX-2-selective NSAID plus a PPI should be used. Patients with established cardiovascular disease who require aspirin or other antiplatelet agents should be on PPI therapy and generally can have antiplatelet therapy reinstituted after bleeding ceases (ideally within 1 to 3 days and certainly within 7 days).

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
Hiatal Hernia

Definition: Involves protrusion of the stomach through the diaphragm via the esophageal hiatus

History and Physical:
- Type 1: sliding hernia → GE junction and stomach slide into mediastinum (MC)
  - Increase refluxes, treat like GERD
- Type 2: rolling hernia → fundus of stomach protrudes through diaphragm with GE junction, remaining in its anatomic location
  - Surgical repair to avoid complications

Health Maintenance:

Scientific Concepts:

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></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Onset sudden or gradual</td>
<td>Some genetic predisposition</td>
</tr>
<tr>
<td></td>
<td>Cause = unknown; M = F; 15-35</td>
<td>Onset gradual; waxes and wanes</td>
</tr>
<tr>
<td><strong>History / Physical</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Area affected</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 tenesmus, urgency</td>
<td>abdominal pain: RLQ pain (crampy); weight loss more common in Crohn’s</td>
</tr>
<tr>
<td></td>
<td>blood diarrhea hallmark (stools with mucous/pus), hematochezia MC in UC</td>
<td>diarrhea with no visible blood usually</td>
</tr>
<tr>
<td><strong>Complications</strong></td>
<td>primary sclerosing cholangitis; colon CA; toxic megacolon (more common in UC)</td>
<td>perianal dz; fistulas, stricture, abscesses, granulomas</td>
</tr>
<tr>
<td></td>
<td>smoking decreases risk for UC</td>
<td>malabsorption: B12 and iron deficiency</td>
</tr>
<tr>
<td><strong>Colonoscopy</strong></td>
<td>uniform inflammation +/- ulceration in rectum / colon = “sandpaper” appearance</td>
<td>smoking makes worse</td>
</tr>
<tr>
<td></td>
<td>skip lesions (normal areas btwn inflamed areas) with cobblestone appearance</td>
<td></td>
</tr>
<tr>
<td>Barium studies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• pseudo polyps</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• stovepipe sign (loss of haustral marking)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• string sign (Ba flow through narrowed inflamed / scarred area due to transmural stricutres)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Labs</th>
</tr>
</thead>
<tbody>
<tr>
<td>• P-ANCA (more common in UC)</td>
</tr>
<tr>
<td>• ASCA</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>curative</td>
</tr>
<tr>
<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
  - Ant9-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

### Therapeutics:
- Elemental diet
  - Crohn’s: supplement with vitamin B12, folic acid, vitamin D
  - Smoking cessation
  - Surgery not curative in crohn's; curative in UC
- Aminosaliclylates (sulfasalazine, mesalamine) → corticosteroids → immune modifying agents
  1. 5-aminosalicylates: anti-inflammatory agents; good for flares and remission
    - Oral mesalamine: especially active in terminal small bowel and colon; long acting works throughout entire small intestine and colon; best for maintenance
    - Topical mesalamine: rectal suppositories and enemas: topical are effective in distal colon
    - Sulfasaline: works primarily in the colon; s/e: higher side effect profile with sulfasalazine (heptatitis, 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 methotrexare = steroid sparing
  4. Anti-tnf drugs: inhibits proinflammatory cytokines (-mab)

### Scientific Concepts:
- Etiology = idiopathic (most likely immune reaction to GI tract flora); MC in Caucasians 15-35y
  
  **Irritable Bowel Syndrome**

**Definition:** chronic, idiopathic functional disorder without known pathology; combination of altered motility, hypersensitivity to intestinal distention; psychological distress – intermittent, lifelong problem; sx begin early / late adulthood
- MC cause of chronic / recurrent abdominal pain in US

### History and Physical:
- Abdominal pain with altered defecation / bowel habits
- Abdominal pain anywhere or localized to hypogastrium / LLQ
  - May be worsened by food intake, relieved with defecation
- Bowel distention form accumulation of gas and spasm of smooth muscle; postprandial urgency = common
o Physical exam: generally normal; may include tender, palpable sigmoid colon and hyperresonance on percussion over abdomen
o Constipation, diarrhea, alternation constipation and diarrhea, dyspepsia, urinary frequency and urgency in F
o Alarm symptoms:
  o evidence of GI bleeding: occult blood in stool, rectal bleeding, anemia
  o Anorexia or weight loss, fever, nocturnal symptoms, family hx Gl cancer, IBD, celiac sprue
  o Persistent diarrhoea causing dehydration, severe constipation, fecal impaction; onset >45y

Diagnostics:
  o Diagnosis of exclusion; lab findings usually normal – test stool for blood, bacteria, parasites, lactose intolerance
  o Colonoscopy, barium enema, US, CT to r/o pathology
  o Endoscopic studies for those with persistent symptoms, weight loss, anorexia, bleeding, hx of GI pathology
  o Differential: lactose intolerance, cholecystitis, chronic pancreatitis, intestinal obstruction, chronic peritonitis, celiac disease, carcinoma of pancreas or stomach
  o Diagnosis based on Rome IV criterial
    o recurrent abdominal pain at least 1 day per week in the last 3 months on average, associated with ≥2 of the criteria below. The criteria are fulfilled with symptoms onset 6 months prior to diagnosis.
      ▪ Increasing or improving pain with defecation
      ▪ Change in stool frequency
      ▪ Change in stool form (appearance)

Therapeutics:
  o Lifestyle changes
    o Smoking cessation, eat low fat/ unprocessed foods, avoid beverages with sorbitol / fructose, avoid cruciferous vegetables, sleep, exercise
    o Reassurance, avoid triggers
    o High fiber diet, bulking agents (psyllium hydrophilic mucillloid = mainstays of treatment)
  o Diarrhea sx: anticholinergics / spasm, anti diarrhoea
  o Constipation: prokinetics, bulk forming laxatives, saline, osmotic laxatives
  o TCA (amitriptyline) and serotonin receptor agonists for intractable pain
  o antidepressants can be used if indicated

Scientific Concepts:
  o Abnormal motility, visceral hypersensitivity, psychosocial interactions = pathophysiology

Definition:
  o Yelloeing of skin, nail beds, sclera by bilirubin deposition as consequence of hyperbilirubinemia; not a disease but sign of disease
  o Occurs with increased bilirubin overproduction (hemolysis), decreased hepatic bilirubin uptake, impaired conjugation, biliary obstruction, hepatitis

History and Physical:
  o Hemolytic = prehepatic
    o Increased indirect / unconjugated bilirubin, mild hyperbilirubinemia
    o Dark urine due to hemoglobinuria; dark stool
  o Obstructive = post hepatic
    o Cholestasis = bile duct blockage → increased conjugated bili
    o Ex. Cholestasis / pancreatic CA
    o Increased direct/conjugated hyperbilirubinemia
    o GGT and ALP elevated
    o Dark urine = increase direct bilirubin
    o Acholic stools = biliary obstruction (white)
  o Hepatocellular (intra hepatic)
    o Increased indirect and direct bilirubin; ALT and AST markedly elevated
    o Dark urine = increased direct bilirubin
    o 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 > 2.5 mg/dL
- Increased bilirubin without increased LFTs = suspected familial bilirubin disorders (gilbert’s, dubin-johnsons) and hemolysis

Therapeutics:

Health Maintenance:

Scientific Concepts:

Pancreatitis

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 > 130 bpm \( \rightarrow \) 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 (analgesis and Demerol) consider abx but not used prophylactically – when necessary, broad spectrum imipenem (nectroziing 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

Health Maintenance:
- STOP DRINKING

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 post 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; Cl in premenopausal women bc abortifactent
  - 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

Viral Hepatitis

Definition:

History and Physical Exam:
- 1. Prodromal phase: malaise, arthralgia, fatigue, URI sx, anorexia, decreased smoking
  - hep A = spiking fevers
- 2. Icteric phase: jaundice
- Chronic hepatitis: dz >6months (only HBV, HCV, HDV assoc with chronic liver dz); may → ESLD or HCC
- Fulminant: encephalopathy, coagulopathy, jaundice, edema, ascites, asterixis, hyperreflexia

Diagnostic:
- Lab: increased ALT > AST > 500; <500 if chronic

Hep A:
- Feco-oral; international travel 40%
- Prodomal: decreased smoking; only one associated with spiking fevers
- Icteric: jaundice
- Diagnosis: IgM HAV ab
- Management: self-limiting
- Post exposure prophylaxis: HAV immune globulin

Hep C:
- Parenteral (IVDU) > sexual; increased risk of blood transfusion before 1992; 80% patients develop chronic infection
- Diagnosis: anti HCV positive in 6 weeks does not imply recovery; HCV-RNA
- Management: pegylated interferon alpha-2b and ribavirin s/e: psychosis and depression

Hep B:
- Perinatal, percutaneous, sexual, parenteral
  - Acute
Chronic asymptomatic carrier: most infected as asymptomatic

Testing:
- HBsAG: 1st evidence HBV infection before symptoms - if positive for 6 mo = chronic infection
- HBsAb: 1. distant resolved infection OR 2. Vaccination – it pt. doesn’t develop 6 mo = chronic
- HBcAb
  - IgM: acute infection (1st ab to appear)
  - IgG: chronic infection or distant resolved infection
- HBeAg: increased viral replication and infectivity
- HBeAb: waning viral replication and decreased infectivity
- HBV DNA: presence in serum correlated with active replication in liver

Management:
- Acute: supportive
- Chronic: HBsAg, HBV DNA – treat if increased ALT, inflammation on biopsy or HBeAg – alpha interferon 2b, lamivudine, adefovir
- Vaccine contraindicated if allergic to baker’s yeast – give at 0, 1, 6 mo

Hep D (need Hep B)
- More severe hepatitis, faster progression to cirrhosis

Hep E (feco-oral)
- Similar to hep A, associated with waterborne outbreaks, self-limiting
- Diagnosis: IgM anti-HEV
- Highest mortality during pregnancy – fulminant hepatitis esp during 3rd trimester

ENT

Acute / chronic sinusitis
- Definition: often follows URI – can be viral or bacterial (bacterial = s. pneumo, h. flu)
- r/f: cigarette smoke / exposure, history of trauma, presence of foreign body
- symptoms: purulent nasal discharge, facial pain and pressure, nasal obstruction, fever
- p/e: tenderness to palpation over affected sinus, decreased light transmission with transillumination
- dx: clinical, XR not recommended, MRI indicated if malignancy or intracranial spread of infection is suspected
- tx: NSAIIDs for pain, saline washes, steam, oral/nasal decongestents – improvement in 2 weeks
  - abx recommended with extended duration (10-14 days) or more severe symptoms → amoxicillin (or doxy with allergy) is first line 7-10 days; augmentin if no improvement after 3 days of amox x10days; quinolones used with tx failures or recent abx use

Allergic Rhinitis
- symptoms: allergic shiners (bluish discoloration below the eyes), rhinorrhea, itchy or atery eyes, sneezing, nasal congestion, dry cough, pale, boggy, bluish mucosa, children = allergic salute
- tx: avoid known allergens and use antihistamines, cromolyn sodium, nasal or systemic corticosteroids, nasal saline drops or washes, and immunotherapy

Aphthous ulcers
- aka canker sores, ulcerative stomatitis; etiology unclear, may be associated with HHV 6
- features: single or multiple painful, round ulcers with yellow-gray centers and red halos; occur on nonkeratinized mucosa (buccal / labial) and are usually recurrent
- tx: nonspecific; topical corticosteroids can provide relief; 1 week oral pred taper helpful; cimetidine used for maintenance therapy

Blepharitis
- chronic inflammation of lid margins caused by seborrhea, staph, or strep / dysfunction of Meibomian glands
- anterior blepharitis: eyelid skin, eyelashes; may be ulcerative (s.aureus) or seborrheic
- posterior: inflammation of Meibomian; may be infectious (s. aureus) or caused by glandular dysfunction
- features: red rims, scales, adherent eyelashes, hyperemic lid margins, dandruff like deposits (scurf) and fibrous scales (collarettes); clear or slightly injected conjunctiva; thick cloudy discharge visible when Meibomian glands obstructed
o tx: lid scrubs with diluted baby shampoo on cotton tipped swabs; massage to express the gland; topical abx used if infection suspected

Cholesteatoma
o clinical features: squamous epithelium that is trapped within the skull base that can erode and destroy important structures within the temporal bone.
o s/s: painless draining, conductive hearing loss, dizziness
o dx: physical exam
o tx: excise

Conjunctivitis

Viral
o MC caused by adenovirus; highly contagious, transmission via direct contact / swimming pools
o Features: acute onset unilateral or bilateral erythema of conjunctiva, copious watery discharge, tender preauricular lymphadenopathy
o Tx: eye lavage with normal saline bid 7-14 days; antihistamine drops, warm to cool compresses,

Bacterial
o Pathogens: s. pneu, s. aureus = common, chlamydia / gonorrhoeae = rare
o Transmission via direct contact or fomites / swimming pools / autoinoculation
o Features: acute onset purulent discharge from both eyes; “glued” shut in the morning;
o Tx: hand washing, avoid contamination
o Topical abx: sulfonamids, FQs, aminoglycosides = drops > ointment
  o Add systemic abx for rare pathogens

Corneal Abrasion
o Usually caused by minor trauma (fingernail, contact lens, eyelash, small foreign body
o Features: pain and sensation of foreign body + photophobia, tearing, injection, blepharospasm, blurred vision
o Dx: slit lamp with fluorescein → epithelial defect but clear cornea
o Tx: topical anesthetic but ONLY to assist in confirming dx (don’t prescribe!!! Delayed healing!!!), saline irrigation, antibiotic ointment (gentamicin or sulfacetamide), Tylenol for pain
  o Patching for no longer than 24 hours recommended for large abrasions (>5-10mm); refer if not healing

Dacrocystitis
o Inflammation of the lacrimal sac caused by obstruction (s. aureus, B-hemolytic strep, s. epidermidis, candida)
o Sx: pain, swelling, tenderness, redness, tearing, +/- purulent discharge
o Tx: warm compresses and systemic abx
o If abscess forms → I&D

Entropion / Ectropion
o Entropion: lid and lashes are turned in secondary to scar tissue or spasm of orbicularis oculi muscles
o Ectropion: edge of eyelid everts secondary to advanced age, trauma, infection, facial nerve palsy
o Tx: surgical repair if the condition causes trauma, excessive tearing, exposure keratitis, cosmetic distress

Epistaxis
o Features: MC occurs anteirior from kiesselbach plexus / usually anterior, posterior bleed = less frequent (woodruff plexus)
o r/f: nasal trauma, dryness, HTN, nasal cocaine, alcohol
o tx:
  o anterior: direct pressure; sitting position while leaning forward for 15 minutes; topical decongestant and topical anesthetics can be used as vasoconstrictor or topical cocaine; cauterize if visualize bleeding source or anterior packing
  o posterior: high risk complications – specialist eval and inpatient monitoring; nasal arterial supply ligation via surgery in some cases

Glaucoma
o increased IOP with optic nerve damage; impediment to the flow of aqueous humor thorugh trabecular meshwork; canal of schlemm with increase pressure in anterior chamber
o open-angle = more common - >40yo, African americans + family history = more common
o angle-closure glaucoma: OPHTHALMIC EMERGENCY – COMPLETE CLOSURE OF ANGLE
**Hordeolum**

- **sx:** small, mildly painful module or pustule within gland of upper/lower eyelid
- **types:**
  - internal – inflammation and infection of Meibomian gland with pustula formation in that gland
  - external – inflammation and infection of glands of Moll or Zeis, with pustular formation of those glands
- **MC:** caused by Staph aureus
- **s/s:** acute onset pain and edema of eyelid; palpable indurated area in involved eyelid
- **tx:** warm compress several times per day; topical abx if secondary infection develops; I and D if doesn’t resolve in 48 hrs

**Hyphema**

- **features:** pooling or collection of blood inside the anterior chamber of the eye (the space between the cornea and the iris). The blood may cover most or all of the iris and the pupil, blocking vision partially or completely; usually painful
- **s/s:** visible blood in front of eye, sensitivity to light, pain, blurry/clouded vision, blood might not be visible if hyphemia is small
- **causes:** eye trauma = MC, eye infection (herpes), blood clotting problems, intraocular lens problem, eye cancer
- **tx:** steroid drops, patch, bed rest, limited eye movement, check eye pressure daily

**Labrynthitis**

- **features:** acute severe vertigo, hearing loss (several days to a week); vertigo progressively improves, hearing loss may not resolve
- **tx:**
  - abx indicated with fever or signs of bacterial infection
  - vestibular suppressants (lorazepam, clonazepam) are helpful during initial acute symptoms

**Laryngitis**

- **clinical features:** usually viral and follows a URI; bacterial: *m. catarrhalis, h. flu*
- **tx:** supportive is usually sufficient – vocal rest, avoid singing/shouting
  - bacterial: erythromycin, augmentin to decrease hoarseness/cough
  - oral or IM corticosteroids may also hasten recovery

**Macular Degeneration**

- **sx:** may be age related or secondary to toxic effects of drugs (chloroquine or phenothiazine); increases after 50 years
- **p/e:** drusen deposits accumulate in Bruch membrane → degenerative changes/atrophy (dry macular degeneration) / neovascular degeneration → hemorrhage and fibrosis (wet macular degeneration)
- **s/s:** insidious onset and chief clinical feature is gradual loss of central vision
- **metamorphopsia:** wavy or distorted vision, measured with Amsler grid
- **mottling, serous leaks, hemorrhages may be seen on retina
- **tx:** no effective treatment → laser therapy or intravitreal injections of vascular endothelial growth factor inhibitors
- **slow progression of wet**

**Meniere Disease** aka endolymphatic hydrops

- **sx:** unknown etiology; related to distension of inner ear’s endolymphatic compartment
- **s/s:** recurrent vertigo, vertigo (lasting minutes to hours), lower range hearing loss, tinnitus, one sided aural pressure
Nystagmus lost on impaired side

Osmotic: low sodium diet and diuretics, unresponsive → intratympanic corticosteroid therapy, surgery

- Pale, boggy masses on nasal mucosa, allergic rhinitis
- Hx of nasal polyps + asthma - aspirin = contraindicated possibly causing bronchospasm (triad asthma – Samter triad)
- Chronic congestion and decreased sense of smell
- Tx: 3 month course of topical nasal steroid = initial treatment choice; oral steroids also help reduce size; surgery if therapy unsuccessful / large polyps

Nasal Polyps

Otitis Externa

- Aka “swimmer’s ear” – water exposure, trauma, exfoliative skin conditions – pseudomonas, proteus, fungi
- S/s: ear pain, redness / swelling of ear canal, purulent exudate
- Tx: abx otic drops (aminoglycoside or fluoroquinolone +/- corticosteroids) + avoid moisture
  - Diabetic / immunocompromised: malignancy otitis externa → necrotizing infection → hospitalization with IV abx

