Angina Pectoris

• MOA: Insufficient oxygen supply to cardiac muscle, most commonly caused by atherosclerotic narrowing and less commonly by constriction of coronary arteries; CAD = MC cause

• Stable angina: syndrome of precordial discomfort of pressure from transient myocardial ischemia → PREDICTABLE!
  o >70% stenosis; normal troponin/CK-MB; resting EKG normal; during episode >1mm ST depression +/- T wave
  o Diagnosis:
     • Symptoms
     • ECG: ST depression 1mm = positive test; inversion / flattening T waves; normal in 25%
     • myocardial imaging: exercise stress test (most useful / cost effective), echo, coronary angiography
  o Treatment: aspirin, nitrates, B-blockers, Ca channel blockers, ACE-I, statin, coronary angioplasty, CABG

• B-blockers prolong life in patients with coronary disease and are first line therapy for chronic angina

• Unstable: when pain is less responsive to NTG, lasts longer, occurs at rest / less exertion – ANY CHANGE → eval
  o Diagnosis: EKG = normal between attacks; stress test; angiography = gold (assess severity of coronary artery lesions when considering PCI or CABG)
  o Treatment: antiplatelets, B-blockers, NTG, CCB, revascularization, ACE-I, statins

• Prinzmetal: transient coronary artery vasospasm within normal coronary anatomy or site of atherosclerotic plaque
  o Smoking = #1 RF!!
  o Diagnosis: ACS work up (CK, CK-MB, troponins – may be normal / EKG – may have transient STE); coronary angiography with injection of provoking agents into coronary artery = gold
  o Treatment: nitrates and CCB; propranolol = contraindicated

• Variant: transient, abrupt, marketed reduction in luminal diameter of coronary artery → symptomatic MI
• Long-acting nitrate should include daily 8-10 hour tx free interval to prevent drug tolerance

Cardiac Arrhythmias/Conduction Disorders

• Normal sinus: normal rate (60-100) and rhythm; impulse originates in SA node; p waves upright; regular intervals
• Sinus tach: HR >100; exercise, excitement, illness
• Sinus brady: HR 50-60
• Atrial fibrillation/flutter: irritable sites in atria fire rapidly (400-600bpm); rapid pacemaking → atrial quivering; ventricles beat slower bc AV node blocks some atrial impulses
  o Sx: Elderly/alcohol use; syncope, dyspnea, palpitations
  o Dx: EKG: no discrete p waves; irregularly irregular
  o Tx: rate – CCB (diltiazem / verapamil) or beta blocker (metoprolol); rhythm (<48 hrs = cardioversion, amiodarone; >48 hrs = anticoagulate 21 days prior to cardioversion)
     • CHADS2 score (CHF, HTN, age >75, DM, stroke hx) – 2+ points = heparin → coumadin; 1 = aspirin or coumadin; 0 = no therapy or aspirin

• AV block:
  o 1st degree: PR >.2 – delay at AV node or bundle of His
  o 2nd:
     • 1: longer, longer, drop → Wenckebach (some impulses are blocked)
     • 2: some dropped (impulse blocked in bundle of His)
  o 3rd: p’s and q’s have no correlation – no atrial impulses transmitted to ventricles → complete AV dissociation

• Bundle branch block: QRS > .12sec; possible due to MI
  o Left: R and R’ (upward bunny ears) V4-V6
  o Right: R and R’ (upward bunny ears) V1-V3

• Paroxysmal SVT: HR 150-250
  o Paroxysmal SVT – no structural abnormalities; faster than normal HR begins above two lower chambers in atria, AV or SA node
  o AV nodal re-entrant tachy
  o WPW: impulse travels between atria and ventricles through bundle of kent;
     • EKG: bundle of kent fibers and delta wave on EKG; short PR, long QRS, delta wave
     • Don’t give adenosine or CCB
  o MAT: irregularly tachy, narrow QRS, abnormal 3 p waves with different morphology; HR >100
Premature beats: usually benign; may cause palpitations/ increased frequency with caffeine
- PVC = widened QRS; PAC = abnormal p wave earlier than expected; PJC: narrow QRS (<0.10 sec)
- Every third beat = trigeminy; every 2nd = bigeminy
- Tx: none or beta blocker if symptomatic

Sick sinus: dysfunction in sinus node automaticity and impulse generation
- Sinus brady, sinus pause <3 asec; sinus arrest >3 sec; tachy-brady = alternates
- Tx: pacemaker

Sinus arrhythmia: variation in SA nod pacing with phases of respiration; HR increases with inspiration and decreased with expiration

Torsades de pointes: form of Vtach; twisting around a baseline; usually from hypokalemia or hypomagnesemia; tx = IV mag sulfate

Vtach: 3 PVCs in a row = vtach; rate 120-200;
- usually monomorphic
  - Stable: amiodorone / lidocaine
  - Unstable: CPR / defibrillation
- Polymorphpoinc: vary in shape and size

Pacemaker rhythm: vertical spikes of short duration; may be difficult to see in all leads

Cardiomyopathy

Dilated: MC type; an index event or process (MI) damages myocardium, weakening heart muscle → decreased ventricular contraction strength + dilation left ventricle; systolic heart failure
- Reduced contraction strength; large heart; caused by ischemia (CAD, MI, arrhythmia)
- PE: dyspnea, S3 gallop, rales, JVD
- Tx: no alcohol!!!; ACE-I, diuretic

Hypertrophic Obstructive (HOCM): hypertrophic portion of septum; LV outflow tract narrowed – worse with contractility; diastolic heart failure
- young athlete with positive family history sudden death of syncopal episode; inherited; SCREEN FAMILY
- PE: sustained PMI, bifid pulse, S4 gallop; high pitched mid-systolic murmur at LLSB increased with Valsalva and standing (less blood in chamber); decreased with squatting
- Tx: refrain from physical activity; BB or CCB; surgical or alcohol ablation of hypertrophied septum and defibrillator insertion

Restrictive: right heart failure; history of infiltrative process; diastolic heart failure; stiff heart muscle
- Amyloidosis, sarcoidosis, hemochromatosis, scleroderma, fibrosis, cancer
- PE: pulmonary HTN; normal EF, normal heart size, lg atria, normal LV wall, early diastolic filling
- Tx: non-specific; diuretics, ACE-I, CCB

CHF

MC cause: CAD, HTN, MI, DM – LV remodeling → dilation, thinning, mitral valve incompetence, RV remodeling
- Sx: exertional dyspnea → rest, chronic nonproductive cough, fatigue, orthopnea, nocturnal dyspnea, nocturia
- Signs: Cheyne-stokes breathing, edema, rales, S4 (diastolic HF, preserved EF); S3 (systolic; reduced EF); JVD >8cm, cyanosis, hepatomegaly, jaundice
- NY heart failure classification:
  - Class 1: <5% - no limitation of physical activity
  - Class 2: 10-15% - slight limitation physical activity; comfortable at rest
  - Class 3: 20-25% - marked physical limitation; comfortable at rest
  - Class 4: 35-40% - can’t carry on physical activity; anginal syndrome at rest
- Dx: BNP, EKG, CXR (kerley B lines); echo = gold (best to assess size and function of chambers)
- Tx:
  - Systolic: ACE-I + B-blocker + loop diuretic
  - Diastolic: ACE-I + B-blocker or CCB

Coronary Vascular Disease

CAD is #1 killer in USA and worldwide ➤ Death rates ↓ yearly since 1968 – MC cause of cardiovascular death and disability
- RF: smoking, diabetes, dyslipidemia (↑ LDL, ↓ HDL), hypertension, family hx, men <55, women <65
- Dx: high-sensitivity high CRP, lipids, triglycerideyf, carotid U/S
- Tx: Smoking cessation, lifestyle (BP, LDL/HDL, obesity)
- **Primary prevention** = platelet inhibitors (Aspirin, etc.) = cornerstone
- **Secondary prevention**: aspirin, β-blockers, ACE-i/ARB, statins; nitro if symptomatic

**MOA:**
- 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 → plaque clot
- Fibrous plaque forms over lipid core: Complete clot → ST elevation MI; Incomplete clot → unstable angina / NSTEMI
  - Vulnerable plaque is easy to rupture; thick plaque is stable
  - Adhesion, activation, aggregation, propagation of clot, platelet adherence

**Endocarditis**

- Mitral = MC valve involved; M>A>T>P
- HACEK: haemophilus, actinobacillus, cardiobacterium, eikenella, klingella → assoc. with lg. vegetations; IVDU think staph
- 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 (for new arrhythmias), echo (TEE = gold > TTE), CBC
- 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)
- Tx: culture first → 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

**Murmurs**

- **Aortic Stenosis**: harsh systolic ejection crescendo-decrescendo at right upper sternal border with radiation to neck and apex
  - Dyspnea, angina, syncope with exertion; squatting increases intensity; split S2
  - Increased BNP, helmet cells (schistocytes); cardiomegaly
- **Aortic Regurge**: soft high pitched, blowing, crescendo-decrescendo along left sternal border; loud leaning forward / squatting
  - Leaflets of aorta don’t close during diastole → blood regurge from aorta into elft ventricle → volume overload left ventricle
  - S3 or S4 with severe; water hamer pulse (arterial pulse large and bounding)
- **Mitral Stenosis**: diastolic low pitched decrescendo rumbling with opening snap heart best at apex with pt. lying lateral decub
Leaflets of mitral valve thicken, stiffen from rheumatic fever → valve doesn’t open well in diastolic; cause = rheumatic heart
- Left atrial hypertrophy, may also have mitral regurge