Otitis Media

- Acute:
  - Viral URI → eustachian tube dysfunction / blockage; most common in infants; s.pneumo, h.flu, Moraxella, s. pyogenes
  - S/s: fever, ear pain, ear pressure, hearing impairment; TM erythema / limited mobility; bulging / rupture of TM
  - Tx: watchful waiting for older children
    - First line abx: amoxicillin, OR cephalosporin, Bactrim, zpack
    - Recurring: tympanostomy, tympanocentesis, myringotomy
- Chronic: repeated episodes of acute otitis media, trauma, cholesteatoma
  - Organisms: pseudomonas, s. aureus, proteus, anerobies
  - S/s: perforated TM and chronic ear discharge +/- pain, conductive hearing loss
  - Tx: removal of infected debris, avoid water exposure, topical abx drops; surgery (TM repair)

Papilledema

- D/t: increase in intracranial pressure from malignany hypertension, hemorrhagic strokes, acute subdural hematoma, pseudotumor cerbri
- P/e: swollen disc, blurred margins, oliteration of vessels
- S/s: may be asymptomatic or complain of transient visual alteration that last for seconds
- Tx: treat underlying cause

Parotitis

- Acute bacterial suppurative parotitis is caused most commonly by Staphylococcus aureus and mixed oral aerobes and/or anaerobes. It often occurs in the setting of debilitation, dehydration, and poor oral hygiene, particularly among elderly postoperative patients.
- Viral parotitis can be caused by paramyxovirus (mumps), Epstein-Barr virus, coxsackievirus, and influenza A and parainfluenza viruses. Acute suppurative parotitis is generally caused by Staphylococcus aureus, Streptococcus species, and rarely, gram-negative bacteria.
- Presentation: recurrent episodes of acute or subacute parotid gland swelling with fever, malaise, and pain; disorder is usually unilateral, but can affect both sides. Episodes may last days to weeks and occur every few months
- Dx:
- Tx:
  - Supportive care with adequate hydration, gland massage, warm compresses, sialagogues, and antibiotics
  - Antibiotics should be administered intravenously in acute bacterial parotitis after obtaining blood cultures. Staphylococcus aureus is the most common organism in community-acquired parotitis and first-line antibiotic therapy should include antistaphylococcal antibiotic (nafcillin, oxacillin, cefazolin). Surgical drainage may be necessary when pus forms

Peritonsillar abscess

- Results from penetration of infection through tonsillar capsule and involvement of neighboring tissue
- Sx: hot potato voice, sore throat, pain with swallowing, trismus, deviation of soft palate / uvula
- Tx: aspiration, I&D, abx; tonsillectomy in 1-% of patients
  - Amox, clindamycin IV OR in less severe cases oral for 7-10 days
**Pharyngitis / Tonsillitis**

- **Sore throat; viral > bacterial**
- **sx:**
  - GABHS = fever, tender anterior cervical adenopathy, no cough, pharyngotonsillar exudate
  - Presence of all four (centor) = likely GABHS; ¼, rapid strep test >90% positive; only one – GABHS not likely
  - Coryza, hoarseness, cough = not suggestive of strep
- **dx:** rapid strep has 90-99% sensitivity; if negative and still suspected, throat culture is confirmatory
- **tx:** IM penicillin if doubt pt. compliance; oral penicillin / cefuroxime; erythromycin with allergy
  - Inadequate tx → scarlet fever, glomerulonephritis, acute rheumatic fever, abscess formation

**Pterygium**

- **Definition:** slowly growing thickening of the bulbar conjunctiva; unilateral or bilateral
- **Features:** highly vascular, triangular mass groing form nasal side toward cornea; eventually encroaches on the cornea and interferes with vision
- **Tx:** excision if interferes with vision; recurrence common and more aggressive

**Retinal Detachment**

- **General:** separation of retina from pigmented epithelial layer; commonly begins at superior temporal retinal area; tear can happen spontaneously or be secondary to trauma, extreme myopia, or inflammatory changes in the vitreous, retina, or choroid
- **Features:** acute onset painless blurred or blackened vision that occurs over several minutes to hours and progresses to complete or partial monocular blindness; curtain drawn over th eye from top to bottom; floaters or flashing lights at initiation of symptoms; IOP is normal or reduced; RAPD will be present
- **Tx:** emergency consult with ophthalmologist re: laser surgery or cryosurgery
  - Remain supine; with head turned to the side of the retinal detachment; 80% recover without recurrence

**Central Retinal Artery Occlusion**

- **OPHTHALMIC EMERGENCY** – poor prognosis; caused by: emboli, thrombotic phenomenon, vasculitides
  - Differentiate from giant cell arteritis (fever, he/a, scalp tenderness, jaw claudication, visual loss)
- **S/s:** sudden, painless, marked unilateral vision loss
- **Exam:** fundoscopy sws pallor of retina, arteriolar narrowing, separation of arterial flow, perifoveal atrophy (cherry red spot)
  - Ganglionic death → optic atrophy and a pale retina = blindness
- **Tx:** emergency referral to ophthalmologist; recumbent position and gentle ocular massage may help reduce damage
  - Workup for atherosclerotic disease or arrhythmias to reduce recurrence

**Central Retinal Vein Occlusion**

- Usually occurs secondary to thrombotic event
- **R/f:** diabetes, hyperlipidemia, glaucoma, hyperviscosity states (polycythemia, leukemia)
- **S/s:** sudden, unilateral painless blurred vision or complete visual loss
- **Exam:** optic disc swelling; blood and thunder retina (dilated veins, hemorrhages, edema, exudates)
- Vision resolved with time (partially); workup for thrombosis
- **Tx:** neovascularization treated with intravitreal injection of VEGF inhibitors

**Retinopathy**

- **Hypertensive:** acute or accelerated HTN = greatest risk; s/s = arteriolar narrowing, copper or silver wiring, arteriovenous nicking
- **Diabetic:** leading cause of blindness in adults in US; diabetics need yearly ophthalmoscopic exams
  - Noproliferative: venous dilation, microaneurysms, retinal hemorrhages, retinal edema, hard exudates
  - Proliferative: neovascularization, vitreous hemorrhage
- **Tx:** optimized glucose control, regulate blood pressure, latser photocoagulation, vitrectomy; severe dz = permanent

**Sialadenitis**

- **Sialadenitis** is an infection of the salivary glands. It is usually caused by a virus or bacteria; Sialadenitis most commonly affects the elderly and chronically ill especially those with dry mouth or who are dehydrated
- The parotid (in front of the ear) and submandibular (under the chin) glands are most commonly affected.
- **S/s:** pain, tenderness, redness, and gradual, localized swelling of the affected area.
Dx: usually made by clinical exam but a CT scan, MRI scan or ultrasound may be done if the doctor suspects an abscess or to look for stones.

Tx:
- warm compresses, gland massage, drink plenty of fluids and eat or drink things that trigger saliva flow (such as lemon juice or hard candy), increasing fluid intake and good oral hygiene. Most salivary gland infections go away on their own or are cured with treatment. Complications are not common.
- antibiotics active against *S. aureus* – dicloxacilling, clinda, 1st gen cephalosporing, modify to culture results

Tinnitus

Tinnitus is one of the side effects of nonsteroidal anti-inflammatory drugs in nearly every case. However, as with aspirin, this effect seems to be dose related. Diuretics (e.g., ethacrynic acid, furosemide) may also produce a dose-related tinnitus. This particular effect may be reversible with these drugs, but it may be permanent in others.

Because tinnitus is associated with depression, seek signs of the condition. A careful assessment of the mental status of patients is an essential part of the initial history.

Treatment:
- Surgery - tinnitus due to a surgical lesion in the ear usually responds to treatment of that lesion. Typical lesions amenable to surgery include those caused by glomus tumors, sigmoid sinus diverticulum, arteriovenous malformation, and conductive hearing loss.
- Alternative: electrical stimulation, biofeedback, Transcranial magnetic stimulation (TMS)
- Pharmacologic therapy helps in the treatment of tinnitus for the 80% of patients who endure related depression. Administration of nortriptyline (50 mg at bedtime) is the most helpful treatment. Nortriptyline may induce dry mouth, often causing patients to terminate treatment before achieving therapeutic effect. Often, 3-4 weeks of therapy are necessary before benefits appear. Other antidepressants may be useful in treating tinnitus, but judgment in their use is paramount. Selective serotonin reuptake inhibitors are considered to have a better safety profile compared with tricyclic antidepressants. Paroxetine (Paxil) in low doses of 10 mg at bedtime has recently been shown to be helpful. Also, sertraline (Zoloft), at a fixed dose of 50 mg/d, demonstrated a significant reduction in tinnitus severity, as well as a reduction in anxiety and depressive symptoms.

Acoustic neuroma: CN VIII, hearing loss + tinnitus + disequilibrium

Ménière disease: recurrent vertigo + tinnitus + hearing loss

Ramsay Hunt syndrome: facial paralysis, zoster lesions, tinnitus

Labyrinthitis: sudden severe vertigo, hearing loss, tinnitus, not recurrent

Head trauma

Electrical injury

Diving

Otoxic agents:
- Salicylates: respiratory alkalosis + anion gap metabolic acidosis + tinnitus
- NSAIDs
- Quinine
- ABX (aminoglycosides, erythromycin, vancomycin)
- Chemotherapeutic agents

Tymppanic Membrane Perforation

- can occur from infection (acute otitis media) or trauma (barotrauma, direct impact, explosion)
- usually resolve on own; surgical repair may be necessary with persistent hearing loss
- water/moisture to the ear should be avoided to prevent secondary infection that impedes closure

**OBSTETRICS / GYNECOLOGY**

**Breast cancer**

- Epi: >1M new cases p/yr; MC cancer in females (1 in 8 women by age 80)
- RF: inherited → BRCA1 and BRCA2 (55-85% incr risk), Increasing age, Previous breast CA, FHx in two close relatives, Radiation exposure, Nulliparity or first birth at age ≥35, North American/northern European, Early menarche, late menopause, Obesity, Urban residence, upper SES, primary cancer in ovary/endometrium
- Screening: Mammogram q2 years ages 50-74
Screening guidelines:
- HPV screening: looks for 13ish of the HR HPV types (16 and 18 included)
- Pap Smear: sample of superficial epithelial cells from cervix
  Screening for cervical CA
  - if Pap returns as high grade change, pt will get a colpo (don’t need reflux HPV)
  - if Pap returns as low grade dysplasia/atypical results
  - Severe dysplasia: CIN3, High grade squamous intraepithelial lesion
  - Carcinoma in situ: HSIL

Cervical Cancer

1. Pap Smear: sample of superficial epithelial cells from cervix – fixed and evaluated by cytopathologist for any abnormal cell growth/dysplasia
   - want to sample from the SCJ \( \rightarrow \) rotate 5x and put in liquid solution
   - can do conventional cytology (rub on slide) or liquid base cytology (more accurate and then have sample for HPV if needed)
2. HPV screening: looks for 13ish of the HR HPV types (16 and 18 included)
   - can be done as a co-test with pap or as a primary screen (US usually does pap then reflex HPV)
   - HPV test may be + with no cell change (body likely to clear it)
   - involves doing a viral screen on the fluid from the pap test
   - doesn’t come back with the type of virus, but just if there is a HR type present

Screening for cervical CA

- Location: SCJ moves caudally with age \( \rightarrow \) area beween old SCJ and new SCJ is the “transformation zone” and this is where most cervical CA occurs; pap should include endocervical cells; if it doesn’t, get a repeat one in a year or so to qualify it as satisfactory

Pap results:
- HPV effect: low grade squamous intraepithelial lesion (LSIL)
- Mild dysplasia: CIN1; LSIL
- Moderate dysplasia: CIN2; LSIL
- Severe dysplasia: CIN3, High grade squamous intraepithelial lesion
- Carcinoma in situ: HSIL
Colposcopy: done for any high grade cervical changes - magnification and illumination to help visual inspection of the cervix/vagina/anogenital area, solutions used to highlight area of concern (usually acetic acid) → raised areas, sharp boarders, white and coarse areas (these are most concerning); if the area is red/yellow and peeling/rolling – this is most concerning for invasive CA

Tx (Preinvasive – CIN 1, 2, 3)
1. CIN 1
   - Follow up with cytology and HPV testing in 12 mos. – if negative, resume routine age-based testing
   - never tx at this stage bc immune system is likely to clear it
2. CIN 2:
   - Young women: observe w/cytology q6 mos; colpo recommended
   - may tx with excision/ablation – many women are in childbearing years at this stage, so f/u may be the only recommended thing
3. Tx of CIN 2/3: excision or ablation
   - recommend tx if done w/ child bearing or if dx is CIN 3
   - LEEP procedure: Loop electrical excision procedure – done in office w/local anesthesia; very little blood loss – can then analyze what you excise

Ablation: doesn’t allow you to further analyze the tissue

Invasive Cervical CA

Sxs: bleeding (often post-coital), late signs are back pain, anorexia, wt. loss
RF: hx of inadequate screening
Natural hx: spreads directly; late stage can spread to pelvic lymph nodes
- can be exophytic (protrusion) or endophytic (deep invasion)
- Staging depends on pelvic exam and is modified by further imaging – we don’t stage operatively

Staging: most important prognostic factor
1. confined to cervix – ~ 80-95% 5 yr survival
2. beyond cervix, upper 2/3 of vagina – 64% 5 yr survival
3. lower 1/3 vagina or pelvic sidewall – 38% 5 year survival
4. adjacent/distant organs – 14% 5 year survival

Tx: radical hysterectomy (take everything at the root) – only early stage

Late stage gets chemo/radiation (brachytherapy an option where is localized to uterus/vagina/cervix)

Cervical CA prevention

HPV prevention: abstinence/delay sexual activity, less sexual partners,
Vaccination: yeast + capsid protein ➔ basically give the virus w/no
- protects against 7 HR types (including 16/18) and against 6 and 11
warts
- ideal age is 11-12 yo bc immune system is most robust and they haven’t been exposed yet
- encourage vaccination at any patient visit

Barrier methods: items that physically prevent sperm from reaching egg
- Condoms - only method that work to prevent pregnancy and STIs; perfect use: ~98% effective
- Diaphragms: low cost and low SE profile but not super reliable ; insert <6 hours before sex w/spermicide and remove >6 hours (but <24) after sex, no STI protection

Periodic abstinence: AKA “Natural Family Planning”
- accounts for the sxs of fertile phase, viability of sperm in GYN tract (2-7 days), and lifespan of ovum (1-3 days)
- typically requires about 17 days of abstinence/protection per cycle (➔ high failure rates)
- Can be very effective if pts are adherent

Hormonal Contraception

Hormones
- Estrogens: estradiol + Ethinyl (prevents inactivation when given orally)- Dosing: 10/20/30/35/50 mcg ➔ 20-35mcg are the mainstay doses
  ➔ may increase if pt is having breakthrough bleeding
- Progestins: derived from Testosterone
  - provide negative feedback to hypothalamus/anterior pituitary to decrease FSH and LH which inhibits follicle development/ovulation
  - Progesterone also thickens cervical mucous, thins endometrium, and decreases tubal peristalsis
- Combined:
  - Estrogen has more of an effect on FSH and Progesterone has more of an effect LH
  - Progesterone can be used alone because it works to prevent ovulation
  - Estrogen helps prevent cyst growth ➔ can be used to protect a remaining ovary in a patient who only has one
  - Bc progesterone decreases tubal peristalsis, if a pt does become pregnant on bc, it has higher chance of being ectopic
  - 3 weeks of hormonal pills and 4-7 days of placebo pills
- Transdermal patch: wear weekly for 3 weeks, then 1 week off; 2x the risk of VTE (unclear why)
Transvaginal Ring: insert vaginally, leave in 3 weeks, 1 week off

Progestosterone-only methods: these will always be “safer” than CHCs

Efficacy and Safety - LARC methods shown to be 20x more effective than OCPs/patch/ring - Women tend to continue LARC methods longer than non-LARC methods

- Injectable contraception: Depot-medroxyprogesterone acetate (DMPA)/Depo-Provera; IM injection q3mos; Highest assc w/weight gain and decrease in BMD; -Can disrupt ovulation/menses after cessation for up to one year
  - BMD loss is a black box warning buuuut...not associated with an increased risk of fracture and once you stop DMPA, the density loss is reversible (ACOG says its okay to continue for >2 years) – studies show osteopenia based on pts having lower density than others in their age group
- Implant (Nexplanon): lasts 4 years - statistically the most effective BC method (> than sterilization) - may cause light, irregular bleeding for first 3-12 mos.
- Levonorgestrel IUD: thickens cervical mucus, partially inhibit ovulation, and thins endometrium; approved for 3-7 years (depending on type); r/f for expulsion: prior expulsion, hx of menorrhagia or severe dysmenorrhea, postpartum / post-second trimester abortion, <25 yo (3-10% chance expulsion)

Non-Hormonal Methods
- Copper IUD: release free copper ions which creates inflammatory response and makes the intrauterine environment very spermicidal; approved for 12 years; copper does degrade over time

Sterilization
- Male: vasectomy; Female: tubal ligation, assure, etc.
  - Male sterilization has advantage of no general anesthesia and lower rates of failure
- Essure: polyester coil placed in proximal tube hysterscopically
  - stimulates xrn causing fibrosis and occlusion (original intention was to keep tube patent)
  - repeat HSG to look for occlusion—has ~99% effective rate
  - has fallen out of favor d/t massive law suits for chronic pain
- Salpingectomy: removal of fallopian tubes
  - has increased in popularity
  - thought to be protective against ovarian CA

Emergency Contraception
1. Plan B (levonorgestrel) - Progestosterone surge blocks LH surge to inhibit ovulation → doesn’t do anything if ovulation has already occurred - can take w/in 72 hours of UPI; reduces risk of pregnancy by ~75%; not harmful to embryo if conception has occurred
2. Ella (Ulipristal Acetate) - Anti-progesterone agent → inhibits ovulation and makes the endometrium inhospitable to implantation - take w/in 5 days of UPI - More effective than plan B (98%) - Theoretical risk to pregnancy if already implanted (but would have to take >1 pill to cause harm)
3. Copper IUD - Can be used as EC if placed within 5 days of UPI → most effective form of EC

*Absolute Contraindications to EE use*
- Thromboembolic d/o
- known/suspected breast CA
- Smokers >35 y.o.
- Uncontrolled HTN
- Migraine w/aura
- SLE w/antiphospholipid ab’s (also shouldn’t get Progesterone – Copper IUD is best choice)

Cystocele
- Definition: Damage to the anterior vaginal wall pubocervical fascia can result in herniation of the bladder (cystocele) and/or urethra (urethrocele) into the vaginal lumen. Injuries to the endopelvic fascia of the rectovaginal septum in the posterior vaginal wall can result in herniation of the rectum (rectocele) into the vaginal lumen.
  - Cystocele is the herniation of the anterior vaginal wall, or anterior compartment prolapse, often associated with descent of the bladder.
- Symptoms: Patients with a cystocele may be asymptomatic or present with urinary symptoms such as urgency, frequency and incontinence.
- Treatment: While many women eventually choose surgical correction of the prolapse
  - first-line treatment is conservative and includes both pessary and pelvic floor muscle training.