**Mitral Regurge:** blowing holosystolic murmur at apex with split S2 radiating to left axilla
- Mitral valve doesn’t close fully in systole → blood regurge from LV to LA → murmur
- Caused by: CAD, HTN, MVP, rheumatic, heart valve infection; apical S3 = volume overload on ventricle

**Mitral Valve Prolapse:** mid-systolic ejection click heard best at apex
- Abnormal systolic ballooning in part of mitral valve into left atrium

**Tricuspid Stenosis:** mid diastolic rumbling murmur at LLB with opening snap
- RARE! Leaflets of tricuspid valve = stiff / immobile → impaired RV filling from decreased tricuspid valve origice = increased RA pressure → right and left heart failure

**Tricuspid Regurge:** high pitched holostystolic murmur at LLB radiates to sternum and increases with inspiration
- Tricuspid fails to close fully in systole, blood regurges from RV → RA = murmur

**Pulmonary Stenosis:** harsh, loud, medium pitched systolic murmur heard best at 2nd/3rd left intercostal space that may increase with inspiration
- Stenosis of pulmonic valve impairs flow across valve; increases afterload on ventricle
- widely split S2; early pulmonic ejection sound; RVH

**Pulmonary Regurge:** high pitched early diastolic decrescendo murmur at LUSB that increases with inspiration
- Blood leaks abnormally backward from pulmonary artery though pulmonic valve → RV (RHF)

**Hyperlipidemia**

**History and Physical exam:** most pt. are asymptomatic; may develop xanthomas; may → pancreatitis (>1000)

**Diagnostic Studies:**
- Evidence of CVD / CHD = fasting complete lipid profile
- LDL goals
  - Pts with CAD or equivalent
    - Start drugs at >130, LDL goal: <100, optimal level <70
  - No CAD but >2 RF
    - Start drugs at >160, LDL goal <130
  - Everyone else:
    - Start drugs >190, LDL goal <160

**Diagnosis / Screening:**
- 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, triglyceride <150

**Clinical Therapeutics:**
- 1. LIFESTYLE CHANGES
- statins: best drug to decrease LDL → inhibits rate limiting step in hepatic cholesterol synthesis (HMGCoA reductase inhibitor); increase LDL receptors (removes LDL from blood)
  - best given at bed time (when cholesterol synthesis is maximal)
    - The only non-statin lipid-lowering agent that has proven to have additive effects on the prevention of cardiovascular adverse events is ezetimibe, which inhibits intestinal absorption of cholesterol. Ezetimibe is administered in an oral daily dose of 10 mg. Common side effects include diarrhea and cough.
- nicotinic acids / niacin (vitamin B3): best drug to increase HDL
- fibrates: best drug to decrease triglycerides (60%)
- bile acid sequestrants: decreased LDL (increased with statins); may increase TG (use in pts with normal TG)
- ezetimibe: inhibit cholesterol transporter; monotherapy or in combination with statin

**Hypertension**

**History and Physical Exam:**
- cardiovascular: coronary artery disease, heart failure, MI, LV hypertrophy, dissection, aneurysms, PVD
o neurologic: TIA, stroke, ruptured aneurysms, encephalopathy
o nephropathy: renal stenosis, sclerosis – HTN 2nd MC cause of end stage renal dz in US
o optic: retinal hemorrhage, blindness, retinopathy

• Diagnosis:
o elevated BP >2 reading on >2 different visits; isolated systolic shows greater risk for CVD than diastolic in pts >50y
o normal: <120/<80; pre-HTN: 120-139/80-89; stage 1: 140-159/90-99 stage 2: >160/>100

• Health Maintenance:
o goal <130/80; bp goals in pt. with CKD: 140/90 (instead of 130/80; bp goal in pt. >60: 150/90

• Clinical Therapeutics:
o Non-pharm: DASH diet, salt restriction, weight loss, exercise, moderate ETOH, pt. education
o 1st line therapy:
o ACE-I (-pril): Cardioprotective, synergistic effect when used with thiazides (decrease preload/afterload); renoprotective (include in CKD)
  ▪ CI: pregnancy; AFRICAN AMERICANS (use thiazides / CCB instead); angioedema; s/e = cough
o ARB (-sartan): consider in pt not able to tolerate beta blockers / ACE-I
  ▪ CI: pregnancy, NOT IN AFRICAN AMERICANS (use thiazides / CCB instead), include in CKD
o CCB
  ▪ Dihydropyridines (nifedipine, amlodipine) – MC used in HTN
  ▪ Non-dihydro (verapamil, diltiazem): affect cardiac contractility and conduction – used with afib
    ▪ CI: pts taking beta blockers / 2nd and 3rd degree HB
o Diuretics (thiazide)
  ▪ HCTZ:
    ▪ tx of choice as initial therapy in uncomplicated HTN; cardioprotective
    ▪ decrease blood volume / blood pressure by decreasing Na/H20 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/H20 absorpction
    ▪ s/e: hyperkalemia, gynecomastia with spironolactone
    ▪ CI: renal failure, hypnatiremia
o 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; CI in bronchospasm
  ▪ Cardioselective (B1): atenolol, metoprolol, esmolol
  ▪ Nonselective (B1, B2): propranolol
  ▪ Both alpha and B1,2: labetalol, carvedilol
o 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

Myocardial Infarction

• STEMI: myocardial necrosis with acute STE or q waves; coronary artery completely blocked; full thickness of myocardial wall involved; elevated troponin I, troponin T, CK, CK-MB (serial troponins – 3 in 24 hours)
o Location:
  ▪ Anterior wall – STE in leads I, AVL, V2-V6
  ▪ Inferior wall: II, III, AVF
  ▪ Lateral wall: lateral leads - I, AVL, V4-6 and reciprocal STD in inferior leads
  ▪ Posterior wall: ST depression in V1-3
o Tx: beta blocker + NTG + aspirin and Plavix + heparin + statins + reperfusion
  ▪ Door to balloon time = 90 minutes!!!; thrombolytic therapy in first 3 hours if PCI not available
  ▪ CI fibrinolytics: intracranial hemorrhage, ischemic stroke 3 months, suspected dissection, active bleeding

• NSTEMI: myocardial necrosis but coronary artery not completely blocked – rise in cardiac markers without STE or q wave
laboratory tests:
- Troponin: at 2-4 hours, peaks 12-24 hours, lasts 7-10 days
- CK/CK-MB: appears at 4-6 hours, peaks 12-24, returns normal 48-72 hours
- Myoglobin: appears at 1-4 hours, peak is 12 hours, returns to baseline within 24 hours

Treatment: beta blocker + NTG + aspirin and Plavix + heparin + ACE-I + stain + reperfusion (less time sensitive than STEMI)

**Myocarditis**
- Etiology: viral, bacterial, parasitic, cardiotoxin, systemic disorders, radiation, hypersensitivity
- Dx: endomyocardial biopsy = gold; clinical presentation, cardiovascular MRI; echo = decreased ventricular EF with hypokinesis
- Clinical: fatigue, fever, chest discomfort, dyspnea, palpitations, tachycardia disproportionate to fever or discomfort
- Tx: supportive, heart failure treatment prn, antidyshthmics prn

**Pericarditis**
- Causes: SLE, uremia, coxsackievirus, TB, RA, neoplasm, drug, radiation, scleroderma, MI, open heart surgery, radiotherapy
- MOA: inflammation of pericardial sac; often \( \rightarrow \) pericardial effusion
- S/s: pleuritic chest pain worse when supine during inspiration; better when leaning forward; pericardial friction rub
- Dx: diffuse STE in precordial leads; echo may show pericardial effusion / tamponade
- Tx: treat underlying disease!!!
  - NSAIDS 7-14 days; steroids if sx >48 hrs; abx to treat bacterial endocarditis; pericardiocentesis; head at 45 degrees
- Dressler’s syndrome: pericarditis 2-5 days after acute MI

**Peripheral Artery 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 dysfunction
  - aortic bifurcation / common iliac = buttck, hip ground claudicatios
  - femoral artery: thigh / upper calf 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
- 6ps caused by acute arterial embolism: pain, pulselessness, pallor, paresthesias, poiklothermia, paralysis
- Diagnostics: arteriography = gold standard (clinically only done if revascularization is planned); doppler ultrasonography; ankle-brachial index <0.9 (normal = 1-1.2)
  - Falsely high index may indicate severely hardened, non-compressible leg vessels
- Management: risk factor modification: discontinue tobacco, control diabetes, hypertension, hyperlipidemia
- Medications: B-blocker, ACE-I, statins
  - Platelet inhibitors:
    - cilostazol = mainstay of treatment (helpful for intermittent claudication)
    - aspirin
    - clopidogrel (Plavix)
  - tx: aspirin, cilostazole, rosuvastatin, smoking cessation, structured exercise

**Varicose Veins**
- Sx: asymptomatic; aching and fatigue
- Signs: dilated, tortuous veins; greater saphenous = MC; flat, reticular veins; telangiectasia; spider veins
- Diagnostics: duplex ultrasonography
- Management: weight loss, control risk factors; graduated compression stockings
- Interventions: exercise programs, elevation, radiofrequency or laser ablation, compression, sclerotherapy, surgical stripping