Dysfunctional uterine bleeding

Definitions
- Menorrhagia: prolonged/heavy bleeding (>7 days or >80 mL); regular intervals
- Metrorrhagia: variable amounts of bleeding at irregular, frequent intervals
- Polymenorrhea: short intervals (<21 days)
- Oligomenorrhea: long intervals (>35 days)
**Etiology**

1. **Organic causes**
   - Reproductive tract disease → pregnancy, gestational trophoblastic disease, uterine lesions, iatrogenic causes (IUDs, contraception, HRT, psychotropic agents)
     - Uterine lesions: menorrhagia or metrorrhagia d/t increase in endometrial surface area/distortion of vasculature/having friable or inflamed surface → includes endometrial CA/sarcoma, endometrial hyperplasia, submucous fibroid, endometrial polyps, endometritis, adenomyosis
     - Systemic disease → blood dyscrasias (vW disease, prothrombin deficiency, leukemia, severe sepsis), Hypothyroidism, hyperthyroidism, cirrhosis
       - Hypothyroidism asss w/ menorrhagia or metrorrhagia; hyperthyroidism asss with oligomenorrhea and amenorrhea
       - Cirrhosis can cause excessive bleeding d/t low plt's and less metabolization of estrogens

2. **Endocrine causes** → anovulatory vs ovulatory DUB
   - Anovulatory
     - continuous production of estradiol-17 beta without corpus luteum formation and no progesterone release
     - unopposed estrogen = continuous proliferation of endometrium which eventually outgrows its blood supply and sloughs off in an irregular unpredictable pattern
   - Ovulatory
     - Mid-cycle spotting after LH surge

**Dx**
1. Rule out pregnancy
2. Med reconciliation
3. PE → thymomegaly, hepatomegaly, GU infections, GI problems (hemorrhoids), pelvic structural abnormalities (polyps, fibroids)
4. Labs → CBC, iron levels, TSH, coags
5. Eval of uterus → endometrial bx or hysteroscopy, pelvic US

**Management**
- Structural problems can be corrected surgically → D&C (therapeutic and dx), hysteroscopy, endometrial ablation, hysterectomy
- OCPs can regularize cycles
- IV estrogen can be used acutely if pt is presenting with acute hemorrhage d/t DUB
- NSAIDs reduce menstrual blood loss

**Dysmenorrhea**
- **Definition**: commonly found in those who ovulate regularly; pain usually lasts 1-2 days and is relieved by NSAIDs and OCPs
  - Assc w/ endometriosis; pain begins prior to menses
  - Pain isn’t relieved by NSAIDs and OCPs
- **Primary Dysmenorrhea**: begins w/in 6-12 mos of menarche → not caused by another disease process
  - Patho: d/t excess PG and leukotriene production at menstruation → incr uterine contraction; blood vessels are vasoconstricted = decr blood flow; ischemia from contractions can cause pain
  - Secondary dysmenorrhea involves the same symptoms but occurs in **women with a disorder** that could account for their symptoms, such as endometriosis, adenomyosis, or uterine fibroids.
  - Sxs: severe cramp that start w/menses and last 2-3 days (highest in first day, resolve by 3rd day), lower abd pain that radiates to back/thighs, h/a, nausea, diarrhea, headache, fatigue, and general malaise
  - PE: normal
  - Dx:
    - diagnosis of primary dysmenorrhea is based upon the presence of characteristic clinical features in the absence of demonstrable disease that could account for the pain
    - Secondary dysmenorrhea may present as **dysmenorrhea with onset at age 25 years or older, abnormal uterine bleeding (oligomenorrhea, menorrhagia, intermenstrual bleeding), non-midline pelvic pain, presence of dyspareunia, progression in symptom severity, and absence of nausea, vomiting, diarrhea, back pain, dizziness, or headache during menstruation**. Evaluation should exclude other causes of lower abdominal cramping pain and determine how severe the symptoms are to help guide the treatment.
  - Tx: NSAIDs = **first line**, OCPs (prevent ovulation), menstrual suppression, surgical (endometrium resection); explore for secondary dysmenorrhea

**Menopause**
- Def: cessation of menstruation for 12 mos d/t termination of ovarian follicle dvt + elevated gonadotropin levels (FSH and LH); avg onset is ~51 yrs;
- RF for early menopause include smoking and surgery (hysterectomy)
- Laboratory studies are not routine but may be helpful
  - High FSH, high estrone, low estradiol, prolactin, TSH, testosterone unchanged
- Phys: ovaries stop producing estrogen and only estrogen source is now from peripheral conversion of androgens; elevated FSH and LH d/t lack of estrogen – these drive ovarian stroma to produce androgens
- Perimenopausal period - Climacteric phase of menopause: transition pd before menopause; can begin as early as 35 but most women notice changes in their mid 40’s
- Adjunct/alternative meds:
  - Hormone replacement therapy
    - Osteoporosis (estrogen slows bone resorption)
    - Genital atrophy: lower vagina, labia, urethra, trigone are all estrogen dependent
    - Mood disturbances – fatigue, anxiety, h/a, insomnia, depression, irritability; clear link between menopause and mood isn’t established
  - Counseling, botanical agents, antidepressants

Hormone replacement therapy
- Need for HRT is based on sxs of menopause
- Pros: treats hot flashes, osteoporosis, genital atrophy, and mood disturbances; may decr risk of alzheimers, osteoarthritis, colon CA, tooth loss, skin aging
- Risks: endometrial hyperplasia/adenocarcinoma (if given w/out progesterone), incr risk of breast cancer
- SE: nausea, erratic vaginal bleeding, headaches, breast tenderness
- Regimens: Premarin (equine estrogen) on days 1-25 + Provera (days 13-25); or Premarin/estradiol/transdermal estrogen + Provera continuously
  - Compliance can be an issue bc of long term course and no immediate benefits
  - Recent concerns that HRT increases risk of breast cancer, thromboembolism and stroke
- Recent concerns that HRT has fallen out of use slightly

Adjunct/alternative meds:
-Raloxifene – SERM w/estrogen agonist effects on bones and cholesterol but antagonistic effects on breast and endometrium
- Gabapentin, paroxetine, and venlafaxine can help w/hot flashes
- Botanicals (soy products, isoflavones, St. Johns Wort, black cohosh) can help w/hot flashes and depression (potential interactions and efficacy not well known)

Pelvic Inflammatory Disease
- Definition: ascending infection of the female GYN tract
- Sxs: potentially axsymptomatic, abdominal/pelvic/LBP, abnormal vaginal discharge, inter-menstrual bleeding or post-coital bleeding, fever, N/V if severe
- PE findings: uterine/adnexal tenderness, cervical motion tenderness w/chandelier sign, mucopurulent discharge from cervix, friable cervix, fever (<1/3)
- Sequelae: chronic pelvic pain d/t adhesions, infertility d/t tubal occlusion, ectopic pregnancy d/t salpingitis, Fitz-Hugh Curtis syndrome
- RF: Age <25, Multiple partners/partners w/multiples, Hx of STD, Inconsistent condom use
- Dx: Sexually active and <= 25 OR hx of STI/multiple partners, tenderness on pelvic exam + no other etio dx and tx if these are met (err on the side of overtx d/t high incidence of adverse outcomes)
- Tx
  - Outpatient: Ceftriaxone IM x1 (or Cefoxitin IM/probenecid PO) AND Doxycycline (14 days)
    - +/- add metronidazole if BV is present
    - If not getting better in 48 hours, they should return for inpatient tx w/IV abx
  - Inpatient: 24 hours of IV abx then sent home on 14 day course
    - Cefotetan IV or Cefoxitin IV AND doxycycline (PO or IV)
    - Indications: other condition can’t be r/o, pt is pregnant, doesn’t respond to OP tx, pt has severe N/V/high fever

Rectocele
- Definition: posterior vaginal wall defect associated with anterior prolapse of the rectum; defect in the rectovaginal septum rather than the rectum.
- Sx: increased pelvic pressure or the sensation that something is “falling out” of the vagina.
  - Patients often utilize splinting (firm application of pressure on a particular anatomical location) of the vagina, perineum or rectum in order to achieve a bowel movement.
- Diagnosis: vaginal and rectal examination - bulge in the posterior vaginal area upon bearing down.
- Tx:
  - Nonsurgical management: medications for constipation to minimize straining that may make the rectocele worse / pessary placement / pelvic floor training
  - Surgical repair with a posterior colporrhaphy has an anatomic cure rate of up to 96%. Dyspareunia is a potential complication that patients should be educated about when undergoing surgical repair of a rectocele.

Spontaneous Abortion
- Termination of pregnancy before 20 weeks – occurs in 15-20% of pregnancies
- r/f: smoking, infection, maternal systemic disease, immunologic parameters, drug use
- s/s: bleeding = variable, fundus of uterus may be boggy or tender
- dx: serial hCG titers, serum progesterone, serial US to confirm; US: inappropriate development or interval growth, poorly formed/unformed fetal pole, fetal demise; blood type and Rh status necessary tests to preclude Rh sensitization in mother
- tx: empty uterus, follow up with pelvic exams, serial hCG titers, transvaginal US, dilation and curettage to ensure complete emptying of uterus; immunoglobulin administered to Rh negative women, septic/infected abortion requires complete evacuation of uterine contents, medical support, abx

### Vaginitis

1. **Bacterial vaginosis** - lack of lactobacilli = low hydrogen peroxide = high pH (>4.5); infection is polymicrobial (mostly *Gardnerella vaginalis*)
   - Sxs: thin, discharge; fishy odor
   - Dx: milky vaginal discharge, pH > 4.5, amine “whiff” test (fishy odor), clue cells (gram negative)
   - AMSEL criteria: thin, white homogenous discharge, presence of clue cells in microscopic exam (stippled epithelial cell), pH >4.5, fishy odor – must have 3/4
   - Tx: metronidazole (PO or intravaginally) OR clindamycin → recurrence is common d/t biofilm production → may need prolonged tx (6 mos).

2. **Candidiasis**: infection of the vaginal tract w/candida (MC is *albicans*)
   - Sxs: itching/burning, dyspareunia, thick white discharge, beefy red vaginal mucosa
   - Dx: wet mount → KOH and saline + microscopy; pH < 4.5 (normal); BD affirm → DNA test; Yeast cx if recurrent (look for different type of yeast
   - Tx: Fluconazole (150 mg PO x1) – if its really bad, can give another dose 72 hours later, vaginal cream (Miconazole, terconozole, clotrimazole, etc) – 7 day course typically works better
   - **Diabetes mellitus** is a predisposing factor for **recurrernt vulvovaginal candidiasis** since hyperglycemia enhances the ability of *Candida albicans* to bind to vaginal epithelial cells. The patient above should be tested for **diabetes** due to recurrent **vulvovaginal candidiasis**, especially since she has other known risks (elevated body mass index and history of hypertension). **Glycated hemoglobin** (also called A1C) is used to screen, diagnose and monitor prediabetes and diabetes. Patients with vaginitis may present with **vulvar pruritus and burning**, as well as erythema and edema of the labia majora and minora.

3. **Trichomonas**: caused by parasitic protozoan *Trichomonas Vaginalis*; can affect fertility so we want it treated well
   - Sxs: itching/burning, dyspareunia, thick white discharge, beefy red vaginal mucosa
   - Dx: saline wet mount → must look at very quickly (parasites die fast); pH >/= 4.5; BD affirm; Strawberry cervix on spec exam (only present ~10% of time but is pathognomonic
   - Tx: Metronidazole (PO only)
     - Partner tx: warrants full STI screenin, pt should be retested in 2 weeks-3 mos. To ensure successful tx (“test of cure”)

4. **Atrophic vaginitis**
   - Def: atrophy of vaginal and vulvar tissues d/t hypoestrogenic state (often seen in menopause)
   - Sxs: dryness, burning, irritation, low lubrication, pain/discomfort w/sex, urinary urgency, dysuria, recurrent UTIs
   - PE: fragile tissue, fissures, petechiae, labia minora resorption, loss of moisture and rugae and elasticity, prominent meatus, urethral eversion or prolapse
   - Dx: clinical
   - Tx: First-line therapy for symptomatic relief of vulvovaginal atrophy is with **non-hormonal vaginal moisturizers and lubricants**. If therapy does not result in symptom relief, low-dose vaginal *estradiol* (insert, ring, cream) therapy may be used if the woman has no contraindications (estrogen-dependent malignancy). Sexual activity and/or use of vaginal dilators can help maintain healthy vaginal epithelium.
   - **Prescribing a vaginal ring that contains 2 mg of estradiol** to be placed once every 3 months is an appropriate initial intervention for patients with symptoms of **post-menopausal vaginal atrophy**. A daily intravaginal estradiol tablet is also effective.

---

**ORTHOPEDICS / RHEUMATOLOGY**

### Acute and chronic lower back pain

- MC cause lower back pain = prolapsed intervertebral disk and low back strain; usually occurs within 24 hours of injury/overuse
- Features:
  - pain originating in the back and radiating down the leg = nerve irritation
MSK usually located to one region / point tenderness
- Sciatica felt in buttock, posterior thigh, posterolateral aspect of leg around lateral malleolus to lateral dorsum of foot
- Unilateral low back and butt pain that gets worse with standing → SI joint involvement
- Pain in elderly increased by walking and relieved by leaning forward = spinal stenosis

Dx: XR usually not required if h&p is benign / Normal neuro exam
- Red flags: fever, weight loss, morning stiffness, IVDU / steroid history, trauma, cancer, saddles anesthesia, loss of anal sphincter tone, motor weakness = emergent XR
- CT helpful in demonstrating bony stenosis and identifying lateral nerve root entrapment
- MRI helpful for cord pathology, neural tumors, stenosis, herniated disks, and infections
- Get XR if pain persists

TX: short term rest (max 2 days), with support under knees and neck + NSAIDs
- Progressive walking to normal activities if pain subsides
- Postural exercises / back rehab
- No improvement in 6 weeks → imaging to r/o spinal tumor / infection (if normal → rehab)
- Surgery if conservative tx fails (~5%)

Bursitis
- Inflammation of a bursa (thin walled sac lined with synovial tissue); caused by trauma / overuse
- Pain, swelling, tenderness – may persist weeks
- Tx: prevention of precipitating factors, rest, brace/support, NSAIDs, steroid injections

Tendonitis
- Inflammation of the tendon commonly d/t overuse injuries and systemic disease (arthritis)
- Features: pain with movement, swelling, impaired function; resolves over several weeks but recurrence common
- Tx: ice, rest, stretching for inflammation
  - NSAIDs help but don’t penetrate tendon circulation; steroid injection + anesthesia may be beneficial
  - Surgery for excision of scar tissue / necrotic debris if conservative measures fail

Costochondritis
- Inflammation of cartilage in the rib cage
- s/s: pain to the touch, radiating pain down limbs, unbearable chest pain with/without inspiration
- r/f: age >40, high-impact sports, manual labor, allergies, rheumatoid arthritis, ankylosing spondylitis, reactive arthritis
- dx: physical exam (rule out other things with imaging / blood tests / ECG)
- tx: NSAIDs, lifestyle changes, RICE

Fibromyalgia
- s/s: central pain disorder; non-articular MSK aches, pains, fatigue, sleep disturbance, mood changes, cognitive disturbance, multiple tender “trigger points”, anxiety, depression, dysmenorrhea, IBS
- dx: exclusion of underlying disease – hypothyroid, hep C, vitamin D deficiency; no lab studies (dx of exclusion)
- tx: SSRIs, SNRIs, TCAs helpful; NSAIDs not helpful
  - pregabalin/ gabapentin for reducing pain and improving sleep
  - exercise improves functioning; avoid overtraining
  - CBT and mindfulness also helpful; patient education, stress reduction, sleep assistance & treat psych conditions

Ganglion Cysts
- Most common soft tissue tumor of the hand; arise from torn or degenerated joint capsule or tendon
- Wrist and finger = MC locations
- MC caused by repetitive activity
- commonly seen at the dorsum of the wrist at the scapholunate joint, but may occur at any joint or tendon sheath.
- patients may present with an obvious swelling or may have joint pain as the primary complaint without an obvious etiology.
- Initial treatment is with nonsurgical measures including observation or needle aspiration. Many patients experience spontaneous resolution of the ganglion cyst without intervention and recurrence is common regardless of intervention used.

Gout
- Altered purine metabolism and sodium urate crystal precipitation into synovial fluid, M>W (9:1) until menopause (1:1)
- Usually young, >30 yo, asymmetric; great toe; tophi
o MC = podagra (attack of MTP of great toe) (70% of cases); pain, swelling, redness, exquisite tenderness. In chronic gout = tophi
  o dx: joint fluid – rod shaped negatively birefringent; serum uric acid level >8 (not diagnostic)
  o imaging: small, punched out lesions on XR = high likelihood diagnosis
  o tx:
    o lifestyle: elevation, rest, decrease purines (meats, bear, seafood, alcohol), weight loss, increase protein, limit alcohol
    o pharm: NSAIDs = drug of choice (indomethacin tid); colchicine = effective but bad GI s/e; steroid injections for those who can’t take NSAIDs, oral pred if other meds not tolerated
      ▪ thiazide diuretics and aspirin should be avoided
      ▪ management between acute attacks: colchicine, allopurinol
        • don’t start someone on allopurinol in acute attack
  o CPPD: pseudogout; usually >60yo; large joints, lower extremity; no tophi
    o Similar gout symptoms
    o Dx: rhomboid shaped calcium pyrophosphad crystals – positively birefringent
      ▪ XR shows fine, linear calcifications in cartilage
    o Tx: NSAIDs, colchicines, intra-articular steroid injections
      ▪ Colchicine = prophylaxis, NSAIDs = acute attacks
  
  **Osteoarthritis**
  o MC arthropathy in adults; progressive loss of articular cartilage with reactive changes in bone → pain / joint destruction
  o 90% have XR evidence of disease
  o s/s: decreased ROM, joint crepitus, morning stiffness, pain worsens throughout the day
  o dx: labs = nonspecific; XR = asymmetric narrowing, subchondral sclerosis, bony cysts, marginal osteophytes
  o tx: weight reduction, moderate exercise, Tylenol, NSAIDs, intra-articular steroids, bracing, canes, muscle strengthening
    o Tylenol = 1st line tx
  o Total joint replacement for advanced cases
  
  **Osteoporosis**
  o Disease of abnormal bone remodeling; decrease in total bone volume (less dense) → increased risk of fracture
  o Primary:
    o Type 1 – postmenopausal – most prevalent form → mostly in women
      ▪ Loss of estrogen in women / testosterone in men → trabecular bone affected
      ▪ Vertebrae, hip, distal radius = MC fracture site
    o Type 2 – senile → >75 = high risk type 2; trabecular and cortical bone affected; hip and pelvis = MC
  o Secondary: conditions in which bone is lost due to presence of other disease (malignancy, steroid, GI / hormone issue)
  o Dx: calcium phosphate, alk phos, serum protein electrophoresis should be measured and serum markers
    o DEXA = most helpful way to measure
      ▪ Postmenopausal women < 65 yo with 1+ risks; all postmenopausal women >64; women on HRT; fractures with minimal trauma, osteopenia on XR, patients with RA
  o Tx:
    o Prevention: weight bearing exercises, adequate calcium vitamin D, phosphorus intake, smoking cessation, limit alcohol
    o Bisphosphonate = 1st line – take on empty stomach, sit upright for 30-60 min after; long term use = jaw necrosis
    o HRT may be used (risk for MI, breast cancer, thromboembolic events)
  
  **Overuse Syndrome (Repetitive Strain Injury)**
  o Repetitive strain injury impacts muscles, nerves, ligaments, and tendons. These types of injuries can be caused by improper technique or overuse. The elderly are most commonly affected.
  o Symptoms include tenderness, stiffness, or tingling in the affected area.
  o Treatment may include anti-inflammatory drugs, physical therapy, ergonomic evaluation, and rarely surgery

  **Plantar Fasciitis**
  o Common in runners / overweight pt., caused by microscopic tears in plantar fascia at calcaneal origin
  o s/s: pain with first few steps in the morning + heel pain at night
  o exam: pain at calcaneal origin and inflexible achilles
  o imaging: XR normal +/- calcaneal fracture or bone spur; MRI = calcifications of plantar fascia
Reactive Arthritis (Reiter Syndrome)
- Seronegative – tetrad of urethritis, conjunctivitis, oligoarthritis, muosal ulcers; usually sequela of STD
- Features: asymmetric arthritis that involves large joints usually below the waist, mucocutaneous lesions, urethritis, conjunctivitis; leading cause of nontraumatic monoarthritis
- Dx: 50-80% = HLA-B27 positive; synovial fluid culture negative; evidence of permanent and progressive joint disease present on XR
- Tx: physical therapy and NSAIDs = mainstay; abx given at time of infection reduce chance of developing disorder but don’t alleviate sx

Rheumatoid Arthritis
- Chronic inflammatory disease with synovitis affecting multiple joints + extra-articular manifestations; 3:1 W>M
- 6/10 obtained for diagnosis
- S/s: joint pain and deformity, muscle weakness, myositis, myopathy, osteopenia, osteoporosis
  - Extra-articular: changes in skin, lungs, kidneys, eyes, liver, blood system, heart
- Dx: Aspiration and joint fluid analysis – quantify inflammation and exclude presence of gout / septic arthritis
  - EST and CRP elevated; RF positive in 80% of patients
  - ACPA / anti-CCP = more specific and positive in 95% of pt. with RA – soft tissue swelling / juxta articular demineralization seen on radiograph
- Tx: Consult rheum
  - Physical and occupational therapy
  - Pharm: do it early and aggressively to reduce pain, preserve function, prevent deformity
    - NSAIDs may be used in conjunction with DMARDs
      - Methotrexate = first line; combination usually required (steroid / -biologics)
  - Reconstructive surgery for severe cases

Strains / Sprains
- Strain = injury to bone-tendon unit at myotendinous junction or muscle itself
- Sprain = involves collagenous tissue, such as ligaments or tendons
- Both often follow sudden stretch; can lead to avulsion of tendon (e.g. mallet finger → splint 6 weeks)
- Can lead to ligamentous sprain (anterior talofibular ligament stretch → common ankle sprain)
- Tx: supportive therapy: rest, ice, compression, elevation, support/bracing (RICES)

Systemic Lupus Erythematosus
- Autoimmune disorder – inflammation, positive ANA, involvement of multiple organs; commonly affects women of childbearing age (MC in AA women)
- S/s: at least 4
  - Malar rash, discoid rash, photosensitivity, oral ulcers, arthritis, serositis, renal disease, ANA, hematologic disorders, immunologic disorders (LE cell, anti-DNA, anti-SM, false positive serologic test for syphilis), neuro disorders (seizures / psychosis with no other cause)
  - Significantly high titer ANA
  - R/o drug induced lupus – procainamide, hydralazine, isoniazid, methyldopa, quinidine, chlorpromazine
  - Relapsing remitting pattern = characteristic
- Dx: CBC, BUN, creatinine, urinalysis, ESR, serum complement (C3/C4);
  - Antibodies to anti-smith antigen, double stranded DNA or depressed levels serum complement may be used for markers of disease progression; ANA present 99% of the time
- Tx:
  - Regular exercise, smoking cessation, sun protection
  - NSAIDs for MSK issues
  - Antimalarials for MSK complains / cutaneous manifestations (hydroxychloroquine / quinacrine)
  - Steroids – topical for cutaneous stuff; oral for disease flares – taper as sx resolve
  - Methotrexate at low doses for arthritis, rashes, serositis, constitutional sx

NEUROLOGY
Alzheimer’s Disease

- Dementia, an age-related progressive cognitive decline, affects 5% of those aged 71-80 years, and near 40% of those aged over 90 years. Many patients are unaware or ignore these cognitive changes, chalking them up to “getting old.” Dementia presents with gradual, progressive memory loss. It is associated with word-finding and concentration problems, emotional lability, personality changes, social withdrawal, and difficulties with dressing, cooking, balancing the checkbook, and maintaining hygiene. There are multiple types of dementia: Alzheimer disease, vascular dementia, and other less common dementias. Alzheimer dementia is most common. Dementia can be evaluated with brief quantitative screening tests of cognitive function, such as the Folstein Mini-Mental State Examination (MMSE) or the Memory Impairment Screen. The MMSE may be useful to provide a baseline for future comparison. Controversy exists over the use of memantine and anticholinergic medications in the treatment of dementia.

- Alzheimer disease: 2/3 dementai cases; irreversible; early language / visuospatial defects
  - Severe memory deficits; clues don’t help memory retrieval
  - r/f: advanced age, family hx
- vascular: ¼ cases; r/f: HTN, dyslipidemia, DM, smoking, adv age
- lewy body: cognitive flucutaitons, visual hallucinatins, Parkinsonism
- frontotemporal dementia: personality, and social behavior changes, nonfluent speech
- neurodegenerative conditions: Huntington disease, metabolic abnormalities

Delirium

- rapid onset, fluctuating course
- acute changes in mental status – inattention, poor concentration, fluctuating levels of consciousness
- commonly occurs in hospitalized elderly pt.
- tx: identify and treat underlying cause; minimize use of physical restraints
  - may take weeks/months to resolve

Ischemic Stroke

- Anterior cerebral artery: frontal lobe dysfct, apraxia, contratlat paralysis (lower > upper)
- Middle cerebral artery: contratlat paralysis (upper > lower), hemianopsia, aphasia
- Posterior cerebral artery and VBI: LOC, nausea/vomiting, CN dysfct, ataxia, visual agnosia
- Rule out hypoglycemia
- CT reveals loss of grey-white interface, acute hypodensity
- Tx: Thrombolytics

Transient Ischemic Attack

- Transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia without acute infarction
- Atherosclerotic plaque reduces blood flow in the internal carotid artery Transient episode of neurological dysfunction without acute infarction
- 10% of TIA patients will have a stroke within 90 days
- Aspirin + dipyridamoloe or clopidogrel monotherapy (antiplatelet therapy!!!
- ABCD² score: predicts likelihood of subsequent stroke within 2 days
- 30% of those with CVA had TIA; risk is highest 24 hours after initial event

Bell Palsy

- s/s: sudden onset unilateral facial nerve parlysis with no other focal neurologic or systemic findings.
Sx peak in 48 hours
60% have viral prodrome
Incomplete closure of eyelids → corneal exposure keratitis (lubricating eye drops needed / patch at bed time)

**Differential diagnosis:** infectious, traumatic, and neoplastic etiologies, the most common diagnosis is idiopathic Bell's palsy.

**Tx:**
- PE will show CN VII nerve palsy that does not spare the forehead
- Most commonly caused by HSV
- Treatment is prednisone, artificial tears, tape eyelid shut
- Comments: Bilateral: Lyme disease, infectious mononucleosis

**Idiopathic Intracranial Hypertension (Pseudotumor Cerebri)**

- Patient will be a young obese female
- With a history of vitamin A toxicity, use of steroids or tetracyclines
- Complaining of HA and visual sx
- PE will show papilledema, CN VI palsy
- Labs will show opening pressure on LP
- **Treatment is acetazolamide, serial LPs, weight loss**

**Essential Tremor**

- Characteristics: usually presents with a postural tremor of the hands or head that is often worsened by psychic stress; can occur at any age
  - When laryngeal muscles are involved, the patient’s voice may shake as well
  - Legs are usually spared in this condition.
  - Essential tremor usually becomes more prominent with age, though significant disability is rare.
  - Improved after drinking alcohol; exacerbated by action
  - MC caused by autosomal dominant – usually have family member with similar symptoms
- **Tx:** beta-blocker (propranolol); Symptomatic treatment is limited to patients with lifestyle-limiting tremors. In addition to propranolol, other therapies include primidone, alprazolam, topiramate, or gabapentin.

**Headache**

**Cluster** – always unilateral, but can change sides with new attack; pt. is usually a man

- **s/s:** pain (excruciating unilateral pain, periorbital and temporal)
  - Autonomic sx: ptosis, miosis, lacrimation, conjunctival injection, rhinorrhea, nasal congestion
  - Circadian periodicity: short-lived (15-180 min) cluster attacks; attacks occur daily in clusters followed by remission
  - **tx:** 100% oxygen, sumatriptan (prophylaxis: CCBs)

**Tension:** MC type of headache

- Bilateral, non-pulsating, bandlike pain occurring in frontal and occipital regions; with neck muscle tenderness
- MC caused by stress, or fatigue, glare, noise
- **Tx:** NSAIDs, smoking cessation

**Migraine:** F>M

- Triggers: menstruation, pregnancy, contraceptives, food (chocolate, cheese, MSG, nitrites), alcohol
- Gradual onset unilateral > bilateral, throbbing, pulsating headache,
  - Without aura = most common, N/V, photophobia, phonophobia
- Aura: scotoma, flashing lights, sound
  - HA follows aura w/in 30 min; visual = MC

- Dx: clinical
- Tx:
  - Abortive – triptans, DHE, antiemetics, NSAIDs
  - Prophylaxis – beta blockers, CCBs, TCAs
  - Triptans / DHE CI in HTN or CV disease

**Status Epilepticus**

- **s/s**: > or equal to 5 min continuous seizure activity or more than oe seizure without recovery from postictal state in between episodes
- **always check finger stick blood glucose!!!** Consider pyridoxine (B6) for INH toxicity!!!
- **MC** caused by change in med regiemtn of someone with seizure disorder
- **Tx:**
  - Place in left lateral decubitus position (suppressed gag reflex → prone to aspiration of gastric conents)
    - IV route is preferred

- **Absence Seizure (petit mal)**
  - Brief mental status change; without motor activity – blank stare
  - No aura, no post-ictal state, no loss of postural tone
  - MC in 5-10 yo
  - EEG → brief 3-Hz, spike and wave discharged
  - Tx: ethosuximide

**Parkinson Disease**

- Patient will be complaining of rigidity, bradykinesia, postural instability, micrographia
- PE will show a resting pill-rolling tremor, mask-like facies, cog wheeling of extremities, shuffling gait
- Most commonly caused by dopamine depletion in basal ganglia
- Treatment is levodopa-carbidopa

**Vertigo**

- **Definition**: perception or sensation of movement; results from dysfunction in the vestibular system from either its peripheral or central components.
- **s/s**: feeling of swaying, spinning, whirling, leaning, or tilting, feeling intoxicated, nausea and vomiting may also be seen.
- **Physical examination**: reveal nystagmus in nearly all patients, often lateralizing to the affected side
  - Although less common than peripheral etiologies, central causes of vertigo are more concerning and include conditions such as vertebrobasilar insufficiency, brainstem and cerebellar infarct or hemorrhage, basilar artery migraine, and degenerative diseases such as multiple sclerosis.
  - In general, when compared to peripheral vertigo, the symptoms of central vertigo are less acute and more persistent and may be associated with neurologic deficits. However, exceptions exist, particularly in those patients with cardiovascular risk factors. Limb ataxia is not seen with peripheral causes and is usually attributable to cerebellar lesions.
- **dx**: A noncontrast head CT is a reasonable screening test with suspected central vertigo (and is most appropriate to evaluate for potential intracranial hemorrhage). Secondary to the fact that it does not permit adequate visualization of the cerebellum, it is not considered the definitive test. Magnetic resonance imaging with angiography is the study of choice in most cases of suspected central causes of vertigo.
- **Peripheral causes**: BPPV, acute otitis media, labyrinthitis, meniere’s, vetibular neuroma trauma (horizontal nystagmus); sudden onset
  - **BPPV →** rapid onset with nausea nad vomiting elicited with moving head to certain position; relieved by moving head away from that position
  - **Test: dix-hallpike; positive test = nystagmus**
  - Tx: Epley maneuvers – dislodge otolith from semicircular canal
- **Central causes**: meningitis, encephalitis, vertebral basilar insufficiency, cerebellar hemorrhage,tumor, temporal lobe epilepsy (vertical nystagmus); gradual / sudden onset
**DERMATOLOGY**

### Acanthosis Nigricans

- Patient will be obese or diabetic
- PE will show **thickened, velvety, darkly pigmented plaques on the neck or axillae**
- Comments: Screen for diabetes in those not yet diagnosed
- Benign: obesity related, drug-induced, diabetes, endocrine disorders
- Paraneoplastic: underlying GI / GU malignancy, lymphoma

### Acne Vulgaris

- Patient will be an adolescent
- Complaining of rash on the face, neck, upper chest, and back
- PE will show **open comedones (blackheads), closed comedones (whiteheads)**, papules and pustules
- Most commonly caused by *Propionibacterium acnes*
- Treatment is:
  - Mild to moderate: topical retinoids, topical antibiotics, or benzoyl peroxide
  - Moderate to severe: add oral antibiotics
  - Severe: oral isotretinoin *(Pregnancy class X, must have two forms of birth control)*

### Actinic Keratosis

- Patient will be a man
- With a history of an **outdoor occupation and sun exposure**
- Complaining of rough bumps on his head
- PE will show **rough, scaly, erythematous papules on sun-exposed areas**
- Comments: **Potential to progress to squamous cell carcinoma**
- **r/f**: UV radiation, fair skinned, sun exposed areas

### Alopecia Areata (!)

- Patient with a history of an autoimmune disorder
- Complaining of hair loss
- PE will show **patches of smooth, non-scarring hair loss with patches of smaller hairs termed exclamation hairs**
- Most common cause is autoimmune
- Treatment is intralesional corticosteroids

### Basal Cell Carcinoma

- Patient will be complaining of a painless slow growing **lesion on the face, ears, or neck**
- PE will show **pearly papule with rolled borders and telangiectasia**
- Diagnosis is made by **shave biopsy**
- Most common type of skin cancer; rarely metastasizes
- Treatment is surgical excision

### Bullous Pemphigoid

- Autoimmune disease of the elderly; subepidermal; begins as pruritic papules
• Patient will be older than 60-years-old
• Complaining of intensely pruritic papules that became large, tense blisters/bullae
• PE will show tense and firm blisters that do not extend with lateral pressure (Nikolsky sign negative)
• Most commonly caused by chronic autoimmune blistering disease
• Treatment is corticosteroids and immunosuppressants

Cellulitis

• Patient will be complaining of pain, redness, swelling
• PE will show tenderness, erythema with poorly demarcated borders, lymphedema
• Most commonly caused by Staph aureus and Streptococci
• Admit if don’t improve after 48-72 hours of abx
• Oral abx: Bactrim, doxy, linezolid (highly active)

Condyloma Acuminata

• Patient will be complaining of genital lesions
• PE will show cauliflower-like lesion
• Most commonly caused by HPV 6 & 11 (strong association → genitourinary and rectal cancer)
• Comments: most common STD

Dermatitis

• Atopic (eczema)
  • Patient with a history of asthma or hay fever
  • commonly causes diffuse xerosis, as well as eczematous plaques, papules, and crusting. In infants, plaques are most commonly distributed over the cheeks, as well as the extensor surfaces of the extremities. In older children, the face is less often involved, and the flexor surfaces of the extremities, such as the antecubital fossa, are frequently affected. In addition to causing pruritus and irritation, the compromised skin integrity in atopic dermatitis may result in bacterial and viral superinfections. Children with eczema are also predisposed to other atopic diseases such as asthma and allergic rhinitis, as well as to food allergies.
  • Complaining of itchy, scaly rash on arms, often worse in the winter; itch, scratch, rash, itch (“itch that rashes”)
  • PE will show thick, leathery, hyperpigmented areas on flexor surface
  • Treatment is topical corticosteroids, lubricating ointments
• Seborrhea:
  • Seborrheic dermatitis, also known as Cradle Cap, is a common pediatric skin condition that results in superficial scaling. The scales are classically greasy and yellow. Seborrheic dermatitis is distributed in areas where seborrheic glands are most concentrated such as the scalp, central face (“T zone”), preauricular skin, and intertrigenous area.
• Perioral:
  • Patient will be a woman age 16 - 45-years-old
  • Complaining of eczema or acne-like perioral rash
  • PE will show irregularly grouped, discrete red papulopustules on a red base on the face, but spare the vermilion border
  • Treatment is metronidazole and erythromycin
• Herpetiformis:
  • Associated with celiac disease most commonly (also associated with thyroid disease)
  • Multiple intensely pruritic papules and vesicles that occur in grouped arrangements
  • Chronic, very itchy skin made up of bumps and blisters
  • Tx: dapsone, gluten free diet
• Allergic:
  • Patient will complain of a rash
PE will show erythematous, scaly plaques, vesicles, and bullae
Diagnosis is made by patch test
Most commonly caused by nickel, poison ivy, soaps, and clothing
Comments: Cell-mediated reaction type IV
Tx: resolves spontaneously 1-3 weeks; symptomatic therapy, topical corticosteroids, systemic steroids (severe cases)

**Drug Eruptions**

Patient with a history of recent medication use
Complaining of a rash which appears in nearly the same location each time the medication is used
Systemic symptoms absent, itching / burning / stinging, may occur anywhere on body
PE will show erythematous and edematous plaque
Eliciting drugs: Bactrim, tetracyclines, penicillins, quinolones, dapsone, NSAIDs, Tylenol, bartitutures, antimalarials
Tx: drug withdrawal, avoidance, supportive care (benedryl)

**Dyshidrosis / Dyshidrotic Eczema**

Patient will be complaining of intense pruritus on their palms and sides of the fingers
PE will show vesicles that appear to contain grains of tapioca; vesicles last 2-3 weeks
Treatment is avoidance of long exposure of the hands to water, topical corticosteroids for acute flares

**Eryspilas**

Patient will be complaining of malaise, fever, chills, or nausea; acute onset with systemic manifestations
PE will show intense and deeply erythematous, sharply demarcated elevated shiny patch
Most commonly caused by Streptococcus pyogenes infection (group A beta strep)
Treatment is penicillin V, amoxicillin, azithromycin, or clarithromycin

**Erythema Multiforme**

Patient will be complaining of acute onset of symmetric target lesions on palms and soles, face and trunk may also be involved
PE will show target-like with a central dark papule surrounded by a pale area and a halo of erythema
Most commonly caused by herpes simplex virus (HSV)
Treatment is usually self-limiting, supportive
Comments:
- Common drugs that cause EM: Sulfa, Oral hypoglycemics, Anticonvulsants, Penicillin, NSAIDs (SOAPS)
- Erythema multiforme minor: localized eruption of the skin with minimal or no mucosal involvement
- Erythema multiforme major: one or more mucous membranes are involved