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

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

Rheumatic Fever

- Not an infection, but an inflammatory response to infection; caused by acute inflammatory immune response to Group A strep with formation of antistreptolysin antibodies which react with proteins on synovium, heart muscle, heart valves
- Damage from fever → rheumatic heart disease
- Episode occurs 2-4 weeks after strep infection
- Dx: modified Jones: 2 major or 1 major 2 minor along with evidence of GAS infection (elevated anti-strep ab titer, positive throat / rapid ag test)
  - Major: carditis, chorea, erythema marginatum, polyarthritis, subcutaneous nodules
  - Minor: arthralgia, elevated ESR or CRP, fever, prolonged PR on EKG
- Tx: aspirin / NSAID, steroid, abx
  - Prophylaxis against recurrent GAS: penicillin G benzathine 1.2million units IM q3-4 weeks for 5-10+ years (more with heart damage) – some say indefinitely!
- Leading cause of cardiac death among young in developing countries = rheumatic fever

Valvular Heart Disease

Diastolic Murmur: almost always mean heart disease
- Early = regurg flow through incompetent valve (usually aortic)
- Rumbling = mid / late diastole suggests stenosis of AV valve (usually mitral)
  - Aortic regurg: soft, high pitched, blowing diastolic along LSB with pt sitting, leaning forward after exhaling
  - Mitral stenosis: diastolic low pitched decrescendo and rumbling with opening snap at apex
  - Pulmonary regurg: high pitch, decrescendo murmur at LUSB, increases with inspiration
  - Tricuspid stenosis: mid diastolic rumbling at LLSB with opening snap

Midsystolic Murmurs: aka ejection murmurs; MC kind of heart murmur; peak near midsystole and stop before S2; gap between murmur and S2

1. pathologic - secondary to structural cardiovascular abnormalities
2. physiologic – secondary to physiologic alteration in body
3. innocent – not associated with detectible physiologic / structural abnormality
  - Aortic stenosis: systolic ejection crescendo-decrescendo RUSB
  - Pulmonic stenosis: hard midsystolic ejection crescendo-decrescendo murmur with widely split S2 at LSB that radiates to left shoulder and neck
  - HCOM: medium-pitched, mid-systolic murmur that decreases with squatting and increases with straining
    - S4 gallop and apical lift with thick, stiff left ventricle
  - Mitral valve prolapse: midsystolic ejection click at apex

Pansystolic (holosystolic) murmurs – pathologic; heard when blood flows from high to low pressure chamber; begins immediately with S1 and continues up to S2
  - Mitral regurg: blowing holosystolic murmur at apex with split S2
  - Tricuspid regurg: high pitched holosystolic murmur at mid LSB
  - Ventricular septal defect: harsh holosystolic murmur heard at LSB with wide radiation and fixed, split S2

Vascular Disease

- Aortic aneurysm: flank pain, hypotension, pulsatile abdominal mass; screen if male >65 and hx of smoking
  - Tx: immediate surgical repair if >5.5cm or expands >0.5cm per year; monitor annual if >3cm, q6mo >4cm; beta blocker
- Aortic dissection: sudden onset tearing chest pain between scapula; diminished pulses; widened mediastinum; unequal bps on arm
  - Tx: ascending aorta = surgical emergency; descending: beta blocker
- Arterial embolism / thrombosis: sudden arterial occlusion
  - Pain, pallor, pulselessness, paresthesia, paralysis, poiklothermia
  - Angiography = gold standard
  - Tx: IV heparin if not limb threatening call vascular surgeon for angioplasty, graft or endarterectomy
- Giant cell arteritis: inflammation of large and medium vessels – jaw claudication and HA, thickened temporal artery scalp pain elicited by touching scalp / hair brush; acute vision disturbances; associated with polymyalgia rheumatica
  - Amaurosis fugax (temporary monocular blindness) secondary to anterior ischemic optic neuritis
  - Dx: ESR >100, temporal artery biopsy
  - Tx: high dose prednisone URGENTLY – don’t wait for biopsy results

- Peripheral artery disease: intermittent claudication, ABI <0.9
  - s/s: lower extremity hair loss, brittle nails, pallor, cyanosis, hypothermia; ulcers pale to black, PAINFUL, lateral / distal
  - arteriography = gold standard
  - tx: definitive = arterial bypass; medical: antiplatelet, antilipid, manage r/f, diistolazol, aspirin, Plavix

- Phlebitis / Thrombophlebitis: spontaneous / after trauma or IV/PICC lines – dull pain, erythema, induration of vein, palpable cord
  - dx: duplex U/S = gold standard
  - tx: symptomatic: NSAIDs, warm compress

- Venous insufficiency: edema, atrophic shiny skin, brawny induration, stasis dermatitis, brown hyperpigmentation, varicosities, ulcers above medial malleolus
  - dx: ABI, Trendelenburg tests, U/S
  - tx: sclerotherapy, vein stripping, compression hose

- Venous thrombosis: unilateral (asymmetrical) swelling of lower extremity
  - Virchow’s triad: stasis, trauma, hypercoagulability (OCP, cancer, surgery, factor V leiden)
  - Dx: d-dimer, venous duplex U/S = gold standard; Homan sign = discomfort behind knee on forced foot dorsiflexion
  - Tx: heparin to coumadin bridge

PULMONOLOGY

Acute / Chronic Bronchitis

- Acute: cough >5 days; can last 1-3 weeks
  - s/s: fever = unusual; 95% viral (common bacterial = m. catarrhalis; 
  - dx: CXR – if dx uncertain / sx persist despite conservative tx
  - tx: symptomatic / supportive!!!! Hydration, expectorant, analgesic, B2 agonist, cough suppressant
    o abx indication: elderly, underlying cardiopulm dz, cough >7-10 days; immunocompromised
- Chronic: results from enlargement of mucous glands and goblet cell hypertrophy in large airway; 3 months!!!!
  - dx: CXR show interstitial markings; diaphragms not flattened
    o labs: increased Hgb and Hct (chronic hypoxic state); lung biopsy = gold standard
    o PFTs: FEV1/FVC ratio <.7; airflow limitation is reversible or only partially reversible
      - decreased FEV1/FVC, normal / decreased FVC, normal or increased TLC, roughly normal DLCO
  - tx:
    o acute exacerbations: O2, beta-agonist, anticholinergic, inhaled/IV steroids, abx (mild = amox/doxy/bactrim; severe = augmentin, levooquin/cipro)
    o chronic disease: smoking cessation (BEST); ambulatory O2, bronchodilator, steroids, vaccines

Asthma

- chronic, reversible inflammatory airway disease → recurrent attacks of breathlessness and wheezing; FEV1:FVC <80
  - lack of wheezing = EMERGENCY!!!

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<th>Classification of Severity of Chronic Stable Asthma</th>
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<th>Nighttime Symptoms</th>
<th>Use of Rescue Medication</th>
<th>Lung Function</th>
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</thead>
<tbody>
<tr>
<td>Intermittent</td>
<td>Symptoms ≤2 days/week</td>
<td>≤2 times/month</td>
<td>≤2 days/week</td>
<td>FEV1 &gt;80% predicted</td>
</tr>
<tr>
<td>Mild persistent</td>
<td>≤2 days/week but not daily</td>
<td>3–4 times/month</td>
<td>&gt;2 days/week but not daily and not more than once on any day</td>
<td>FEV1/FVC normal</td>
</tr>
<tr>
<td>Moderate persistent</td>
<td>Daily symptoms</td>
<td>≤1 time/week but not nightly</td>
<td>Daily</td>
<td>FEV1 &gt;60% but &lt;80% predicted</td>
</tr>
<tr>
<td>Severe persistent</td>
<td>Continual symptoms</td>
<td>Often 7 times/week</td>
<td>Several times/day</td>
<td>FEV1/FVC reduced &gt;5%</td>
</tr>
</tbody>
</table>

Lung Function:
- FEV1 >80% predicted
- FEV1/FVC normal
- FEV1 >60% but <80% predicted
- FEV1/FVC reduced 5%
- FEV1/FVC reduced >5%
• Tx:
  o Intermittent (<2x/week or <2 night/month) – SABA prn
  o Mild persistent (>2x per week or 3-4 night/month) – low dose ICS dialy
  o Moderate persistent (daily sx or >1 night/week) –
    ▪ Low dose ICS + LABA daily
    ▪ Medium dose ICS + LABA daily
  o Severe persistent (sx several times / day + nightly) –
    ▪ High dose ICS + LABA Daily
    ▪ High dose ICS + LABA + oral steroids
  o acute treatment: oxygen, nebulized SABA, ipratropium bromide, oral steroid, magnesium

Bronchiectasis

• permanent dilation or destruction of bronchial walls; common endpoint of disorders that cause chronic airway inflammation
  (CF, immune defects, recurrent infection)
  o CF = MC (<18 = staph; >18 = pseudomonas)
• Irreversible dilation and destruction of bronchi, inadequate clearance of mucus in airways, cycles of infection and inflammation
• Think: cystic fibrosis, recurrent pneumonia or aspiration, tumor
• Dx: CXR = linear “tram track” lung markings, atelectasis, dilated and thickened airways – “plate-like” atelectasis; CT chest = gold
  o crackles, wheezes, purulent sputum
• Tx: ambulatory oxygen, aggressive abx, CPT (chest physiotherapy = bang on back); eventual lung transplant

Carcinoid Syndrome

• Neuroendocrine tumor that secretes vasoactive material (serotonin, histamine, catecholamine, prostaglandin, peptide)
• MC caused by carcinoid tumor – GI tract cancer that has metastasized to lung (cancer of appendix = MC); adenoma = MC
• Presentation: usually hemoptysis, cough, focal wheezing, recurrent pneumonia
  o Syndrome: Cutaneous flushing, diarrhea, wheezing, low blood pressure (hallmark sign) → RARE
• Dx: CXR – pedunculated sessile growth in central bronchi; elevated 5-HIAA in 24 hr urine excretion (main metabolite of serotonin)
• Tx: surgical excision; lesions resistant to XRT and chemo; ocreotide decreases secretion of serotonin by tumor

COPD

• progressive, largely irreversible airflow obstruction due to 1. Loss of elastic recoil 2. Increasing airways resistance
  o Includes emphysema and chronic bronchitis – both usually coexist with one being more dominant
• 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)
• 30 pack year history = low dose chest CT
• Emphysema:
  o DOE = hallmark symptom
  o Hyperinflation of lungs + hyperresonance to percussion, decreased / absent breath sounds, decreased fremitus, barrel chest (increased AP diameter), quiet chest, pursed lip breathing
  o ABG / labs: respiratory alkalosis
  o Matched V/Q defects, mild hypoxemia, normal CO2
  o Cachectic with pursed lip breathing → “pink puffers”
• Chronic Bronchitis:
  o Productive cough hallmark sx
  o rales (crackles), rhonchi, wheezing, signs of cor pulmonale (peripheral edema, cyanosis)
  o Respiratory acidosis (increased Hct/RBC bc chronic hypoxia stimulates erythropoiesis)
  o Severe V/Q mismatch, severe hypoxemia, hypercapnia
  o Obese and cyanotic = blue bloaters

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

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)
    - **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 and coffee drinkers - don't initiate in acute exacerbation
- 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%

<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>

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
- 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

**Cor Pulmonale**

- Right ventricular enlargement and eventually failure secondary to lung disorder that causes pulmonary artery HTN
  - Caused by COPD (MC), PE, vasculitis, ARDS, asthma, ILD
  - **s/s:** peripheral edema, neck vein distention, hepatomegaly, parasternal lift
  - **dx:** echo or radionuclide imaging; sometimes right heart catheterization
  - **tx:** diagnose and treat underling condition before cardiac structure change becomes irreversible
    - diuretics not helpful!!! May be harmful

**Hypoventilation Syndrome**

- **s/s:** sluggish / sleepy during day
- sequelae: pulmonary hypertension, cor pulmonale, secondary erythrocytosis
- **dx:** PFTs, sleep studies, CXR, arterial blood gas, serum bicarb
• tx: lifestyle, healthy weight, physical activity, CPAP, tracheostomy
• sleep apnea falls into this category

**Idiopathic Pulmonary Fibrosis**

- chronic lung disorder; increasing scarring → reduces lung capacity; etiology unknown; MC of all interstitial lung diseases
- non-idiopathic: smoking, viral infections, environmental (silica, hard metal dusts), medication, genetics, XRT, GERD
- dx: CXR shows fibrosis; CT chest = diffuse patchy fibrosis with pleural based honeycombing
  - PFTs = restrictive (decreased lung volume, normal FEV1/FVC ration
- Tx: corticosteroids, O2, eventual lung transplant

**Pneumoconiosis**

- Definition: pulmonary fibrosis with a known cause
- Types:
  1. Coal worker’s: coal mining; complication = progressive massive fibrosis
     - CXR: small opacities in upper lung fields
  2. Silicosis: mining, sandblasting, stone, quarry work; increased risk TB and progression to massive fibrosis
     - CXR: small rounded opacities seen throughout lung, hilar lymph nodes may be calcified
  3. Asbestos: insulation, demolition, construction; complication = mesothelioma
     - CXR: interstitial fibrosis, thickened pleura, calcified plaques appear on diaphragms or lateral chest wall
  4. Berylliosis: high tech field, nuclear power, ceramics, foundries; requires chronic steroids
     - CXR: diffuse infiltrates and hilar adenopathy
- s/sx: SOB + nonproductive cough + chronic hypoxia, cor pulmonale
- dx: PFTs = reduced long volumes - restrictive dysfunction and reduced diffusing capacity
  1. CXR: interstitial fibrosis
- Tx: primarily supportive → oxygen, vaccinations (pneumococcal, flu) and rehab
  - Steroids to relieve chronic alveolitis; smoking cessation!!!!
  - Smoking cessation = synergistically linked to lung cancer

**Pneumonia**

1. Viral: adults → flu = MC cause; kids → RSV; comes on fast
   - Dx: CXR = bilateral interstitial infiltrates; rapid antigen testing for flu, RSV nasal swab, cold agglutinin titer negative
   - Tx: flu with Tamiflu (A and B) if sx began <48 hrs; symptomatic tx = beta 2 agonist, fluids, rest
2. Bacterial: fever, dyspnea, tachycardia, tachypnea, cough, +/– sputum
   - Dx: patchy, segmental lobar, multilobar consolidation; blood cultures x2, sputum gram stain
   - Tx: outpatient = doxy, macrolides; inpatient = ceftriaxone + azithromycin/respiratory FQs
3. Fungal: common in immunocompromised pt (AIDs, steroid use, organ transplant)
   - Coccidioides (valley fever); non-remitting cough/bronchitis non-responsive to conventional tx
     - Fungal inhalation in western states; test with EIA for IgM and IgG
     - Tx: fluconazole / itraconazole
   - Pulmonary aspiergillosis: usually those with healthy immune systems
     - Tx: fluconazole / itraconazole
   - Cryptococcus: found in soil; can disseminate and → meningitis
     - Lumbar puncture for menigitis
     - Tx: amphotericin B
   - Histoplasma capsulatum: pulmonary lesions that are apical and resemble cavitary TB; worsening cough and dyspnea, progression to disabling respiratory dysfunction; no dissemination
     - Bird or bat droppings (caves, zoo, bird); Mississippi ohio river valley
     - Signs: mediastinal or hilar LAD (looks like sarcoid)
     - Tx: amp B
4. HIV:
   - PJP (pneumocystis jirovci)
     - Common in HIV patients with CD4 count <200
     - XR: diffuse interstitial or bilateral perihilar infiltrates
     - Dx: bronchoalveolar lavage PCR, labs, HIV test; low O2 sat despite supplemental oxygen
     - Tx: Bactrim and steroids; pentamidine for allergy
     - Prophylaxis for high risk pt with CD4 <200 = daily bacctrim
   - CURB65: confusion, urea >7, RR >30, SBP <90 OR DBP <60, age >65
0-1 = low risk, consider home tx
2 = probable admission vs close outpt management
3-5 admission, manage as severe

**Pulmonary Hypertension**
- Blood pressure in lungs usually 15/5. In pulmonary HTN → >25mmHg
- Usually caused by underlying disorder (constrictive pericarditis, mitral stenosis = MC, LV failure, mediastinal disease compression pulmonary veins)
  - Mitral stenosis: mitral valve = tight so blood can’t pass into left ventricle → pressure backs up to lungs
    - When right heart can’t pump against vascular resistance → R heart failure = cor pulmonale
- Dx: CXR, CT, PFTs, ECHO, right heart cath = gold standard
- Tx: identify and treat underlying cause

**Pulmonary Neoplasm**

1. **Carcinoid tumors**: GI tract cancer metastasized to lung (CA of appendix = MC; appendix → liver → lung)
   a. Presentation: hemoptysis, cough, focal wheezing, recurrent pneumonia
      i. Carcinoid syndrome = cutaneous flushing, diarrhea, wheezing, hypotension (telltale sign)
   b. **Adenoma = MC type (slow growing, rare)**
   c. Dx: bronchoscopy – pink/purple central lesion, well vascularized; elevated 5-HIAA
   d. Tx: surgery
2. **Lung cancer**
   a. Small cell = 15% cases – 99% smokers; doesn’t respond to surgery; mets at presentation
      i. Central location, very aggressive
      ii. Tx = combination chemo
      iii. Paraneoplastic syndromes; cushing’s, SIADH
      iv. Tx: can’t have surgery; needs chemo
        1. Associated manifestations: SVC syndrome, Pancoast tumor, horner’s syndrome, carcinoid syndrome
   b. Non small cell (85%)
      i. Squamous cell (central mass): presents with hemoptysis, central location, hypercalcemia, elevated PTHrp
      ii. Large cell: rarely responds to surgery; periphery location, gynecomastia
      iii. Adenocarcinoma: MC peripheral mass; smoking / asbestos exposure; thrombophlebitis
      iv. Tx: stage 1-2 = surgery; 3 = chemo then surgery; 4 = palliative
3. **Pulmonary nodules**: <3cm = nodule; >3cm = mass
   a. Found on CXR → get CT
      i. If suspicious → biopsy (ill-defined lobular or spiculated suggests cancer)
      ii. Not suspicious → <1cm monitor at 3mo, 6mo, then yearly for 2 yr (calcification, smooth well defined edges = benign)

**Sarcoidosis**
- Systemic granulomatous disease → noncaseating granulomas that affects multiple organ systems; MC n. European and African American
- Lungs = most common; skin = 2nd most; skin and lymph = most common areas
- Sx = variable, etiology unknown – fever, weight loss, arthralgias, erythema nodosum = initial presenting sx
- Lupus pernio (chronic, violaceous, raise plaques and nodules commonly found on cheeks, nose, eyes) = pathognomonic for sarcoid and most specific physical exam finding
- Dx: hypercalcemia; ACE levels 4x normal, elevated ESR; can be made from biopsy of peripheral lesions or fiber optic bronchoscopy for central pulmonary lesions; serial PFTs to assess disease progression / guide treatment
  - **Dx = endobrachial lung biopsy!!**
- Tx: corticosteroids, methotrexate, other immunosuppressive meds; 90% respond to steroid
  - ACE-I for periodic HTN
- Prognosis depends on disease severity; spontaneous improvement common
  - Pulmonary fibrosis = leading cause of death

**Pulmonary Nodules**
- **Pulmonary nodules**: <3cm = nodule; >3cm = mass
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    - If suspicious → biopsy (ill-defined lobular or spiculated suggests cancer)
- Not suspicious → <1cm monitor at 3mo, 6mo, then yearly for 2 yr (calcification, smooth well defined edges = benign)
- Radiographic characteristics help define malignant potential of solitary pulmonary nodules
  - Growth rate determined by comparison of previous CXR or CT
    - Lesion that hasn’t grown in >2yr = benign
    - Double from 21-40 days = malignant
  - Small (<1cm) monitor at 3, 6, mo then yearly for 2 yr
    - Calcification suggests benign especially if central, concentric, popcorn
    - Margins that are speculated or irregular → CA
    - Diameter <1.5 cm strongly suggests benign; diameter >5.3cm strongly suggests CA
- Tx: management depends on radiographic findings
  - If malignant → biopsy!