**Exanthems**

Common viral exanthems:
- Varicella: fever, respiratory sx 1-3 days, vesicular erythematous, torso and face. To extremities (dew drop on rose petal); pruritic
- Erythema Infectiosum (fifth disease, slapped cheek): human parvovirus B19; 4-14 days; no prodrome; red face flapped cheek, lacy pink macular rash on torso
- Roseola: HHV 6 or 7; fever (4 days), pink macular rash; fever resolves before rash
- Measles (rubeola): 8-14 days incubation; fever, cough, anorexia, coryza; maculopapular, koplak spots in mouth
- Rubella: maculopapular rash head to toe; teratogenic

**Folliculitis**
- Inflammation of hair follicles; MC caused by s. aureus
- Noninfectious: common in hot, oily environments
- Erythematous papules / pustules usually not painful; abscesses may form at site of more severe folliculitis
- Tx: gentle cleansing with antibacterial soap and mild compresses; protect from offending substances and use drying agents
- Topical clinda / erythromycin for infectious
- Oral abx sometimes necessary
- “hot tub” folliculitis usually resolves without treatment (oral FQ with severe)

**Herpes Simplex**

- Patient will be complaining of painful oral lesions – vesicle on erythematous base
- PE will show painful vesicles and erosions on the tongue, buccal mucosa, and lips
- Labs will show multinucleated giant cells on Tzanck smear
- Diagnosis is made clinically. Gold standard is tissue culture with polymerase chain reaction (PCR)
- Most commonly caused by herpes simplex virus (HSV) type 1
- Treatment is topical antiviral therapy or oral acyclovir

**Hidradenitis Suppurativa**

- Patient will be a woman
- With a history of lesions that have waxed and waned over the past few years
- Complaining of tender nodules in her axillae and anogenital area; affects apocrine sweat glands
- PE will show lesions that are tender, malodorous, often with exudative drainage, sinus tracts
- Triggers: sweating, hormonal changes related to menstrual cycle, friction
- Persistent lesions → sinus tracts
- Treatment is intralesional triamcinolone, topical clindamycin
- Comments: Hurley staging system describes the severity of disease

**Histamine Skin Poisoning**

- Dark fleshed, peppery tasting fish → Tuna, mahi-mahi, mackerel
- Histamine ingestion from decarboxylate histidine
- Sx: Anxiety, flushing, headache, palpitations, vomiting
- Tx: Antihistamines

<table>
<thead>
<tr>
<th>Hypersensitivity Reactions Table</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type</td>
</tr>
<tr>
<td>------</td>
</tr>
<tr>
<td>I: Anaphylactic (immediate)</td>
</tr>
<tr>
<td>II: Cytotoxic</td>
</tr>
<tr>
<td>III: Immune complex</td>
</tr>
<tr>
<td>IV: Cell-mediated (delayed)</td>
</tr>
</tbody>
</table>
Impetigo

- common pediatric bacterial skin infection that is highly contagious and auto-inoculable usually caused by *Staphylococcus aureus* and *Group A beta-hemolytic Streptococci*.
- MC: seen in children ages 2-5 years.
- Risk factors for impetigo include warm, humid conditions, poverty, crowding, and poor hygiene.
  - Secondary impetigo can occur at sites of minor abrasion or scratches.
- s/s: Impetigo typically begins as *papules that progress to vesicles and surrounding erythema*. Over about one week, the *vesicles eventually rupture and form a thick, adherent, golden crust*. Regional *lymphadenopathy* is a common finding.
- dx: Gram stain and culture is recommended to determine bacterial etiology.
- Tx:
  - Topical mupirocin
  - Widespread / outbreaks = treated with kephlex
  - Beta-lactamase resistant antibiotics with *staphylococcal and streptococcal coverage* are typically used.
  - Patients with suspected or confirmed methicillin-resistant *S. aureus* should be treated with doxycycline, clindamycin, or trimethoprim-sulfamethoxazole. Antibiotic treatment is usually for seven days. Children may return to school 24 hours after starting antibiotics.

Kaposi Sarcoma

- Patient with a history of HIV infection
- PE will show red, purple, brown, or black skin lesions that are nodules/papules
- Labs will show CD4 counts < 200 cells/mm3
- Diagnosis is made by biopsy (presence of spindle cells)
- Most commonly caused by *Human herpesvirus 8 (HHV-8)*
- Comments: Is an AIDS-defining illness
- Tx: ART
  - Local topical therapy may be considered in patients with cosmetically unacceptable lesions or those with advanced disease causing significant patient discomfort. Cytotoxic agents like liposomal doxorubicin have been approved for the treatment of AIDS-related Kaposi sarcoma.

Lichen Planus

- Patient will be complaining of pruritic skin discolorations on ankles or wrists
- PE will show pruritic, purple, polygonal, and papules *(four P’s)* and fine, white lines *(Wickham’s striae)*
- Koebner’s phenomenon applies here
- Treatment is corticosteroids
- Autoimmune; may affect skin, mucous membranes (oral mucosa), scalp, nails, genitalia

Lichen Simplex Chronicus

- Patient will be complaining of generalized pruritus and frequent scratching
- PE will show *multiple linear excoriations and thickened skin*

Lipoma

- Benign fatty tumor
  - Type depends on histology
  - Mostly in adults, M>F
  - Associated with syndromes
  - Treatment: Excision
  - Treatment is reducing pruritus and *minimizing rubbing and scratching*

Lyme Disease

- Patient will be complaining of generalized pruritus and frequent scratching
- PE will show multiple linear excoriations and thickened skin


• Patient with a history of being in the woods hiking or camping
• Complaining of:
  • Stage I: erythema migrans (pathognomonic), viral-like syndrome (fever, fatigue, malaise, myalgia, headache)
  • Stage II: arthritis, myocarditis, bilateral Bell’s palsy
  • Stage III: chronic arthritis, chronic encephalopathy
• PE will show slightly raised red lesion with central clearing, erythema migrans (bull's-eye) rash
• Most commonly caused by Borrelia burgdorferi carried by Ixodes tick
• Treatment is doxycycline, children - amoxicillin or doxycycline (if used for < 21 days), pregnant - amoxicillin
• Comments: Bilateral facial nerve palsy is virtually pathognomonic for Lyme disease

Melanoma

• Patient will have fair skin
• With a history of severe blistering sunburns, a family history of melanoma, dysplastic nevus syndrome
• Complaining of an itching, tender lesion that won't heal
• PE will show ulceration and ABCDE
  • A - Asymmetry
  • B - Border irregularity
  • C - Color variation
  • D - Diameter
  • E - Evolution
• Diagnosis is made by biopsy - excisional or punch
• Treatment is excision with adequate margins, interferon reduces recurrence
• Comments: Most important factor is depth

Melasma

• Patient will be a woman who is pregnant or using oral contraceptives; “mask of pregnancy”
• Complaining of discoloration on parts of the face
• PE will show dark, irregular, well demarcated hyperpigmented macules/patches
• Most commonly caused by hormonal changes
• Treatment is sunscreen and sun avoidance; tretinoin can be used in combo with meds that use hydroquinone and or fluocinolone – combos = more promise than monotherapy
• Comments: During pregnancy, it is called chloasma

Moluscum Contagiosum

• Patient will be a school-age child; spread by direct contact
• Complaining of "warts"; painless w no systemic symptoms; spares palms and soles
• PE will show multiple waxy, dome-shaped papules with umbilicated appearance
  • Umbilicated, central keratin plug
• Most commonly caused by Poxvirus
• Treatment is self-limiting – resolves spontaneously, cryotherapy, curettage, cantharidin, podophyllotoxin

MRSA

Methicillin-resistant Staphylococcus aureus (MRSA) is a strain of staphylococcus that has developed resistance to beta-lactam antibiotics. This resistant strain is often healthcare-associated, but is also found in the community. For limited community-acquired infections not requiring hospitalization, patients may be treated with oral antibiotics. A tetracycline (such as doxycycline) is an acceptable choice to treat community-acquired MRSA. Clindamycin or a sulfa drug (such as TMP-SMX) is another acceptable
alternative. Healthcare-associated strains of MRSA are typically not susceptible to these oral antibiotics and will require IV vancomycin. Vancomycin is a bactericidal drug that inhibits cell-wall biosynthesis.

- Doxy, clindamycin, Bactrim

**Nummular Eczema**

- Pruritic, inflammatory disorder that affects young adults and elderly; M>F; occurs more in winter
- Small, grouped vesicles coalesce to form coin-shaped plaques with erythematous base and well-demarcated borders, MCon extremities; crusting/ excoriations common
- Tx: chronic disorder that responds to moisturizers / topical steroids
  - Tar baths / UVB phototherapy for refractory cases

**Onychomycosis**

- Patient will be complaining of thickened and discolored toenails
- Diagnosis is made by KOH preparation of nail scraping
- Treatment is oral terbinafine x12 weeks
  - Check liver functions first
- Comments: Serum aminotransferases should be monitored before starting treatment with terbinafine and during the treatment due to hepatotoxicity

**Paronychia**

- PE will show an infection of lateral nail fold
- Most commonly caused by *S. aureus*
- Treatment is ABX, warm soaks, I&D
  - incision and drainage. The finger should be soaked in warm water and the edge of the skin at the nail should be gently lifted away from the nail using a scalpel blade. Subsequently, the cavity should be irrigated and packing gauze should be placed under the eponychium for 24 hours

**Pediculosis Capitis (Head Lice)**

- Patient will be a child
- Complaining of itching on their head; direct contact with head of infested person (lice don’t jump, fly,m or use pets as vectors)
- PE will show nits (eggs) or actual lice
- Most commonly caused by *Pediculus humanus capitis*
- Treatment is topical pediculicides, such as permethrin
- Comments: A second treatment should be applied on day nine after the first treatment to ensure eradication

**Pilonidal Disease**

- Abscess in sacrococcygeal cleft associated with subsequent sinus tract development
- M>F; more common in hirsute and obese individuals
- Clinical presentation = painful, fluctuant area at sacrococcygela cleft
- Tx: surgical draining may be supplemented with abx; follicle removal may be required with unroofing of sinus tracts

**Pityriasis Rosea**

- Patient with a history of a larger lesion one week prior, herald patch
- Complaining of rash on the back
- PE will show diffuse papulosquamous rash on the trunk, Christmas tree-like distribution
- Treatment is self-limiting disease, topical corticosteroids or oral antihistamines for itching
Psoriasis

- Patient will be complaining of a rash on extensor surfaces of arms & legs
- PE will show bilateral sharply marginated papules/plaques with silvery scales, Auspitz sign (scale removal produces blood droplets), nail pitting, koebner phenomenon (plaque formation on site of prior trauma 1-2 weeks after skin injury)
- First line tx: topical corticosteroids

Rocky Mt. Spotted Fever

- Patient with a history of recently being in the woods hiking or camping
- Complaining of abrupt onset of severe headache, photophobia, vomiting, diarrhea, and myalgia
- PE will show maculopapular eruption on the palms and soles
- Diagnosis is made by skin biopsy
- Most commonly caused by Rickettsia rickettsia
- Treatment is ALWAYS doxycycline, even in children

Rosacea

- Patient will be complaining of acne-like rash on the forehead, cheeks and nose that gets worse with ingestion of ETOH, hot drinks and spicy foods
- Flushing, erythema, papules, pustules; no comodones
- Distribution on cheeks, nose, forehead, chin
- PE will show facial flushing, telangiectasia, skin coarsening, rhinophyma (big nose) and absence of comedones
- Treatment is topical metronidazole

Scabies

- Scabies is an infestation with Sarcoptes scabiei. These mites burrow into the epidermis causing inflammatory papular lesions secondary to a host hypersensitivity reaction
- Spread by skin-to-skin contact or by fomites such as bed sheets or clothing.
- Sensitization to the mite may take several weeks before pruritus will occur.
- Symptoms are widespread itching that patients often describe as the “worst itching of my life”. Worse at night
- Common sites of infestation include the axillae, finger webs, wrists, elbows, girdle area, and feet. Men will often present with papular lesions of the penis.
- p/e: small papules vesicles, and burrows in webbed spaces of fingers and toes
- dx: made my microscopic eval
- The diagnostic finding is a thin, serpiginous burrow with a black speck at the end where the mite resides.
- First-line treatment is with Permethrin cream applied in a single application to the entire body and washed off after 8 hours.

Seborrheic Keratosis

- Patient will be older
- PE will show a lesion, flat or raised, smooth or velvety with “stuck-on” appearance noted on the face, shoulders, chest, and back
- Most commonly caused by a benign, epidermal neoplasm → does not lead to cancer
- Treatment is liquid nitrogen, curettage, shave removal
  - Biopsy needed to r/o cancer, though it does not lead to cancer

Spider Bites

- Brown recluse:
  - brown violin shape on cephalothorax; initial bite is painless
  - dark colored, depressed center 1-2 days after bite → dry eschar and ulceration → necrosis
• Southern midwestern United States
• Cytotoxin → local tissue destruction
• Antivenin unavailable
• Rx: wound care, pain control, administer tetanus vaccine, supportive care

Steven Johnson Syndrome

• PE will show vesicles and bullae involving < 10% of the body surface area including mucous membranes
• Most commonly caused by a reaction to medications
• Treatment is a referral to a burn center
  • Start immediate treatment: abx, steroids, pain meds, fluid hydration IV
  • May need debridement and skin grafting
• Comments: SCORTEN score is used to determine prognosis and clinical setting for treatment

SLE

• Patient will be an African-American or a woman
• Complaining of fever, lymphadenopathy, weight loss, general malaise, or arthritis
• PE will show malar rash ("butterfly rash")
• Labs will show anti-nuclear antibodies (ANA), anti-dsDNA antibodies, anti-smith antibodies, anti-histone antibodies
• Treatment is NSAIDs, steroids, immunosuppressants, hydroxychloroquine
• Comments: Drug-induced: Hydralazine, INH, Procainamide, Phenytoin, Sulfonamides (HIPPS). False-positive test for syphilis

Telogen Effluvium

• Transient, diffuse hair loss
• Common causes: child birth, major illness/surgery, stress, medications, malnutrition
• Treat reversible causes

Tinea

• Capitis: children, kerion
• Corporis: red, annular, raised, central clearing – ring worm
  • Patient will be complaining of arash
  • PE will show an erythematous, scaly plaque that is annular, has raised edges, sharply marginated, with a central clearing
  • Diagnosis is made by KOH preparation of skin scrapings
  • Most commonly caused by dermatophyte
  • Treatment is topical antifungal clotrimazole
• Cruris: obese males, sweat
• Pedis: sweaty feet
  • MC dermatophyte infection
  • Caused by direct contact – walking barefoot in locker rooms
  • Dx: segmented hyphae in skin scrapings with KOH prep
  • Tx: topical clotrimazole
• Unguium: raised, discolored nail; nail plate white, thick, crumbly
• Capitis/unguium rx: BLACK DOT PATTERN; PO antifungals
- **Tinea capitis** must be treated with systemic antifungal agents because topical agents do not penetrate the hair shaft. However, adjunct treatment with selenium sulfide shampoo or 2% ketoconazole shampoo should be used for the first two weeks because it may reduce transmission.
- **Tx**: oral antifungal, griseofulvin (first line), terbinafine (alternative first line)

**Versicolor**:
- Involves trunk and extremities; hypopigmented usually
- **Dx**: KOH prep – spaghetti and meatballs appearance
- **Tx**: topical antifungal / oral for severe
- Patient will be complaining of hypopigmented areas do not ta
- PE will show scaly patches on the chest and trunk
- Diagnosis is made by KOH preparation of skin scraping
- Most commonly caused by *Malassezia furfur*
- Treatment is topical selenium sulfide

**Toxic Epidermal Necrolysis**
- Mucocutaneous blistering disorder, most often caused by drug reaction
- Commonly associated: sulfonamides, aminopenicillins, quinolones, cephalosporins, tetracyclines, pohenobarb, carbamazepine, phenytoin, valproic acid, oxicam, allopurinol, steroids
- TEN >30% of body
- Fever, photophobia, sore throat, mucosal inflammation, sore mouth; TEN has higher fever and more severe epidermal separation and loss compared with SJS
- Regrowth of skin = 3 weeks
- 90% have lesions mouth to anus
- Biopsy is diagnostic
- Treatment is withdraw offending agent, transfer to burn unit if necessary; treat for fluid / electrolyte imbalance
  - Treat with steroids = debated / no conclusively researched
  - IVIG commonly used – data do not show improvement in mortality

**Urticaria**
- **urticarial rash (hives)**. Urticaria may occur from a variety of agents, including foods (lobster, strawberry); drugs (penicillin, aspirin); infections (hepatitis, mononucleosis, coxsackie); change of temperature; and connective tissue disorders, to name a few; MC caused by IgE mediated reaction to allergen
- It can occur in isolation or as part of a systemic anaphylactic reaction. The characteristic rash appears as edematous plaques with pale centers and red borders.
- The rash of urticaria is transient, lasting less than 24 hours, though new lesions may continuously develop.
- Dermatographism is the most common form of physical urticarial and results in development of an urticarial wheal within 30 minutes of firm skin stroking.
- **Tx**: supportive care, antihistamine, steroids (if associated with angioedema)

**Varicella Zoster**
- Patient will be older
- Complaining of a painful, papulovesicular rash preceded by tingling or hyperesthesia
- PE will show a rash with unilateral/dermatomal distribution that does not cross midline
- **Hutchinson’s sign** is blistering associated with herpes zoster infection at the tip of the nose. This is often associated with and precedes ophthalmic involvement of the zoster infection.
- Tzanck smear will reveal the presence of multinucleated giant cells
- Most commonly caused by reactivation of latent varicella-zoster virus
- Comments: Postherpetic neuralgia: persistent pain > 3 months

**Verrucae**
- Caused by HPV
• Tx: spontaneous regression is typical over time
• Type, location, age dictate treatment
  • Salicyclic acid plasters for common warts

Vitiligo

• patient will be complaining of patches of pale skin affecting the neck, upper back and the chest
• PE will show white, non-scaling, well-demarcated areas of hypopigmentation
• Most commonly caused by autoimmune destruction of melanocytes (alopecia, psoriasis, T1D, RA, IBD, pernicious anemia, MG, lupus, Sjogren)
• Comments: Woods lamp examination will accentuate the hypopigmentation
• Tx:
  • Psychosocial support
  • Stabilize: oral steroid, phototherapy, minocycline, methotrexate, vitamin D
  • Regimentation: topical steroid