GASTROINTESTINAL

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 >130bp → grave prognosis
- Hemorrhagic pancreatitis: may cause bleeding into flanks (Grey turner) or umbilical (Cullen sign)
- Chronic = fat malabsorption / steatorrhea
  - Triad of calcifications, steatorrhea, diabetes mellitus
- Left sided exudative pleural effusion

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

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

Health Maintenance:
- STOP DRINKING

Acute vs chronic:
- Acute pancreatitis: isolated episode of abdominal pain accompanied by elevations in blood enzyme levels.
  - Inflammation of the pancreas. >80% of the cases of acute pancreatitis are related to biliary stones or alcohol use.
  - Acute pancreatitis may lead to chronic pancreatitis.
- Chronic pancreatitis is a painful disease of the pancreas in which inflammation has resolved, but with resultant damage to the gland characterized by fibrosis, calcification and ductal inflammation. It is possible for patients with chronic pancreatitis to have episodes of acute pancreatitis.
  - Malabsorption of fat soluble vitamins may occur but rarely clinically symptomatic
  - Check fasting glucose bc increase≥.,,.d risk diabetes type 3c
Anal Fissure

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

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

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

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

Health Maintenance:
- High fiber diet

Anorectal Abscess / Fistula

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

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

Treatment of perirectal abscess: incision and drainage – no antibiotics

Celiac Disease

- s/sx: diarrhea, steatorrhea, flatulence, weight loss, weakness, abd distention
  - Associated with dermatitis herpetiformis (chronic, itchy skin rash on elbow, knees, butt, scalp)
- Associated conditions: T1DM, autoimmune hepatitis, autoimmune thyroid dz, down, turner, williams syndrome, increased incidence of small bowel lymphoma
- Labs: IgA ant9-endomysia (EMA) and anti-tissue transflutaminase (anti-tTG) ab
- Dx: made by small bowel biopsy (duodenum)
- Tx: gluten free diet

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)
- Hypoactive bowel sounds are an indicator that a perforation has occurred. Other symptoms include high fever, systemic signs of toxicity (tachycardia and increased respiratory rate), and increased abdominal pain with rebound tenderness.

Diagnostics:
- US = initial test of choice: thickened GB <3mm, distended GB, sludge, gallstones, pericholecystic fluid, sonographic murphy’s sign
- Abdominal XR: 10% of stones seen
- Labs: leukocytosis with left shift, increased bilirubin after 24 hours, alk phos and LFTs
- HIDA scan (heptoiminodiacetic acid): gold standard – positive HIDA = nonvisulization of gallbladder in cholecysitis
  - HIDA shows gallbladder ejection fraction and if stone are present in the cystic ducts
  - If pt. is fasting, HIDA may show falsely decreased ejection fraction (if this is the case, inject with morphine or CCK)
- ERCP can identify cause, location and extent of biliary obstruction

Therapeutics:
- Conservative: NPO, IVF, abx (3rd gen cephalosporin + metronidazole)
- Cholecystectomy
Meperidine preferred (morphine assoc with sphincter of oddi spasm)

Acute acalculous cholecystitis: MC occur in seriously ill pt.

Chronic cholecystitis: assoc with gallstones; strawberry GB (interior of GB resembles strawberry secondary to cholesterol submucosal aggregation) → porcelain GB (premalignant condition)

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

**Cholelithiasis**

**Definition:** gallstones in the gall bladder (NO INFLAMMATION)
- 90% cholesterol

**History and Physical Exam:**
- Risk factors = 5Fs: fat, fair, female, forty, fertile: OCP’s (increased estrogen), Native Americans, bile stasis, chronic hemolysis, cirrhosis, infection, rapid weight loss, IBD, TPN, fibrates, increased triglycerides
- MC asymptomatic (may be incidental finding)

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

**Therapeutics:**
- If asymptomatic may observe or use oral bile dissolution treatment
- Cholecystectomy in symptomatic pt (usually laparoscopic)

**Complications:**
- Choledocholithiasis: gallstones in biliary tree → +/- biliary colic or jaundice
  - Dx = MRCP
  - Tx = stone extraction via ERCP

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

**Ascending Cholangitis:**
- Definition: biliary tract infection secondary to obstruction by gallstone
  - Dx: ERCP
  - Charcot’s triad: fever/chills, RUQ pain, jaundice, increased liver enzymes
  - Reynold’s pentad: shock + AMS
  - Tx: abx (PCN + aminoglycoside), decompression of biliary tree via ERCP stone extraction

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

### Esophageal Neoplasms

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

### Esophageal Strictures

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

### Esophagitis

- **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 esophagram
- **Therapeutics:**
  - treat underlying cause

### Gastric Carcinoma

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

Health Maintenance:

Scientific Concepts:
- Causes: autoimmune disorders / other noninfectious factors
- H. pylori

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:
- Stool culture

Therapeutics:
- Self-limited
- Treat symptomatically
- Specific tx with Bactrim, ampicillin, cipro required for severely ill / malnourished pt. with sickle cell or pt who develop bacteremia

GERD

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

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

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

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

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

Health Maintenance:
6. Lifestyle modifications: elevate head of bed by six inches, avoid recumbency for 3 hours after eating, eating small meals, avoid fatty/spicy, citrus, chocolate, caffeinated products, peppermint; decrease fat and alcohol intake, weight loss, smoking cessation
7. Predisposing factors: obesity, pregnancy, diabetes, hiatal hernia, connective tissue disorders

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

Hemorrhoids

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

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

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

Therapeutics:
- conservative (stage I and II): high fiber diet, increased fluids, warm sitz baths, bulk laxatives
  - higher stage = suppositories and anesthetic and astringent properties
  - topical rectal hydrocortisone for pruritus and discomfort +/- analgesics
- surgical (stage IV): if failed conservative management, debilitating pain, strangulation
Hepatic Carcinoma

Hepatocellular carcinoma (HCC) is an aggressive tumor that often occurs in the setting of chronic liver disease and cirrhosis.

- Liver cancer in adult men is the fifth most frequently diagnosed cancer worldwide and is the second leading cause of cancer-related death in the world.
- It is typically diagnosed late in its course, and the median survival following diagnosis is approximately 6 to 20 months.
- Hepatitis B viral (HBV) infection, chronic hepatitis C virus (HCV) infection, hereditary hemochromatosis, and cirrhosis of almost any cause will increase the risk of hepatocellular carcinoma.
- Benign lesions include cavernous hemangioma, hepatocellular adenoma, and infantile hemangioendothelioma.

The diagnostic approach to a solid liver is determined by the size of the lesion.

- Image lesions less than 1 cm with dynamic contrast-enhanced magnetic resonance imaging (MRI) to look for imaging features of HCC. If negative, obtain follow-up ultrasounds every three months.
- ↑ α-fetoprotein - A rise in serum AFP in a patient with cirrhosis or hepatitis B should raise concern that HCC has developed.
- If there are radiologic hallmarks of HCC a diagnosis of HCC is made. If the radiologic hallmarks of HCC are not seen, a biopsy should be obtained and assessed by an expert pathologist.

HCC Screening

- Screening patients with cirrhosis is reasonable, although this measure is controversial and has not been shown to reduce mortality.
- Screening with periodic AFP measurement and ultrasonography is sometimes recommended for high-risk patients.
- One common screening method is ultrasonography every 6 or 12 mo. Many experts advise screening patients with long-standing hepatitis B even when cirrhosis is absent.

Although the mainstay of therapy is surgical resection, the majority of patients are not eligible because of tumor extent or underlying liver dysfunction.

- Transplantation if tumors are small and few.
- For single tumors < 5 cm or ≤ 3 tumors that are all ≤ 3 cm and that are limited to the liver, liver transplantation results in as good a prognosis as liver transplantation done for noncancerous disorders.
- Surgical resection may be done; however, the cancer usually recurs.

Hiatal Hernia aka paraoesophageal hiatus 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 reflux, 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

Diagnostics: physical exam / US

Therapeutics:

- Acid suppression may suffice (type 1)
- Surgical repair can be used for more serious cases (type 2) – fundoplication

Inflammatory Bowel Disease

<table>
<thead>
<tr>
<th>UC</th>
<th>Crohn’s</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
<td>Onset sudden or gradual</td>
</tr>
<tr>
<td><strong>History / Physical</strong></td>
<td>Abdominal cramps, bloody diarrhea, anemia, fatigue</td>
</tr>
<tr>
<td><strong>Area affected</strong></td>
<td>Limited to colon (begins in rectum with contiguous spread proximally to colon)</td>
</tr>
<tr>
<td>Depth</td>
<td>Rectum always involved</td>
</tr>
<tr>
<td>-------</td>
<td>------------------------</td>
</tr>
<tr>
<td>MC in terminal ileum / right colon (RLQ pain)</td>
<td>abdominal pain: RLQ pain (crampy); weight loss more common in Crohn’s disease; diarrhea with no visible blood usually</td>
</tr>
<tr>
<td>Mucosa and sub mucosa only</td>
<td>transmural</td>
</tr>
<tr>
<td>abdominal pain; LLQ MC, colicky</td>
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</tbody>
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**Clinical manifestations**
- abdominal pain; LLQ MC, colicky
- tenesmus, urgency
- blood diarrhea hallmark (stools with mucus/pus), hematochezia MC in UC
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**Complications**
- primary sclerosing cholangitis; colon CA; toxic megacolon (more common in UC)
- perianal dz; fistulas, stricture, abscesses, granulomas
- malabsorption: B12 and iron deficiency
- smoking makes worse
- smoking decreases risk for UC

**Colonoscopy**
- uniform inflammation +/- ulceration in rectum / colon — “sandpaper” appearance
- pseudo polyps
- skip lesions (normal areas between inflamed areas) with cobblestone appearance

**Barium studies**
- stovepipe sign (loss of haustral marking)
- string sign (Ba flow through narrowed inflamed / scarred area due to transmural strictures)

**Labs**
- P-ANCA (more common in UC)
- ASCA

**Surgery**
- curative
curative
- noncurative
- noncurative

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

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

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

**Aminosalicylates (sulfasalazine, mesalamine) → corticosteroids → immune modifying agents**

1. 5-aminosalicylates: anti-inflammatory agents; good for flares and remission
   - 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 (hepatitis, pancreatitis, allergic reaction, fever, rash); give folic acid with sulfasalazine

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

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

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

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

Scientific Concepts:
  o 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
  o MC cause of chronic / recurrent abdominal pain in US

History and Physical:
  o Abdominal pain with altered defecation / bowel habits
  o Abdominal pain anywhere or localized to hypogastrium / LLQ
    o May be worsened by food intake, relieved with defecation
    o 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 GI cancer, IBD, celiac sprue
    o Persistent diarrhea 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 mucilloid = mainstays of treatment)
  o Diarrhea sx: anticholinergics / spasm, antidiarrhea
  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

Pancreatitis

• Think AB DISCOMFORT: alcohol, biliary, don’t know, iatrogenic, scorpion, Ca (hypercalcemia), obstruction, mom, familial, triglyceride/trauma

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

History and Physical:
Epigastric pain radiating to back – **boring, constant radiating to back / other quadrant**; lessens when pt. leans forward, sits, or lies in fetal position

- N/V, fever, leukocytosis, dehydration / shock, sterile peritonitis, severe hypovolemia, ARDS, tachycardia >130bp → grave prognosis
- Hemorrhagic pancreatitis: may cause bleeding into flanks (Grey turner) or umbilical (Cullen sign)
- Chronic = fat malabsorption / steatorrhea
  - Triad of calcifications, steatorrhea, diabetes mellitus
- Left sided exudative pleural effusion

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

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

Health Maintenance:
- STOP DRINKING

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**Peptic Ulcer Disease**

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

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

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

Therapeutics:
- H.pylori: clarithromycin + amoxicillin + PPI (CAP)
- H. pylori negative: PPI, H2RA, misoprostol, antacids, bismuth compounds, sucralfate
  - PPI: drug of choice / most effective drug to treat PUD – 30m before meals; s/e = B12 deficiency
  - H2 blocker: cimetidine = lots of drug interactions + anti-androgen effects
  - Misoprostol: good for preventing NSAID related ulcers but not for healing already existing ulcers; CI in premenopausal women bc 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

- Weight loss is experienced by up to 30% of patients post-surgically. Patients may limit food intake due to early satiety. Distention or discomfort may occur shortly after even a moderate-sized meal as the residual gastric pouch is smaller. Patients should be counseled to eat smaller and more frequent meals. Additionally, weight loss can be a result of other postsurgical complications such as malabsorption or dumping syndrome.

ORTHOPEDICS / RHEUMATOLOGY

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

Gout / Pseudogout
- 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
- s/s: MC = podagra (attack of MTP of great toe) (70% of cases); pain, swelling, redness, exquisite tenderness. In chronic gout = tophi
- dx: joint fluid – rod shaped negatively birefringent; serum uric acid level >8 (not diagnostic)
- imaging: small, punched out lesions on XR = high likelihood diagnosis
- tx:
  - lifestyle: elevation, rest, decrease purines (meats, bear, seafood, alcohol), weight loss, increase protein, limit alcohol
  - 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
- CPPD: pseudogout; usually >60yo; large joints, lower extremity; no tophi
- Similar gout symptoms
- Dx: rhomboid shaped calcium pyrophosphad crystals – positively birefringent
- XR shows fine, linear calcifications in cartilage
- Tx: NSAIDs, colchicines, intra-articular steroid injections
  - Colchicine = prophylaxis, NSAIDs = acute attacks

Polyarteritis Nodosa
- Systemic vasculitis of medium and small arteries; men aged 40-50
  - Most = idiopathic; 20% have hepatitis B / C
- s/s: Damage to affected artery → hypertension, aneurysm, thrombosis, necrosis
  - Renal: HTN 2/2 increased renin production (may progress to renal failure)
  - Constitutional: fevers, myalgias, arthritis
  - CNS: neuropathy, amaurosis fugax, peripheral neuropathy
  - Dermatologic: livedo reticularis, purpura, ulcers, gangrene
- Dx: confirmed by biopsy showing necrotizing arteritis or by arteriography showing the typical aneurysms in medium sized arteries
  - Increased ESR, ANCA negative, renal / mesenteric angiography (microaneurysms with abrupt cut-off of small artery)
- Tx: steroids +/- cyclophosphamide (plasmapheresis in pt with Hep B)

Polymyalgia Rheumatica
- Idiopathic inflammatory condition causing painful synovitis, bursitis, tenosynovitis (aching stiffness of proximal joints (shoulder, hip, neck); age >50; closely related to giant cell arteritis; JOINT PAIN
- s/s: bilateral proximal joint pain and stiffness of pelvic and shoulder girdle (difficult combing hair / dressing); worse in AM
- p/e: normal muscle strength, reduced active and passive range of movement, joint swelling
- dx: ESR >50; temporal arteritis confirmed by biopsy
- tx: respond quickly to low dose steroid treatment (may need for 2 years, then taper); methotrexate also sometimes used
Polymyositis

- chronic, idiopathic inflammatory dz of muscle causing symmetrical, proximal muscle weakness and MUSCLE PAIN
  - i.e. quadriceps femoris
- dx: CONFIRMED W BIOPSY; p/e of muscle strength, blood tests for muscle enzymes, electrical tests of muscle and nerves
  - increased aldolase, CK, ESR, + muscle biopsy, abnormal EMG
  - + ANTI-JO ab, +anti-SRP ab, +anti-Mi-2 ab
- Tx: steroids and sometimes immunosuppressants (methotrexate / azathioprine) until sx resolve

Reactive Arthritis

- Triad: conjunctivitis, urethritis, oligoarthritis (can’t see, can’t pee, can’t climb a tree)
- MC seen in chlamydia +/- gonorrhea and GI infections (salmonella, shigella, campylobacter, yersinia)
- Dx: + HLA-B27 (80%), increased WBC, ESR, IgG; synovial fluid = aseptic with negative bacterial cultures
- Tx: NSAIDs = mainstay; abx for infections (methotrexate / steroids with no response to NSAIDs)

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; bilateral ulnar deviation at MCP, boutonniere deformity, swan-neck deformity
  - extra-articular: changes in skin, lungs, kidneys, eyes, lier, 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 deminieralization seen on radiograph
  - Positive RF, anti-cyclic citrullinated peptide ab
- 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)
        - Check CBC and CMP within 6 weeks of changing dose of methotrexate → cause liver toxicity and reduction in WBC, RBC, platelets; also a folate inhibitor – increase folic acid may be needed with increased doses
      - Hydroxychloroquine for pregnant pt
  - Reconstructive surgery for severe cases

Sjogren Syndrome

- Common chronic autoimmune, systemic, inflammatory disorder that attacks exocrine glands
- s/s: dry mouth, eyes, and other mucous membranes d/t lymphocytic infiltration of exocrine gland and secondary gland dysf(n)
  - xerostomia, dry eyes, parotid enlargement
- dx: + RF, shirmer’s tear test (<5mm lacrimation in 5 min)
- tx: artificial tears, pilocarpine for xerostomia (cholinergic drug that increases lacrimation and salivation) / cevimeline (stimulates muscarinic cholinergic receptors)

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

### Systemic Sclerosis (Scleroderma)

- Systemic connective tissue disorder causing thickened skin (sclerodactyly), lung, heart, kidney and GI tract
- **s/s:** tight, shiny, thickened skin d/t fibrous collagen buildup
  - limited cutaneous systemic sclerosis = CREST syndrome: calcinosis, raynaud’s, esophageal dysfunction (GERD), sclerodactyly, telangiectasis
  - diffuse cutaneous systemic sclerosis = skin thickening of trunk and proximal extremities
- **Dx:** + anti-centromere AB (limited crest / better prognosis), + anti-scl-70 ab (diffuse disease / mylitle organ involvement)
- **Tx:** DMARDs / steroids (CCBs / prostacyclin for raynaud’s)