ENDOCRINOLOGY

Adrenal Insufficiency (Addison)
• MC cause is autoimmune destruction of adrenal cortex (80%); secondary cause = pituitary based / TB in areas of TB prevalence
• Crises may be precipitated by infection, trauma, surgery, stress, lymphoma, metastatic cancer, amyloidosis, scleroderma, 
  hemochromatosis, stopping steroid meds
• Features: nonspecific – fatigue / weakness, anorexia, weight loss, irritability, anxiety, GI sx, amenorrhea, salt cravings, 
  orthostatic hypotension, delayed deep tendon reflexes, hyperpigmentation
• Dx:
  o hyperkalemia, hyponatremia, hypoglycemia, hypercalcaemia, low BUN
  o serum cortisol rise >20ug/dL after administrated of cosyntropin
  o Antiadrenal antibodies present in 50%
  o DHEA levels <1000 ng/mL
• Tx: primary disease: steroids and mineralocorticoids
  o DHEA may be given for increased muscle mass
  o Normal prognosis
  o Crisis: aggressive IV saline, glucose, gluocorticoids and treat underlying cause

Cushing Disease
• Cushing syndrome = cortisol excess from excessive autonomous adrenal cortical secretion
• Cushing disease = ACTH secreting pituitary microadenoma usually very small on anterior pituitary; F3x>M
• Features:
  o Hypercortisolism → obesity (centripidel, buffalo hump, moon facies, supraclavicular pads), HTN, thirst, polyuria
  o Proximal muscle weakness, pigmented striae; backache, headache, oligomenorrhea / amenorrhea / ED; emotional 
    lability / psychosis
• Dx: overnight dexamethasone suppression test
  o Suppression <5ug/dL excludes cushing with some certainty; plasma or serum ACTH <20pg/mL suggests adrenal tumor
  o mRI preferred for pituitary tumors, CT may show adrenocortical or other tumors
• tx: transphenoidal selective resection of pituitary tumor cures 75-90%; irradiation provides remission in 50-60%
  o 95% 5 year survival

Diabetes Mellitus
• Type 1: MC in young people
  o Little to no endogenous insulin secretion; elevated plasma glucagon pancreatic B cells don’t respond to stimuli
  o Most are autoimmune (90%) ith antibodies to insulin, islet cells, and glutamic acid decarboxulase
  o Sx: polyuria, polydipsia, polyphagia, nocturia, rapid weight loss despite normal / increased appetite, blurred vision, 
    glucosuria,
    ▪ Random plasma glucose >200mg/dL, blurred vision, pruritus, weakness, paresthesias, vulvovaginitis
**Type 2:** younger persons who are overweight / obese; central obesity

- Features: polydipsia, polyuria, fatigue, candida vaginitis, skin infections, blurred vision, poor wound healing
- **Dx:**
  - Random glucose >200 with symptoms, fasting glucose >126 on more than one occasion, or A1c >6.5%
  - OGTT for pt. with fasting glucose levels between 100-125
- **Tx:** weight loss!!, diet, exercise 150 min/week, monitor feet/eyes, control blood pressure, urine albumin / creatinine screenings
- **Oral agents**

- **Hyperthyroidism**
  - Serum TSH suppressed; Graves = autoimmune
  - Features: weight loss, anxiety, warm, moist skin, onycholysis, insomnia, fine tremor, fatigue, muscle cramps, weakness, amenorrhea, tachycardia, palpitations, systolic HTN, PVCs, afib, brittle hair, heat intolerance, hyperreflexia
  - **Dx:** Elevated T3 and free T4, low TSH
  - **Tx:** beta blocker (propranolol) to control sx; propylthiouracil and methimazole, PTU for breastfeeding, iodine ablation, thyroidectomy

- **Hypothyroid**
  - 95% are autoimmune; many associated with other autoimmune issues
  - Features: weakness, dry/ coarse hair, latheargy, slow speech, cold intolerance, eyelid edema, forgetfulness , facial edema, constipation, oarse hair, eight gain, facial dullness, depression, anemia, bradycardia, hyporeflexia, enlarged thyroid
  - **Dx:** best test is TSH; normal / low normal free T4 and TSH = euthyroid
  - **Tx:** levothyroxine; check levels of thyroid frequently
Anorexia Nervosa

- **general characteristics**
  - distorted body image, intense fear of becoming fat despite being underweight → self-imposed starvation
  - losing weight = desired achievement of self control, gaining weight = unacceptable lack of discipline; egosyntonic (denies seriousness of low body weight)
  - may exercise excessively; commonly have food related obsessions (hoarding food, collecting recipes)
  - ~90% = female ages 15-30ki8, MC in developed countries and in professions that require thinness
  - Two types
    - Restricting: eats very little and does not binge or purge
    - Binge and purge

- **Symptoms:** emaciation, orthostatic hypotension, **bradycardia**, hypothermia, dry skin, lanugo, peripheral edema, amenorrhea/delayed menarche, salivary gland hypertrophy, dental erosion/loss of tooth enamel, calluses or abrasions on back of hand from induced vomiting, constipation, leukopenia, electrolyte abnormalities (hypochloremia, hypokalemia, elevated BUN, metabolic alkalosis, arrhythmias. Osteoporosis and increased likelihood of fx are of concern d/t decreased estrogen, increased cortisol, inadequate calcium and vitamin D intake, increased amylase
  - Total cholesterol may be increased due to increased production of cardioprotective high-density lipoprotein

- **Treatment:** rarely seek tx; family members usually bring it up
  - **Multidisciplinary approach**
    - First goal: restore nutritional state; hospitalization often indicated if pt. more than 20% below expected body weight; 10% mortality rate; correct fluid and electrolyte abnormalities and gradual weight restoration crucial
    - Outpatient management: behavioral therapy, family therapy, supervised weight gain programs – add 500 calories more than amount needed to maintain present weight and increase gradually by spreading feedings to 6/day
    - Antidepressents can be used esp when depression present – amitryptaline, paroxetine, mirtazapine – NOT BUPROPION (LOWERS SEIZURE THRESHOLD) in pt. with electrolyte disturbances
    - Meds don’t play majory role
    - Can give olanzapine to assist with weight gain
      - Elderly patients with dementia related psychosis treated with antipsychtics are at increased risk of death compared with placebo

- **Anorexia nervosa** is characterized by restriction of food intake resulting in low body weight, intense fear of gaining weight or becoming fat, and disturbance of body image. It occurs most often in adolescent females and is often accompanied by depression and other comorbid psychiatric disorders. For low-weight patients with anorexia nervosa, virtually all physiologic systems are affected, ranging from hypotension and osteopenia to life-threatening dysrhythmias, often requiring emergent assessment and hospitalization for metabolic stabilization. **Sinus bradycardia is almost universally present in patients with anorexia nervosa.** It is hypothesized that this is due to vagal hyperactivity resulting from an attempt to decrease the amount of cardiac work by reducing cardiac output. As cachexia progresses, patients with anorexia nervosa lose strength and endurance, move more slowly, and demonstrate decreased performance in sports. Overuse injuries and stress fractures can occur. Bradycardia, orthostatic hypotension, and palpitations may progress to potentially fatal dysrhythmias. The focus of initial treatment for patients who have anorexia nervosa with cachexia is restoring nutritional health, with weight gain as a surrogate marker. Feeding tubes may be needed in severe cases when the patient has a high resistance to eating. Refeeding syndrome can occur in a malnourished individual when a rapid increase in food intake results in dramatic fluid and electrolyte shifts, and is potentially fatal.

- **Hypophosphatemia** is the hallmark and predominant cause of refeeding syndrome. Refeeding syndrome can be fatal and patients are most at risk during the first two weeks of nutritional supplementation. The syndrome is caused by fluid and electrolyte shifts that occur secondary to nutritional rehabilitation. During episodes of starvation, phosphate stores are depleted. When carbohydrates are reintroduced during aggressive nutritional supplementation, insulin triggers cellular uptake of phosphate which results in low serum phosphate levels. **Extreme hypophosphatemia can lead to myocardial dysfunction and respiratory failure.**

- **Avoid bupropion:** Bupropion is an atypical antidepressant that enhances central nervous system noradrenergic and dopaminergic release. Bupropion also has the potential side effect of neuropsychiatric symptoms and patients should be monitored closely when treatment is initiated. **Bupropion is contraindicated in patients with a seizure disorder or a history of anorexia nervosa.** Bupropion is often used in patients who are concerned about gaining weight after quitting. Insomnia, agitation, and dry mouth are the most common side effects of bupropion. MOA not fully understood

Bulimia Nervosa

- Characteristics: binge eating + vomiting / laxatives/ diuretics / exercise to avoid gaining weight
o occur 1 day/week for 3 months
o causes emotional distress/loss of control
o maintain normal body weight or may seem overweight; rapid fluctuations in weight = characteristic
o severity: mild = 1-3 episodes/week, moderate = 4-7; severe = 8-13; extreme: >14
o F > M, onset age 15-30; more prevalent than anorexia; more common to seek treatment
o Egodystonic (upsetting to pt.)
  o High achievers, respond to societal pressure to be thin, increased rate anxiety and mood disorders, bipolar I, impulse control, and history of sexual abuse

s/s: dental erosion, esophagitis, callused or abraded knuckles, hypochloremic, hypokalemia, metabolic alkalosis, hypomagnesium, hypocalcemia, salivary gland hypertrophy, cardiac arrhythmias, elevated amylase, gastric distention

• treatment:
  o first line = CBT
  o prognosis = better than anorexia bc more likely to seek treatment (egodystonic / less denial)
  o 1. Restore nutritional status
  o Antidepressants (SSRIs – fluoxetine) = useful; TCAs and MAOIs may be effective but s/e limit use so not first line
  o Avoid bupropion d/t lower seizure threshold
  o Behavioral psychotherapy used with family therapy; group therapy considered; holistic approach
  o Hospitalization usually not necessary except with SI or metabolic / electrolyte disturbances d/t severe purging

Bulimia nervosa is an eating disorder characterized by recurrent episodes of binge eating and inappropriate compensatory behavior such as self-induced vomiting or laxative abuse. For diagnosis, the behaviors must occur at least once per week for 3 months. Individuals feel out of control with their behaviors and are overly concerned with their physical appearance and weight. Treatment for bulimia nervosa includes psychotherapy, nutritional rehabilitation, pharmacotherapy and management of medical complications. The selective serotonin reuptake inhibitor (SSRI) fluoxetine is the only SSRI approved for use in treating bulimia nervosa.

Hypokalemia, contraction alkalosis

Major Depressive Disorder
• definition: depressed mood + anhedonia with >5 assoc sx almost every day, most of day, 2 weeks
  1. fatigue, insomnia or hypersomnia, guilt, worthlessness, recurring thoughts of death or suicide, psychomotor agitation, significant weight change >5%, decreased concentration / indecisiveness
  2. somatic: constipation, HA, skin changes, chest or abdominal pain, cough, dyspnea
  3. ^^ sx cause clinical distress / impairment in social, occupational or other important areas of functioning
  4. NO MANIA OR HYPOMANIA; STRONG FAMILY HX COMPONENT
• Screening: Beck Depression Inventory for Primary Care
• Childhood maltreatment has been associated with a greater risk of relapse or recurrence of major depressive disorder after successful treatment. The rate of recurrence over twenty years is approximately 40 percent. The risk of recurrence is greatest within the first few months after treatment.
• Management:
  1. Psychotherapy: 1st line in mild to moderate depression – 15% commit suicide (esp in M 25-30 / F 40-50yo)
  2. Medications: SSRI = 1st line, SNRI, TCA
  3. CBT: exposure and response prevention, psycheducation, support groups
  4. ECT in pts who fail medical therapy, previous response to ECT, rapid response in pt with severe sx
• New diagnosis: look for diabetes / lipid disorders
• SIGECAPS (depression)
  ▪ S: sleep changes
  ▪ I: Interest lack thereof
  ▪ G: Guilt excessive
  ▪ E: Energy lack
  ▪ C: Concentration Decrease
  ▪ A: Appetite altered
  ▪ P: psychomotor dysfunction (agitation)
  ▪ S: Suicidal Thoughts
• A major depressive episode must consist of either persistently depressed mood most of the day, nearly every day for at least two weeks or loss of interest or pleasure in most activities, nearly every day for at least two weeks.

Suicide
• **Depression and suicidal ideation** are common complaints seen in the primary care setting. Any patient who reveals having thoughts of suicide should be assessed further to determine more details about the thoughts of suicide, as well as intent and plan. Risk factors for suicide include psychiatric illness, history of previous suicide attempts, individuals who have never been married, previous or active military service, childhood abuse, family history of suicide, and access to weapons. Women attempt suicide twice as often as men, but men are three times more likely to be successful. Management of a patient who is suicidal includes risk factor reduction, managing the underlying cause, close monitoring, and follow up. Determination of the lethality of the patient’s current medication regimen is part of the risk reduction process. **Selective serotonin reuptake inhibitors (SSRIs)** seem to be safer in the case of an overdose than other agents. SSRIs, such as fluoxetine, are therefore the agents of choice in the treatment of depression for patients who are potentially suicidal.

- **Increased risk = SADPERSONS:** male Sex, Age, Depression, Previous attempt, Excess alcohol or substance abuse, loss of Rational thinking, lack of Social supports, Organized plan, No spouse, Sickness
- **Highest rate = Caucasian men, age >85**
- **any patient presenting with a possible suicide attempt should have an emergent psychiatric evaluation**
- **always get acetaminophen level in pt. with overdose – can present without sx early on and is in lots of meds**
- **girls more commonly attempt compared to boys**
- **percentage of students in grades 9 through 12 reported that they had seriously considered attempting suicide in the 12 months preceding the survey = 14.5%**

---

**Generalized Anxiety Disorder**

- **definition:** excessive anxiety or worry a majority of days / more days than not in a 6-mo period associated with >3: fatigue, restlessness, difficulty concentrating, muscle tension, sleep disturbance, irritability, shakiness, HA
- **management:**
  1. antidepressant: SSRI (paroxetine and escitalopram); SNRIs – venlafaxine
  2. buspirone (buspar): simulates serotonin receptors and blocks dopamine – may take weeks for improvement; does not cause sedation; s/e: dizzy, nervous, nausea
  3. benzodiazepines (short term); beta blockers
  4. psychotherapy

Generalized anxiety disorder (GAD) is a common psychiatric disorder often seen in the primary care setting. It is characterized by **excessive and persistent worrying that occurs more days than not for six or more months.** Other clinical manifestations include insomnia, headaches, difficulty relaxing, and fatigue. The anxiety symptoms experienced with GAD are difficult to control and cause significant distress and impairment in activities of daily living. **GAD is two times more common in women than in men** and is the **most common psychiatric disorder seen in the elderly.** Diagnosis is determined using the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria.* Recommended treatment is with a combination of pharmacotherapy and psychotherapy.

- Serotonergic antidepressants, such as sertraline, are prescribed for generalized anxiety disorder in doses equal to those used in major depressive disorder.

---

**Panic Disorder**

- **criteria:** sx not due to substance, medical condition or other mental disorder, recurrent, unexpected expected attacks (at least 2) not related to trigger, familial trait, panic attack followed by concern of more attacks, worry about implication of attacks, significant change in behavior related to attacks
  1. **panic attacks:** feature of many different anxiety disorders but not disorder itself
     - episode of intense fear or discomfort with 4+ following sx developing abruptly, reaching peak in 10 minutes – palpitations, trembling, sweating, choking, SOB, chills, dizzy, nausea, chills, hot flashes, paresthesias, fear of dying, losing control
     - **management:** benzos for acute attack
  2. **dominant symptoms:** sudden onset palpitations, chest pain, choking sensation, dizziness, feelings of unreality, secondary fear of dying, losing control, going mad
  3. **Childhood exposure to sexual or physical abuse and childhood smoking increases the risk of developing panic disorder in adulthood.**
  4. **alcohol use MC associated with panic disorder**
- **management:**
  1. SSRI 1st line long term tx: paroxetine, sertraline, fluoxetine
  2. Benzo: for acute attack; TCAs (imipramine)
  3. CBT – focus on thinking / behaviors (relaxation, desensitization, examine behavior consequence) = most effective, in fact just as effective as anti-depressants
• Check labs first: TSH, CMP, CBC

**Phobic Disorders**

- **General characteristics:** irrational fear and disproportionate excessive anxiety when presented with object / situational event; exposure = immediate increased anxiety and can → panic attack; all → avoidance / apprehension; pt. knows fear is excessive / unreasonable (insight)
- **dx:** made if response to phobic stimuli interferes with daily routine, social functioning, or occupational functioning
- **comorbidities:** MDD, substance abuse, other anxiety disorders, personality disorders
- **Specific phobias:** More common than social phobias; last 6+ months; fear of specific object / situation
  1. Animal: fear of specific animals or insects
  2. Natural environment: fear of natural phenomena (storm, height, water, lightening)
  3. Blood injection injury: fear of needles or invasive procedure
  4. Situation: fear of specific situations (bridges, tall buildings, flying, driving, elevators)
  5. Other: fear of situations → choking, vomiting, illness in children; fear of loud noises / clowns
- **Agoraphobia:** intense anxiety about placing oneself into situation in which incapacitating problem could occur and no help would be available; fear of public or crowded places; may occur with or without hx of panic disorder → avoid situations
  1. Dx criteria: any of sx that are characteristic of panic attack may be present; pt. may have incapacitating or embarrassing rex (lack of bowel / bladder control); duration 6+ months
  2. Sx may render pt. unwilling / unable to leave home

This patient has developed a phobia as the result of hearing stories about her cousin, an example of informational transmission, a cognitive factor in the development of a phobia.

**Tx:**

1. **SSRI** (paroxetine, fluoxetine, sertraline, venlafaxine) = 1\(^{st}\) line – 2. if unsuccessful → benzo 3. TCA (imipramine) but this is less effective
2. **B-blockers** (propranolol) – reduce autonomic hyperarousal sx and tremor associated with performance situations
3. Insight-oriented therapy + graded exposure – systematic desensitization + exposure therapy = most effective
4. Specific phobias can be treated with short-term benzo and b-clocker as adjuncts

**PTSD**

- **MC in young adults – combat, violence, rape, assault**
- 1. Exposed to traumatic event and actual / threatened death / injury or violation to self or others  2. Response may involve helplessness, dissociative sx, avoidance of associated stimuli, emotional numbing, increased autonomic arousal
- **Criteria:** trauma is re-experienced: >1 month recollections, distressing dreams, acting/feeling as if event were recurring, physiologic distress, and avoidance of related stimuli (thoughts, feelings, conversations)
  1. Re-experiencing trauma: intrusive memories, dreams, flashbacks
  2. Avoidance: social withdrawal, emotional detachment, sense of foreshortened future
  3. Hyperarousal: insomnia, irritability, difficulty concentrating, exaggerated startle response
- **Management:** 1. **Antidepressants** (SSRIs = first line → paroxetine, sertraline, fluoxetine, TCA’s, MAOIs); **CBT** (psychotherapy, counseling)
  1. Selective serotonin reuptake inhibitors are used as first-line therapy for post-traumatic stress disorder (PTSD) in combination with cognitive and behavioral therapies. PTSD is a severe disorder characterized by intrusive thoughts, sleep disturbance, nightmares, and hypervigilance as a result of a traumatic experience or event. Pharmacologic treatment is used to decrease the severity of the symptoms. Selective serotonin reuptake inhibitors (SSRIs) are used as first-line therapy because they have been proven to most effectively decrease the symptoms of PTSD.

**Acute Stress Disorder**

- Similar to PTSD but sx <1mo; anxiety as a result of extraordinary life stress event
- Treatment: counseling / psychotherapy; if persistent tx as PTSD

**Bipolar I Disorder**

- **Definition:** >1 manic episode and occasional major depressive episodes
- **r/f:** family history = strongest risk
- **mania:** abnormal and persistently elevated, expansive or irritable mood at least 1 week with marked impairment of social / occupational function – 3+:
  1. **mood:** euphoria, irritable, labile, dysphoric
thinking: racing, disorganized, expansive, grandiose

behavior: physical hyperactivity, pressured speech, decreased need sleep, increased impulsivity / risk taking, increased goal directed activity

DIGFAST: distractibility, impulsivity (poor judgement, spending sprees, reckless driving), grandiosity (increased self esteem), flight of ideas (racing thoughts), activities (psymotor agitation), sleep (decreased need), talkativeness (pressured speech)

• screening: mood disorder questionnaire
  o When staff-assisted mental health care is available, screening for depression and bipolar disorders is recommended for patients 12 to 18 years of age

• treatment: 1. mood stabilizer – 2nd or 1st generation anti-psychotic – may add SSRI for depressive sx 2. Good sleep hygiene 3. Cognitive, behavioral, and interpersonal therapy

Bipolar II Disorder

• definition: hypomania + major depressive episode
  o hypomania: period of elevated, expansive or irritable mood at least 4 days that is different from usual non-depressed mood but does not cause marked impairment (no psychotic features) – 3+:
    ▪ mood: euphoria
    ▪ thinking: racing, disorganized, expansive, grandiose
    ▪ behavior: physical hyperactivity, pressured speech, decreased sleep need, excessive involvement pleasurable activities

• screening: mood disorder questionnaire

• management:
  o mania: lithium, valproate, 2nd generation antipsychotics
  o depression: lithium, valproate, carbamazepine, 2nd gen antipsychotic
  o mixed: atypical antipsychotic, valproate

SUBSTANCE RELATED DISORDERS

• General characteristics: MC abused drugs = alcohol, nicotine, caffeine

• Substance use: inappropriate use of a substance resulting in significant impairment → 2+ of the following 11 maladaptive behaviors in a 12 month period
  1. Tolerance: decreased effect over time when same amt. substance used or need to increase to get to baseline
  2. Withdrawal: sx with onset closest to cessation of substance
  3. Use of increasingly larger amounts of substance or over over longer period than intended
  4. Unsuccessful efforts to stop or decrease amount of substance used
  5. Significantly more time spent attempting to acquire or use substance or recover from its effects
  6. Continued use of substance despite awareness of adverse consequences (fails to meet home / school / work obligations, repeatedly uses substance n hazardous situations, cravings / strong desire to use, continues to use despite interpersonal / social problems)

• Intoxication: maladaptive behavioral or psychological changes attributed to recent ingestion of substance; reversible and not caused by mental disorder / medical condition)

• Severity: mild (2-3 sx), moderate (4-5 sx), sever (6+ sx)

• Epidemiology: lifetime prevalence = 17%; most likely age group = 18-24 M>F; in US substance abusers have 3x greater risk having mental disorder; r/f measured through CAGE questionnaire (below)

• Marijuana, cocaine, hallucinogens = most commonly used

Alcohol Related Disorders

s/s: intoxication = slurred speech, ataxia, facial flushing, erratic behavior, loss of inhibition, euphoria
  o chronic abuse: acne rosacea, palmar erythema, hepatomegaly, dupuytren contracture, testicular atrophy, gynecomastia

• screening: CAGE – cut down, annoyed / criticized for drinking, guilty, eye opener

• alcohol use d/o = strong urge / craving to use alcohol

• diagnostics: elevated GGT (early sign), ALT, AST, lactate dehydrogenase, MCV, decreased BUN, decreased LDL and red blood cell volume

Alcohol Withdrawal Syndrome

- Tremulousness
- Hallucinations
- Seizure
- Delirium tremens
• **withdrawal sx**: shakes, jitters 8-18 hours after stopping, peak 24-48 hours; abnormal perceptions, n/v (8-18 hours), seizures, hallucination (within 2 days), delirium tremens (2-3 days but can occur up to a week after)
  - 12-24 hours: Irritable, diaphoretic, tachycardic, insomnia, tremor, autonomic hyperactivity
  - 24-48 hours: Seizure
• patients may develop **alcohol withdrawal with mild symptoms, alcohol related seizures** or in the most serious and life-threatening form of withdrawal, **delirium tremens**. The patient described here has **several abnormal vital signs** (fever, tachycardia, hypertension). These abnormalities are concerning for **major alcohol withdrawal** which is a constellation of symptoms which may include **anxiety, irritability, tremors, tachycardia, fever, hypertension, decreased seizure threshold** and both **auditory and visual hallucinations**. In its most severe form, patients develop delirium tremens, which is a severe hyper-adrenergic state with confusion, hallucinations and hemodynamic instability. This condition is life-threatening and requires aggressive treatment with benzodiazepines and possibly antipsychotics.
  - In ED: give glucose, thiamine (to regenerate NAD), fluid repletion, supportive measures
• **tx:**
  - **nonpharm**: education, coping skills, relaxation therapy, family therapy, psychotherapy, health and nutritional counseling, AA
  - **pharm**:
    - withdrawal = benzos (diazepam = chlordizepoxide or Librium) + folic acid and MVI, thiamin may prevent Wernicke’s encephalopathy; antipsychotic may be indicated for alcoholic halucinosis
    - disulfiram (Antabuse) – alcohol-deterrent that causes nausea with consumption
    - naltrexone: maintenance therapy (decrease cravings and perhaps relapse rates)

### Cannabis Related Disorders
- mild euphoriants with some sedative effects
- **MOA**:
  - Marijuana is the most widely used illegal psychoactive substance in the world. **Its psychoactive properties come from delta-9-tetrahydrocannabinol** which is chiefly found in the flowering heads of the female plant. **Delta-9-tetrahydrocannabinol is a partial agonist at both the cannabinoid 1 and cannabinoid 2 receptors**. Cannabinoid 2 receptors are found on immune cells and some neurons, and stimulation of these receptors does not lead to positive reinforcing or rewarding effects. In contrast, cannabinoid 1 receptors are located throughout the body, including the dopaminergic mesolimbic brain circuit, otherwise known as the body’s brain reward system. It is the stimulation of these receptors which leads to the potential for abuse of marijuana. Cannabis can be smoked, inhaled as a vapor, or ingested orally. Smoked and inhaled cannabis has a rapid onset of action, while ingested cannabis has a slower absorption and leads to less intense effects. **The potency of cannabis is determined by the ratio of delta-9-tetrahydrocannabinol to cannabidiol contained in the substance**. Cannabidiol is not psychoactive and tends to inhibit or lessen the effects of delta-9-tetrahydrocannabinol, so the lower the ratio of delta-9-tetrahydrocannabinol to cannabidiol, the lower the potency of the drug. Negative health consequences of cannabis use include decreased memory, attention, and concentration. Some users may also experience transient psychosis. A cannabis use disorder, similar to that seen in other drugs of abuse, can develop. Not all users of marijuana develop cannabis use disorder. Genetic predilection and environmental factors play important roles in determining those who will progress from recreational use to cannabis use disorder.
- **s/s intoxication**: disconnected speech, recent memory impairment, emotional lability, depersonalization, confusion, increased HR, conjunctival injection, decreased body temp
- **adverse reactions**: panic, psychosis, depression (rare) – chronic psychotic states secondary to cannabis use have been reported in eastern cultures where doses are presumable much higher
  - amotivational syndrome: low drive, poor judgment, introversion, loss of insight, poor communication skills, depersonalization – occurs in people who use marijuana heavily on regular basis for many months / years
  - no clear whether heavy cannabis use causes or results from this condition of low motivation
  - adverse effects of intermittent use = not study
• **withdrawal does not require medication but anxioytics can be used**
  - **Withdrawal** usually occurs within 24 to 48 hours of stopping the drug, and symptoms include malaise, irritability, insomnia, diaphoresis, night sweats, GI disturbance, and drug craving. The withdrawal symptoms usually peak by day 4 and are resolved by day 10-14.
  - CBT and motivational incentives are successful
• Detectable in urine for 1 month

### Hallucinogen Related Disorders
- psilocybin (shrooms), mescaline (peyote), LSD, DMT
• induce altered states of awareness that resemble those of natural psychoses
• Phencyclidine (PCP) is a hallucinogenic drug that can be insufflated, smoked, ingested, or injected. It can cause violent or bizarre behavior, horizontal and vertical nystagmus, disorientation, and auditory hallucinations. If the intoxication is mild, supportive care is usually sufficient to manage the patient. Should the patient be agitated or violent, the first-line treatment is benzodiazepines.
• s/s intoxication: alteration of mood (euphoria), vividness of real or fantasied sensory illusions and hallucinations, synesthesia (overflow from one sensory modality to another) confusion, time slowing, loss of body boundaries, grandiosity, omnipotence
• adverse reactions: acute panic attacks, psychosis, flashbacks, precipitation of underlying psychosis
• LSD intoxication: dilation of pupils, increased deep tendon reflexes, muscle weakness, HTN, tachycardia, fever
• Tx: supporting and reassuring person and diminishing stimulation around the person until it wears off → quiet room, talking to help distinguish psychotic sx from reality; severe panic = oral diazepam; avoid antipsychotic medication due to adverse anticholinergic reactions from hallucinogen + antipsychotic

Inhalant Related Disorders
• Definition: substances that contain mind-altering properties when inhaled (huffing / sniffing) – high only lasts several minutes (glue, aerosol, shoe polish, gas, lighter fluid, leather cleaner, paint thinner)
• Adverse reaction: seizure, coma, death; addiction uncommon but possible
• Sx: belligerence, aggressiveness, apathy, euphoria, impaired judgement, dizziness, poor coordination, slurred speech, unsteady walk, lethargy, slow movement / reflexes, muscle weakness, tremor, blurred vision, stupor, com; clears within a few minutes to hours after exposure
• erythematous rash about the mouth is a common finding in patients inhaling solvents (glue, paint thinner, lacquer). It is usually caused by a contact dermatitis to the solvent and other chemicals in the substance abused and may be associated with a secondary bacterial infection. Additional clinical features include mood swings, erratic behavior, headache, nosebleed, facial flushing, salivation, visual changes, nausea, vomiting, anorexia, unusual breath or body odor, coughing, wheezing, tachycardia, dysrhythmia, slurred speech, ataxia, disorientation, tremor, loss of consciousness, hallucinations, nystagmus, and poor attention. Chronic pulmonary, neurologic, psychiatric, cardiovascular, hemotologic, renal, and hepatic disorders may result from prolonged abuse. Diagnosis is established by a thorough history and physical. Acute treatment is largely supportive, though respiratory, hemotologic, renal, hepatic, and cardiovascular complications may require more intensive treatment. Ongoing psychiatric and primary care are paramount in long-term treatment, as additional behavioral and psychiatric comorbidities are common, including abuse of other substances.
• Long/term effects: damage kidney, liver, nerve fibers, brain cells
• Tx: educational campaigns; treat the seizure, CBT, individual / family therapy

Opioid Related Disorders – heroin, oxycodone, codeine, fentanyl, morphine
• Intoxication s/s: drowsiness, impaired concentration, bradycardia, hypotension, constricted pupils, slurred speech, flushing
  1. Hypoventilation and respiratory depression, CNS depression, miosis
• Withdrawal s/s: lacrimation, rhinorrhea, sweating, yawning, anxiety hypertension, tachycardia, n/v, abdominal cramps, muscle/joint pain, mydriasis, lacrimation
  1. Adrenergetic hyperactivity (CNS excitation, tachypnea, tachycardia, hypertension)
  2. GI sx: abdominal cramping, n/v/d
  3. Mydriasis
  4. Yawning, lacrimation
  5. Give clonidine for withdrawal
• Tx:
  1. Naloxone to reverse effects
    • Support the airway!
    • Provide supplemental oxygen before administering naloxone
    • Naloxone (pure opioid antagonist)!!!
  2. Slow taper methadone or clonidine with adjuncts (ibuprofen for muscle cramps, loperamide for diarrhea, promethazine (anti-histamine / anti-nausea – Phenergan) or dicyclomine (bentyl) for GI distress; benzos for mild withdrawal
  3. Ongoing maintenance: methadone, naltrexone, buprenorphine, or combination of the latter with naloxone

Sedative, hypnotic, or anxiolytic related disorders – “downers” that affect CNS; most = prescription drugs (benzos, barbiturates → prone to dependence)
• Longer term use of 10-40 mg benzos can result in physical dependence
**Overdose:** sx of intoxication and overdose mimic drunkenness – drowsiness, slurred speech, lack of coordination, memory impairment, confusion, nystagmus, moodiness, faulty judgement – occur in much higher doses with benzos than barbiturates but alcohol + benzos can cause OD
  - **OD tx:** induce vomiting or gastric lavage to clear stomach; send blood / urine / gastric content for tox analysis; monitor and support respiratory / cardiac function for at least 24 hours
  - **Comatose / semi-comatose:** attempt gastric lavage if drug taken <12 hrs earlier, alkalinize urine to increase excretion; support life function via intubation, oxygen, plasma expanders, vasopressors

**Withdrawal:** seizures and cardiovascular collapse and death; sx = agitation, anxiety, anorexia, vomiting, increased HR, postural hypotension, hyperreflexia, tremor, seizures, delirium, hyperthermia, cardiovascular collapse
  - **Withdrawal tx:** substitution of barbiturate or long acting benzo (diazepam) for abused DNS depressant with gradual tapering
  - If dependent on both opioids + barbiturates, barbiturate withdrawal is carried out first
  - May take several weeks to d/c benzo with careful monitoring

**Stimulant Related Disorders** – caffeine, cocaine, amphetamines/methamphetamine, pseudoephedrine, diet pills
  - **Acute intoxication:** agitation / aggression, impaired judgment, euphoria, elevated blood pressure, transient psychosis, tachycardia, dilated pupils, hallucinations
  - **Withdrawal s/s:** fatigue, depression, headache, profuse sweating, muscle cramps, hunger
  - **Tx:** benzos to reduce agitation; short-term antipsychotics for psychotic sx

**Tobacco Related Disorders:**
  - **Definition:** nicotine – changes brain to cause cravings → use / withdrawal; MC substance use disorder in the US; most preventable cause of disability and early death
  - **Sx:** impairmen or distress within a 12 mo period bc of tobacco use, taking tobacco in larger amounts or over a longer period of time than originally intended; having strong craving / urge to use tobacco; having strong desire to cut down; making unsuccessful efforts to do so; spending a lot of time trying to obtain / use tobacco products; use tobacco despite problems ti causes in major areas of life; using tobacco in situations where it’s hazardous; increasing amount to reach desired effect
  - **Withdrawal sx:** irritability, anxiety, difficulty concentrating, increased appetite, depressed mood, insomnia; peak 2-3 days after abstinence and last 2-3 weeks.
  - Those with alcohol / substance use disorders have lower rate quitting; family link
  - **Tx:** pharmacological, behavioral, psychosocial – patch, gum, Chantix
    - **Integrated / combined treatment**
    - **nicotine replacement therapies** (patch, gum, lozenge, inhaler, nasal spray) – low cost but less effective than varenicline and bupropion; more steady delivery nicotine than cigs
    - **Chantix** (varenicline) – relieve craving and withdrawal, reduce reinforcing effects of nicotine – most effective of pharm interventions – BB warning for cardiovascular adverse events; may cause depressed mood, agitation, suicidality
    - **Buproprion** (zyban) – antidepressant that reduces cravings and other withdrawal effects – effective but less than Chantix; do not use with sseizure disorders, current use of bupropion, MAOIs, electrolyte abnormalities, eating disorders
    - **Other - Individual psychical intervention – counseling, motivational interviewing, CBT; Group support; Mobile technologies; Organization interventions**

**Intimate Partner Abuse / Domestic Abuse**
  - 24% women and 12% men experienced domestic violence in their lifetime
  - Increases during pregnancy and postpartum
  - More common among black, Hispanic, native American women
  - when confronted with pt. who may be a victim: 1. Immediate medical attention to address physical sequelae, 2. Recognition of suspected abuse / engagement of pt. with nothreatening questioning to confirm whether abuse has occurred 3. Provision of contact information for referral agencies and make referral immediately
• precautions: present options; woman who leaves abusive partner has 70% greater risk of being killed by batterer than woman who stays
• battered victims have suffered blow to their eg defenses may not be assertive enough to believe that their rights have been violated
• not uncommon to find battered women who believe they deserved beating
• treatment:
  o medical attention to address physical needs; recognition of abuse + non-threatening questioning; contact numbers for referral agencies; present options
  o victim and children: open-ended questions → more direct; supportive psychotherapy in safe environment
  o abuser: referral for therapy, treatment for substance abuse / concurrent psychiatric problems, gradual reuniting with family

Urology / Renal

Balanitis

• Patient will be an uncircumcised male
• Complaining of burning and itching of the penis
• PE will show erythema and inflammation with scant white discharge
• Most commonly caused by Candida
• Treatment is topical antifungal, antibacterial or improved hygiene
• Comments: Recurrent balanitis is seen in diabetics
  o clotrimazole, adequate hygiene and glycemic control

Benign Prostatic Hyperplasia

• proliferation of fibrostromal tissue of prostate → urethral compression → obstruction of urinary outlet
• features: decreased force of urinary stream, hesitancy and straining, postvoid dribbling, incomplete emptying, frequency, nocturia, urgency, recurrent UTIs
• exam: DRE = enlarged prostate
• dx: PSA slightly elevated
• tx: watchful waiting / frequent monitoring
  o meds: alpha antagonist (prazosin) / phosphodiesterase-5 inhibitors (tadalafil, vardenafil), anticholinergic agents
  o behavioral: limit fluids before bed
  o procedures: balloon dilation, microwave irradiation, stent placement
  o surgery: transurethral resection of prostate or transurethral incision of prostate

Chlamydia

• MC cause nongonococcal urethritis; discharge is less painful than gonococal and usually watery
• Females usually asymptomatic and may develop cervicitis, salpingitis, PID
  o Infection with chlamydia is a leading cause of infertility
• Dx: clinically and presumptive, gram stain negative; DNA probes may help confirm
  o Test for coinfection with gonorrhea
• Tx: azithromycin, doxycycline, erythromycin = effective (erythromycin = drug of choice in pregnancy women); treat all partners

Cystitis

• infection of normal bladder MC caused by bacteria (E.coli) = 80-85% of cases; infection usually ascends from urethra
• features: frequency, urgency, dysuria, suprapubic tenderness
  o often appear following sexual intercourse in women
• exam usually unremarkable – sometimes suprapubic tenderness
• dx: UA = pyuria, bacteriuria, +/- hematuria
  o urine culture positive for offending organism
  o imaging only warrented in pyelo, recurrent infection ,anatomic abnormalities
• tx:
uncomplicated: short-term abx: Bactrim or nitrofurantoin 3-5 days, FQs reserved only for people with no alternative options
  - resistant to E.coli = use bactrim
  - rare in men
- increase fluids, prevention (proper hygiene, void after intercourse)
- hot sitz baths / urinary analgesics (phenazopyridine / Azo) may provide sx relief (turns pee orange)