### 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 (abdominal pain / acute abdomen), amenorrhea, salt cravings, orthostatic hypotension, delayed deep tendon reflexes, hyperpigmentation
- **Dx:**
  - hyperkalemia, hyponatremia, hypoglycemia, hypercalcemia, low BUN
  - **serum cortisol rise >20ug/dL** after administrated of cosyntropin
  - Antiadrenal antibodies present in 50%
• DHEA levels <1000 ng/mL
  • **Tx:** primary disease: steroids and mineralocorticoids
    • DHEA may be given for increased muscle mass
    • Normal prognosis
    • Crisis: aggressive IV saline, glucose, glucocorticoids 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:
  • Hypercortisolism → obesity (centripetal, buffalo hump, moon facies, supraclavicular pads), HTN, thirst, polyuria
  • Proximal muscle weakness, pigmented striae; backache, headache, oligomenorrhea / amenorrhea / ED; emotional lability / psychosis

**Dx:** overnight dexamethasone suppression test
  • Suppression <5ug/dL excludes cushing with some certainty; plasma or serum ACTH <20pg/mL suggests adrenal tumor
  • 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%
  • 95% 5 year survival

**Diabetes Mellitus**

• Type 1: MC in young people
  • Little to no endogenous insulin secretion; elevated plasma glucagon pancreatic B cells don’t respond to stimuli
  • Most are autoimmune (90%) ith antibodies to insulin, islet cells, and glutamic acid decarboxulase
  • 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
  • **Dx:** random plasma glucose >200mg/dL with classic symptoms or fasting >126 or > on more than one occasion
    ▪ HbA1c >6.5% or higher
  • **Tx:**
    ▪ diet (Mediterranean)
    ▪ Insulin: regular insulin absorbed most rapidly in abdomen
    ▪ Daily aspirin to decrease DV risk; careful foot care, regular ophthalmology exams, moderate exercise, meticuluous personal hygiene, prompt treatment of infection

• 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

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Hypercalcemia

**Definition:**
- serum total calcium > 10.5 mg/dL
- ionized fraction of calcium > 5.6 mg/dL

**Presentation:** “Stones, bones, abdominal groans, psychiatric moans”, EKG: shortened QT interval.
- Blood: ↑ PTH, ↑ Calcium, ↓ phosphorus
- Associated with malignancy and hyperparathyroidism

**Treatment:** IV normal saline and furosemide; denosumab

Hypercalcemia

**Definition:**
- serum total calcium < 8.4 mg/dL
- ionized fraction of calcium < 4.4 mg/dL

**Presentation:** QT prolongation, Trousseau’s sign, Chvostek’s sign
- Labs: ↓ Ca+ ↓ PTH ↑ phosphate
- EKG = Prolonged QT

**Treatment:** IV calcium gluconate or calcium chloride

Hypocalcemia

**Definition:** serum sodium of < 135 mmol/L

**Peripheral and presacral edema, pulmonary edema, JVD, hypertension, decreased hematocrit, decreased serum protein, decreased BUN/CR**

**Presentation:** Muscle cramps and seizures
- Hypovolemic hyponatremia – CHF, nephrotic syndrome, renal failure, cirrhosis
- Euvolemic hyponatremia – SIADH (steroids, hypothyroid)
- Hypovolemic hyponatremia – sodium loss (renal, non-renal)

**Treatment:**
- Asymptomatic → free water restriction
- moderate hyponatremia → IV normal saline, loop diuretics may be added
- severe hyponatremia → hypertonic (3%) saline

Serum Na should be corrected slowly—by ≤ 8 mEq/L in a 24 hour period.

Hyponatremia

**Definition:** serum sodium of > 145 mmol/L

**Etiology:** Diarrhea, burns, diuretics, hyperglycemia, diabetes insipidus, a deficit of thirst

**Poor skin turgor, dry mucous membranes, flat neck veins, hypotension, increased BUN/CR ratio > 20:1**
- Decrease circulating volume = decrease of flow to kidneys means more bound urea in the blood which means ↑ BUN

**Treatment:** intravenous (IV) 5% dextrose in water (D5W). Rapid overcorrection causes cerebral edema and pontine herniation.*

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

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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
**Hypothyroid**

- Dx: Elevated T3 and free T4, low TSH
  - RAIU study shows increased uptake in graves disease and toxic multinodular goiter
- Tx: beta blocker (propranolol) to control sx; propylthiouracil and methimazole, PTU for breastfeeding, iodine ablation, thyroidectomy

- 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
  - Low free T4 and elevated TSH = primary hypothyroid
  - Low free T4 and low/normal TSH = secondary hypothyroid
  - Normal free T4 and elevated TSH = subclinical hypothyroid
- Tx: levothyroxine; check levels of thyroid frequently

**Hyperparathyroid**

- 1+ parathyroid glands secretes excessive PTH \(\rightarrow\) increased \(\text{Ca}\) >12 (<12 = asx)
- low blood \(\text{Ca}\) \(\rightarrow\) increased PTH \(\rightarrow\) 1. Bone breakdown releases Ca 2. Kidney honds onto Ca and increased Vitamin D 3. Intestine absorbs more Ca \(\rightarrow\) increased Ca in blood levels
- primary: increased PTH caused by PTH secreting parathyroid adenoma
- secondary: increased PTH by response to hypocalcemia or vitamin D deficiency
  - CKD = MC cause
- s/sx: n/v, weakness, fatigue, constipation \(\rightarrow\) bones, stones, groans, psychic moans
  - bone loss from increased PTH and Ca absorption from bones = pain in boans
  - renal loss of Ca and phosphage = kidney stone
  - increase GI absorption of Ca and abdominal cramps = groans
  - irritability, psychosis, depression = moans
- dx:
  - blood = increased Ca, increased PTH, decreased phosphorus
  - urine = hyperphosphaturia, hypercalciuria (all Ca and Phosphorhus go out through kidneys to urine)
- tx: remove parathyroid adenoma (subtotal or total)
  - secondary = replace cause (vitamin D / Ca supplementation)
  - if Ca very high: IV fluids, Lasix, calcitonin; treat osteoporosis with bisphosphonates

**Hypoparathyroid**

- decreased parathyroid hormone \(\rightarrow\) decreased blood levels of Ca
- MC causes = post surgical (neck / thyroid) or autoimmune
- s/s: carpopedial spasm, laryngeal spasm, tingling, tetany, facial grimacing
  - Chvostek = cheek with tapping; trousseau = hand with blood pressure cuff
- Dx: prolonged QT, low pTH, low Ca, high phosphate
- Tx: vitamin D and Ca (tetany = secure airway, IV calcium gluconate)

**Paget disease of the bone**

- Hypervascular bone
- Bone remodeling disorder: unorganized bone that is less compact / weaker than normal bone; idiopathic
- r/f: family history
- MC affects skull, spine, pelvis, femur
- s/s: pain in affected bones; fractures after little force; warmth in affected limbs (increased vascularity); neurological complications (deafness) from compression of nerves by abnormal bone; arthritic problems (kyphosis, bowlegs); may \(\rightarrow\) osteosarcoma
- labs: increased serum alkaline phosphatase and bone-specific alk phos; increased osteoclast activity and osteoblastic activity
- dx: XR = lytic lesions / thickened bone cortices; bone biopsy to exclude malignancies
- tx: bisphosphonates (reduce bone resorption) / calcitonin; surgery to help deformities / decompress impinged nerves

**Pheochromocytoma**
catecholamine secreting adrenal tumor that secretes NE and E autonomously and intermittently → HTN, palpitations, HA, excessive sweating (90% are benign); associated with neurofibromatosis type 1, MEN 2A/B
  o 5 P’s: pressure, pain (HA), perspiration, palpitations, pallor
Dx: 24 hour catecholamines (metanephrine and vaillylmandelic acid); MRI or CT to visualize adrenal tumor
Tx: complete adrenalectomy, surgical resection of tumor, phenoxybenzamine (preop), phentaloamine (acute HTN crisis), sodium nitroprussie (acute HTN crisis), nicardipine (acute HTN crisis)
  o Pre-op nonselective alpha blockade: phenoxybenzamine or phentolamine 7-14 days followed by beta blocker to control HTN (NO solo beta blocker → prevent unopposed alpha constriction → life threatening HTN)

**Pituitary Adenoma**

s/s: diminished temporal vision, or bitemporal hemianopsia = MC visual
lactotroph adenomas (prolactinomas = MC) are pituitary masses → hypersecretion of prolactin
  o s/s: amenorrhea, galactorrhea, and headaches.
  o location of the mass at the sella turcica → tumor applies pressure on the optic chiasm of the optic nerve resulting in loss of vision in the temporal visual fields.
Growth hormone tumor: gigantism, acromegaly
Dx: MRI, CT scan, and evaluation of hormone levels (ACTH, TSH, GH, prolactin)
Tx: dopamine agonists cabergoline and bromocriptine; if dopamine agonists are unsuccessful, transsphenoidal resection of the pituitary tumor should be considered.
  o in women, estrogen therapy can also be used due to the associated hypogonadism.