**Epididymitis**
- acquired by retrograde spread of organisms through vas deferences
  - <35 = chlamydia and gonocci = MC organism
  - >35 = E.coli
- Features: heaviness / dull, aching discomfort in scrotum that can radiate up ipsilateral flank
  - Swollen epididymis / tender; fever / chills
  - Positive prehn sign (pain relief with elevation)
- Dx: UA = pyuria and bacteriuria; cultures are positive for suspected organisms
- Tx:
  - <35: ceftriaxone 250mgIM + doxy 100mg bid x10 days
  - >35: cipro 500 mg bid 10-14 days
  - Supportive care: bed rest, scrotal elevation, analgesics

**Glomerulonephritis**
- Damage of renal glomeruli by deposition of inflammatory proteins in glomerular membranes as result of immunologic response
- 60% in kids; excellent prognosis in kids and worse in adults esp with preexisting renal disease
- Cause: hematuria, Henoch-schonlein purpura, postinfectious GN, IgA nephropathy, hereditary nephritis and others
- Features: hematuria, urine = tea / cola colored, oliguria / anuria, edema of ace and eyes in the morning and of the feet/ ankles at night; HTN is common
- Dx: antistreptolysin-O titer is increased in 60-80% of cases; common cause of GN is streptococcal infection
  - UA reveals hematuria (>3RBCs/high power field) and mishapen RBCs, RBC casts, proteinuria (1-2g/24 hours)
  - Serum complement often decreased
  - Renal biopsy may be done to determine exact diagnosis or severity
- Tx: steroids and immunosuppressive drugs to control inflammatory response; dietary management (salt and fluid intake decreased); dialysis if symptomatic azotemia present
  - Medical: ACE-I = renoprotective (reduce urinary protein loss) in chronic GN
  - Use meds as appropriate for hyperkalemia, pulmonary edema, peripheral edema, acidosis and HTN
- hematuria, RBC casts, proteinuria, HTN, decreased GFR

**Gonorrhea**
- highest incidence 15-29 yo
- incubation period 2-8 days after exposure
- findings:
  - men: burning on urination, serous / milky discharge then 1-3 days later urethral pain more pronounced with yellow, creamy, profuse discharge
    - without treatment → prostatitis, epididymitis, chronic infection
  - women: remain asymptomatic or may develop dysuria, urinary frequency and urgency and purulent urethral discharge; vaginitis and cervicitis are common
    - asx may cause PID and infertility
  - gonococcal bacteremia associated with peripheral skin lesions or septic arthritis of the knee, ankle, wrist
  - conjunctivitis acused by direct inoculation
- dx: gram stain of urethral discharge typically shows gram negative intracellular diplococci
  - culture = essential
- tx: IM ceftriaxone or oral cefixime; doxy + azithro or doxy for chlamydia; treat all partners; reportable infection

**Hernias**
- protrusion of organ or structure through wall that normally contains it; various types can entrap intestines and cause obstruction
- types:
umbilical: congenital and appears at birth; many resolve on own; sometimes need surgery

diaphragmatic / hiatal: protrusion of stomach through diaphragm via esophageal hiatus → can cause GERD – tx with acid reduction possibly need surgery

incisional hernias: associated with vertical incisions, esp with obesity

inguinal:
  - indirect (MC) → passage of intestine through external inguinal ring at inguinal canal, may pass into scrotum
  - direct → passage of intestine through external inguinal ring at Hesselback triangle – rarely enters scrotum
  - femoral – least common – passage through femoral ring

ventral: occurs when there is weakening in the anterior abdominal wall and may be either incisional or umbilical

• tx = surgical

Nephrolithiasis

renal calculi – occur throughout urinary tract and are common causes of pain, infection and obstruction

stones: caused by increased saturation of urine with stone-forming salts (calcium, oxalate, and other solutes) or possible lack of inhibitors (citrate) in urine to prevent crystal formation

calcium stones = most common;
  o calcium > uric acid>cystine>struvite

features: asx until inflammation / complete or partial ureteral obstruction develops
  o unilateral back pain, renal colic that waxes and wanes

sx: hematuria, dysuria, urinary frequency, fever, chills, nausea, vomiting

signs: diaphoresis, tachycardia, tachypnea, restlessness, CVA tenderness, abdominal distention

dx: normal serum chemistries (possible leukocytosis)
  o UA = microscopic / gross hematuria – leukocytes / crystals
  o CT without contrast can detect stones as small as 1mm
  o Plain film can identify radiopaque stones
  o Renal U/s: can identify stones in kidney, proximal ureter, or UVJ

Tx: size indicates management:
  o <5mm: likely to pass on own; lots of fluid; strain urine; adequate analgesics
  o 5-10mm: not likely to pass spontaneously; increased fluid and analgesics; elective lithotripsy / ureteroscopy with stone basket extraction
  o >10mm: not likely to pass spontaneously and increased likelihood complications
    ▪ Treated as inpatient if can maintain adequate oral intake; vigorous hydration; ureteral stent / percutaneous nephrostomy = gold standard – use if renal function jeopardized
    ▪ Ample analgesia (toridol / morphine / meperidine)
    ▪ Extracorporeal shock wave lithotripsy (ESWL)

Orchitis

commonly caused by ascending bacterial infection from urinary tract; occurs in 25% of postpubertal males with mumps

features: testicular swelling / tenderness, usually unilateral; fever / tachycardia

dx: UA reveals pyuria and bacteriuria with bacterial infection

tx: if mumps is cause, treat mumps (+ ice / analgesia)
  o if bacteria is cause, treat like epididymitis (ceftriaxone 250mgIM + doxy 100mg bid x10 days if <35 // cipro 500 mg bid 10-14 days if >35)

Prostatitis

ascending infection of gram-negative rods into prostatic ducts

features:
  o acute: sudden onset high fever, chills, low back / perineal pain
  o chronic: variable – asymptomatic → acute symptomatology
  o all forms present with irritative bladder sx (frequency, urgency, dysuria) and some obstruction
  o prostate = swollen / tender
  o avoid vigorous prostate exam in case of septicemia

dx: UA = pyuria; possible hematuria / bacteriuria
  o prostatic fluid = leukocytosis, culture typically positive for E.coli in acute infections
    ▪ chronic usually has enterococcus

• tx: a
  o bx
hospitalization in acute- may need parenteral fluoroquinolones parenterally
- uncomplicated: cipro 500mg bid or levo 500mg qd 2-6 eeks or Bactrim 6 weeks – culture urine 1 week after conclusion of therapy
- if fever doesn’t resolve in 36 hours, suspect abscess and consult urology
- in chronic, FQ for 1-3 weeks is more effect than Bactrim for 1-3 months
- abx not effective for nonbacterial
  - NSAIDs = effective for analgesia; alpha 1 blocker may be helpful if lower UTI sx present
  - Chronic, recurrent, resistant prostatitis with / without prostatic calculi may require transurethral resection of prostate (TURP) for resolution

**Pyelonephritis**
- Involves kidney parenchym and renal pelvis; more common in diabetics and elderly women
  - E.coli = MC / gram negative most common
  - Chronic is the result of progressive inflammation of the renal interstitum caused by bacterial infection – occurs in pt. with anatomic urinary tract abnormalities such as vesicoureteral reflux
- Features: fever, flank pain, shaking chills, irritative voiding symptoms, n/v/d
  - Young children: fever + abdominal discomfort
  - Fever, tachy, pronounced CVA tenderness
- **Dx:**
  - CBC shows leukocytosis and left shift
  - UA shows pyuria, bacteriuria, varying degrees of hematuria, WBC casts
  - Complicated: US shows hydronephrosis secondary to obstruction
- **Tx:**
  - Outpatient: FQ / Bactrim for 1-2 weeks (longer if immunocompromised)
  - Inpatient: IV FQ, 3rd/4th gen cephalosporins, extended spectrum penicillins, gentamycin
  - Failure to respond → U/S / imagine
  - f/u urine cultures not mandatory following tx in uncomplicated cases

**Testicular Cancer**
- MC malignancy in young men age 15-35, r/f: hx of cryptorchidism or previous hx testicular CA
- Features: painless, solid testicular swelling; pt. may also complain of heaviness in testicle
  - Para-aortic lymph node involvement can present as ureteral obstruction
  - Pt. may also present with abdominal complaint from abdominal mass or with pulmonary sx from multiple nodules
- **Dx:** U/S → suspicious intratesticular echogenic focus
  - CT of chest, abdomen, pelvis
  - Elevated AFP or B-HCG are diagnostic for nonseminomatous germ cell tumors
- **Tx:** depends on pathology and stage – surgery, radiation, chemo
  - Orchietectomy is performed for diagnostic and therapeutic reasons

**Urethritis** – see Gonorrhea / Chlamydia
HEMATOLOGY

Anemia

- Defined as low hemoglobin level, RBC count, or hematocrit compared to normal age and gender controls
- Renal failure may cause anemia from lack of epo production (supplement with recombinant epo)
- Anemic pt with corrected retic count over 2 indicated working marrow and RBC loss from bleeding or hemolysis
- corrected retic count <2 indicates bone marrow failure to produce RBCs from lack of epo no nutritions or damaged nonfunctioning marrow
  - Raw retic count x patientHgb/normal Hgb
- s/s: weakness, fatigue, palpitations, increased HR, dyspnea, positional dizziness, syncope, bleeding from any site, increased/new-onset angina
  - May be suspected when physical findings of tachycardia, tachypnea, orthostasis, pallor, jaundice are observed
- Clues:
  - Hx of melena, NSAID use, PUD, anticoagulant use → upper GI bleed
  - Menstrual history
  - Hx of pica = iron deficiency
  - Poor diet: consider iron, vitamin B12, or folate
  - Hx of gastric bypass, distal paresthesias, gait problems, memory issues, metformin use: B12 deficiency
  - Hx of alcohol abuse: folate deficiency / liver disease
  - Family hx of blood cell dx: sickle cell, G6PD, thalassemia
  - Prolonged bleeding, epistaxis, gum bleeding, heavy menses, easy bruising: thrombocytopenia or von Willebrand
- Physical exam:
  - Pallor skin, spoon nails, palmar creases = iron deficiency
  - Petechiae / purpura = low platelet / VWD
  - Vitals: tachycardia form increased cardiac output in anemia, tachypnea from decreased hemoglobin oxygen transport, orthostatic changes if volume depleted or acute bleeding; fever in infections / drug transfusion reactions; hypothermia in hypothyroid state
  - Mouth: angular stomatitis in iron, folate or vitamin B12 deficiency
  - Guaiac positive stool = GI bleed
  - Decreased vibratory and position sense in vitamin B12 deficiency
- Labs: CBC, peripheral smear for red cell morphology, reticulocyte count, UA (proteinuria / hematuria), BMP (elevated indirect bilirubin and LDH indicated hemolysis); elevated BUN and Cr = renal disease
- Microcytic (<80 MCV)
  - MC found in clinical practice:
  - TICS: thalassemia, iron deficiency, chronic inflammatory block, sideroblastic (lead toxicity until proven otherwise)
  - Dx: serum ferritin, iron, TIBC, percent saturation, elevated CRP/ESR;
  - Thalassemia: underproduction of alpha or beta globin chains → deficiency hemoglobin synthesis and RBC hemolysis
    - Alpha: two normal alpha chains = no sx; three normal chains = carries → no sx
      - One alpha chain instead of four = Hgb Hdz
      - All four chains deleted = hydros fetalis
    - Beta: (cooley anemia) – sx begin by 4-6 mo (swith from fetal hemoglobin to adult hemoglobin → severe anemia, growth retardation, abnormal facial structure, pathologic fractures, osteopenia, bone deformities, heptasplenomeg, jaundice
      - Usually die from cardiac failure by age 30 without effective iron chelation and allogeneic stem cell transplant
    - Dx: serum iron and ferritin usually normal or elevated, Hgb usually between 3-6; more marked microcytosis than iron deficiency
    - Tx:
      - mild disease: no iron (risk of overload)
      - Hgb H need folic acid supplements and avoid iron / oxidative drugs
      - B-thalassemia: transfusions to keep Hgb concentrations at least 12
      - Allogeneic bone marrow transplant
  - IRON DEFICIENCY ANEMIAS: MC cause of anemia worldwide
    - Others: GI blood loss secondary to PUD< NSAIDs use, cancer, heavy menstrual blood loss; low dietary intake; decreased absorption
• **Features:** pallor, easy fatigability, irritability, anorexia, tachycardia, tachypnea on exertion, poor weight gain in infants; pica; severe (Hct <25%) may cause brittle nails, cheilosis, smooth tongue, formation of esophageal webs

• **Dx:** Hgb and Hct decreased; hypochromic microcytic red cells; plasma ferritin <20 = iron deficient anemia; serum iron <30 and TIBC elevated; transferrin decreases to <15%

• **Tx:** ferrous sulfate 325 mg tid in slowly escalating dose; best absorbed on empty stomach with vitamin C – constipation = side effect – should see improvement in 2 months but continue up to 6 mo

**WORKUP FOR OCCULT BLOOD LOSS**

- **Chronic inflammation:** chronic infections, neoplastic diseases, autoimmune inflammatory processes (RA, SLE) increase hepcidin which block iron absorption from gut and release of iron from bone marrow
  - **Labs:** 30% microcytic, 70% normocytic; elevated CRP/ESR; normal / elevated ferritin with high percent saturation = typical
  - **Tx:** treat underlying cause to reduce inflammation; transfusion may be required if symptomatic; pt. do not respond to epo or iron supplements

- **Sideroblastic:** acquired disorders with reduced hemoglobin synthesis causing iron accumulation, especially in mitochondria
  - **Causes:** myelodysplasia, chronic alcoholism, lead poisoning
  - **Labs:** Hct = 20-30%; MCV varies; peripheral smear → hypochromic; in lead poisoning, basophilic stippling of red cells may be present; bone marrow and serum lead levels needed for dx
  - **Tx:** chelation therapy for lead toxicity; transfusion if pt. is symptomatic; removal of toxins; B6 (pyridoxine) may result in reticuloytosis and improved Hgb levels

- **Normocytic:** (MCV 80-100)
  - **Causes:** organ failure, impaired marrow function, acute blood loss, chronic systemic disease elevating hepcidin
  - Impaired marrow function possible due to infection, meds, chemo, toxins, radiation, aplastic anemia, infiltrative marrow disease, pure red cell aplasia
  - **Features:** acute blood loss from GI bleeds / trauma; underlying organ failure / chronic dz
  - **Sx:** weakness, fatigue, increased infection, pallor, purpura, petechiae, bone tenderness
  - **Labs:** normochromic normocytic 70% of the time; hypochromic microcytic 30% of the time; elevated CRP/ESR
  - **Tx:** treat underlying dz / inflammation; recombinant epo is effective if treating anemia of renal failure / secondary to chemo; symptomatic aplastic anemia treated with RB transfusion; severe dz treated with bone marrow transplantation or immunosuppression

- **MACROCYTIC:**
  - **Causes:** MC = folate and B12 deficiency
  - **Labs:** B12, folate, RBC folate, MMA, homocysteine, consider TSH, then bone marrow biopsy
  - **Folic acid deficiency:** MC caused by poor dietary intake; also: defective absorption, pregnancy, chronic hemolytic anemias, alcohol abuse, folic acid antagonists
    - **Inadequate intake:** alcoholics, anorexia, diet low in fruit/vegetables
    - **malabsorption rare**
      - Daily requirement of folic acid is 50-100 and usually met by balanced diet
      - **Features:** sore tongue, vague GI sx, no neuro sx
      - **Labs:** lacro-ovalocytes, hypersegmented polymorphonuclear cells = pathognomonic / howel-holly bodies; RBC <150 = diagnostic; serum B12 normal; serum homocysteine elevated
      - **Tx:** oral replacement (1 mg/day) with folic acid = first line tx; avoid alcohol and folic acid metabolism antagonists (Bactrim, seizure meds)
  - **Vitamin B12 deficiency:** pernicious anemia = MC d/t lack of intrinsic factor (necessary for B12 absorption)
    - **Others:** strict vegan diet, gastric surgery, blind loop syndrome, pancreatic insufficiency, metformin, crohn’s
    - Can cause irreversible neurologic damage
    - **Animal foods = B12**
    - Absorption in terminal ileum; storage in the liver
    - **Features:** smooth tongue, glossitis, cheilosis, stocking glove paresthesias, loss of position,fine touch and sensation, balance problems, dementia
    - **Dx:** hypersegmented neutrophils, serum LDH and indirect bilirubin can be elevated; B12 abnormally low, B12 abnormally low; shilling test
    - **Tx:** lifelong supplemental B12, daily oral cobalamin; reversible malabsorption; strict vegans → B12 supplementation, neuro signs treated if within 6 months

- **HEMOLYTIC:** decreased RBC survival and increased cell lysis
  - **HIT:** hereditary, immune attack, trauma
    - **Hereditary:** thalassemias, sickle cell, G6PD
Immune attack: TTP, HUS, DIC

Trauma: burns
- Clinical features: jaundice, delayed puberty, haptosplenomegaly
- Dx: retic count >2 with falling hemoglobin, elevated indirect bilirubin, elevated LDH, immature red cells
  - Indirect coombs / direct coombs to identify antibodies on RBCs
- tx: depends on underlying disorder

Clinical features: jaundice, delayed puberty, haptosplenomegaly

Dx: retic count >2 with falling hemoglobin, elevated indirect bilirubin, elevated LDH, immature red cells

Indirect coombs / direct coombs to identify antibodies on RBCs

tx: depends on underlying disorder

Clothing Disorders

Leukemia

Lymphomas

Polycythemia

- Patient will be complaining of headache, dizziness, pruritus after showering
- PE will show hypertension, splenomegaly
- Labs will show increased RBC mass, overproduction of all cell lines, increased Hgb
- Most commonly caused by mutation of the Janus kinase 2 gene (JAK2)
- Treatment is phlebotomy, hydroxyurea, aspirin

Thromobytopenia

ITP

- Patient will be a child 2 - 6-years-old
- With a history of recent viral infection
- Complaining of red spots on skin or easy bleeding
- PE will show petechiae, purpura, and gingival bleeding
- Labs will show platelets < 50,000 µL
- Most commonly caused by antiplatelet antibodies
- Treatment is observation, steroids, IVIG

INFECTIONIOUS DISEASES

Lyme

- Patient with a history of being in the woods hiking or camping
- Complaining of:
  - Stage I: erythema migrans (pathognomonic), viral-like syndrome (fever, fatigue, malaise, myalgia, headache)
  - Stage II: arthritis, myocarditis, bilateral Bell’s palsy
  - Stage III: chronic arthritis, chronic encephalopathy
- PE will show slightly raised red lesion with central clearing, erythema migrans (bull’s-eye) rash
- Most commonly caused by Borrelia burgdorferi carried by Ixodes tick
- Treatment is doxycycline, children - amoxicillin or doxycycline (if used for < 21 days), pregnant - amoxicillin
- Comments: Bilateral facial nerve palsy is virtually pathognomonic for Lyme disease