**Thyroid Cancer**
MC risk is radiation exposure; papillary carcinoma = MC (papillary = popular); MC F 40-60y/o
Dx: ultrasound – lesions >1cm should be biopsied; smaller lesions can be followed / reevaluated if they grow
  o High risk malignancy on U/S: microcalcifications, hypoechoogenicity, solid nodule, irregular nodule margins, chaotic intramodular vasculature, nodule that is more tall than wide
  o To eval malignancy: thyroid uptake scan – cancerous does not take up iodine (cold); non-cancerous will take up iodine (hot)
  o If cold → fine needle aspiration
  o U/S → thyroid uptake scan → fine needle aspiration
Tx: depends on staging (99% 5-year survival with local confined,<1cm papillary carcinoma)
  o always involve complete / partial thyroidectomy with chemo and radiation for anaplastic thyroid CA
  o recommended TSH level for pt: 0.1-2.0
  papillary = MC; anaplastic = least common but most aggressive; medullary = associated with MEN type 2, calcitonin.can be used as tumor marker

**NEUROLOGY**

**Bell Palsy**
s/s: sudden onset unilateral facial nerve paralysis with no other focal neurologic or systemic findings.
  o Sx peak in 48 hours
  o 60% have viral prodrome
  o 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:
  o PE will show CN VII nerve palsy that does not spare the forehead
  o Most commonly caused by HSV
  o Treatment is prednisone, artificial tears, tape eyelid shut
  o Comments: Bilateral: Lyme disease, infectious mononucleosis

**Cerebral Aneurysm**
Weak bulging spot on wall of brain artery like thin balloon / weak spot of inner tube; usually there’s genetic predisposition
s/sx: usually asymptomatic unless ruptured; when ruptured → sudden, severe headache
classifications: by size and shape
  o small: diameter <15 mm
  o large: 15-25mm
- giant: 25-50 mm
- super giant: >50mm

- types:
  - saccular (“berry”) aneurysm – MC type; account for 80-90% and MC cause of SAH; occur at arterial bifurcations and branches of lg arteries at base of brain (circle of willis)
  - fusiform: dilation of entire circumference of vessel
  - traumatic: caused by closed head injury / penetrating trauma to brain
  - myotic: infected emboli
  - ruptured (AVM) causes bleeding into CSF in subarachnoid space – ruptured berry account for ~75% - mortality rate 50%
    - r/f: smoking, hypertension, hypercholesterolemia, heavy alcohol use; associated with polycystic kidney and coarctation of aorta
    - s/sx: sudden onset unusually severe worst headache of life, n/v, seizure, altered state consciousness; increased bp, fever 102F
    - herald bleed: less severe headache

- Dx: found incidentally or when pt presents with subarachnoid hemorrhage; non-con head CT for investigational
  - LP has elevated opening pressure, bloody fluid (xanthochromia, RBC)
  - Cerebral angiography = gold standard

- Tx: surgical clipping, endovascular coiling within first 24 hours; restore respiration

**Cerebral Vascular Accident**

- MC caused by ischemia of brain, from thrombus or embolus
  - More common in pt with hypertension, history of smoking ,heart dz
  - Presenting sx depend on are of brain affected – facial droop, slurred speech, unilateral weaken
  - Dx: non-con head CT emergently; rule out hypoglycemia
  - Not hemorrhagic should get tissue plasminogen activator (r-tPA)
  - Anterior cerebral artery: paralysis of contralateral foot/leg (lower>upper); sensory loss over toes, foot, leg; impairment of gait and stance, flat affect, lack of spontaneity, slowness, distractibility, slow to perform acts, cognitive impairment, urinary incontinence
  - Middle cerebral artery: contralateral paralysis (upper >lower), hemianopsia, aphasia
  - Posterior: LOC, n/v, CN dysfunction, ataxia, visual agnosia

**Cluster Headaches**

- 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 Headache**

- 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 Headache:**

- 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

Coma

- All pt have >3; eye response, verbal response, motor response (motor correlates best to outcome)

Complex Regional Pain Syndrome
- Non-dermatomal limb pain following trauma / surgery; pain disproportionate to injury
  - Autonomic and vasomotor dysfunction in extremity; does not follow peripheral nerve distribution; no systemic sx
- Dx: Budapest consensus criteria for clinical diagnosis of continuing pain disproportionate to inciting event
  - At least 1 sx in 3 of 4 categories:
    - Sensory: hyperalgesia and/or allodynia → evidence of hyperalgesia to pinprick / allodynia to light tough / temperature
    - Vasomotor: skin, temperature, color asymmetry → temperature asymmetric >1C and/or skin color changes
    - Sudomotor/edema: edema, sweating changes, sweating asymmetry → edema and/or sweating changes / asymmetry
    - Motor/trophic: decreased rom or motor dysfunction and/or trophic changes (hair, nail, skin) → weakness, tremor, dystonia
- Tx: early intervention is best
  - NSAIDs, steroids, PT/OT, pain management
  - Antidepressants / anticonvulsants (gabapentin)
  - Regional nerve block / spinal cord stimulators

Concussion
- Brief LOC, amnesia
- No focal neurologic deficits
- Negative CT scan
Delirium

- Acute syndrome caused by medical condition, substance, intoxication or withdrawal or medication side effect → AMS
  - Ex: sepsis, sundowning, ETOH withdrawal, opiate withdrawal, sunstroke
- Rapid onset, short-term, reversible!!!
- Criteria:
  - Disturbed level of consciousness (decreased attention span / lack of environmental awareness)
  - Cognitive change – memory deficit, disorientation, language disturbance, visual / auditory hallucinations
  - Rapid onset within hours / days with fluctuating course
  - Evidence of causal physical condition
- Dx: history, CT/MRI, CBC, blood cultures, CXR, UA, BUN, electrolytes, glucose, utox to r/o infection
- Tx: treat underlying cause / supportive care; sedation when necessary

Alzheimer’s Disease

- Age related progressive cognitive decline, affects 5% of those aged 71-80 years, and near 40% of those aged over 90 years.
- s/s: gradual, progressive memory loss, difficulty word-finding, concentration problems, emotional lability, personality changes, social withdrawal, 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
- dx: 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 = MC: 2/3 dementia 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 fluctuations, visual hallucinations, Parkinsonism
  - frontotemporal dementia: personality, and social behavior changes, non-fluent speech
  - neurodegenerative conditions: Huntington disease, metabolic abnormalities
- irreversible causes: alzheimer’s, vascular dementia, Creutzfeldt-jakob
- reversible: depression, B12 deficiency, syphilis, hypothyroidism, NPH, drug use, intracranial mass
- tx: cholinesterase inhibitors (donepezil); NMDA antagonists (memantine) → don’t cure, just slow progression

Encephalitis

- similar to meningitis; AMS, seizures, personality change
- differentiated from meningitis by altered brain functioning
- s/s: flu like illness → mild headache, fever, sore throat, decreased appetite, myalgias (NO kernig / Brudzinski)
- usually viral, HSV = MC, CMV in immunocompromised
- dx: MRI and lumbar puncture (PCR for viruses)
- tx: supportive care and acyclovir (10mg/kg IV q8hr PROMPTLY); empiric abx until meningitis excluded
- reye’s syndrome: rapidly progressive encephalopathy with hepatic dysfunction – lethargy drowsiness, vomiting
  - Babinski reflex positive and hyperreflexia noted; from salicylate use (aspirin, pepto bismol)
  - Vomiting, confusion, rapidly evolving to seizure / coma
  - dx: elevated aminotransferase, PTT, hyperammonemia, hypoglycemia, metabolic acidosis
  - Tx: supportive

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.

Giant Cell Arteritis (temporal arteritis)
- Idiopathic +/- autoimmune viral infection → monocyte activation and inflammatory cytokines production, inflammation and tissue destruction
- Vasculitis affects extracranial branches of carotid artery: temporal, occipital, opthalmic, posterior ciliary
- **MC women >50yo associated with polymyalgia rheumatica**
- **s/s:** headache, jaw claudication with chewing, acute vision disturbance (amaurosis fugax – temporary monocular blindness), thickened temporal artery, aortic aneurysm
- **dx:** ESR >100, temporal artery biopsy
- **complication:** blindness
- **tx:** high dose prednisone to avoid blindness (40-60mg/day for 4 weeks then taper slowly and maintain 1-2 years)

Guillain Barre Syndrome
- **ascending paralysis beginning in distal limbs:** leg weakness → total paralysis of all 4 limbs, facial muscles, eyes, loss of reflexes
- **often present after immunization**
- **post-infectious cause:** campylobacter jejuni = MC, CMV, Epstein-barr, HIV
- **dx:** based on LP → elevated CSF protein with normal CSF WBC
- **tx:** plasma exchange (remove circulating antibodies) and IVIG
  - monitor PFTs for paralysis of chest muscle/diaphragm (respiratory failure)

Huntington Disease
- **autosomal dominant, incurable neurodegenerative dz** → progressive motor / psychiatric dysfunction, dementia, chorea; usually occurs between 30-50yo; life expectancy 15-25 years after symptom onset; high risk suicide
- **50% chance of receiving the disease from a parent**
- **s/sx:** chorea, rigidity, hypotonia, saccadic eye movements, psych symptoms, cognitive decline
- **dx:** clinical symptoms, MRI, family history, genetic testing
  - genetic testing = 40+ CAG repeats
  - MRI = cerebral atrophy and atrophy of caudate nucleus
  - Chorea = nonrepeating complex involuntary rhythmic movements
  - Dementia, mutism, dysphagia
- **Tx:** currently incurable – treat chorea with risperidone / haloperidol
  - Tetrabenazine depletes dopamine → first drug to specifically treat chorea; neuroleptics also help
- **Fatal in 15-20 years**

Intracranial Neoplasm
- **1/3 are glial cell origin; 1/3 meningioma, other:** vestibular schwannoma, pituitary adenoma, neurofibroma, CNS lymphoma
- **Most glial = malignant**
  - Astrocytoma: grade 4 = glioblastoma → MC with poor prognosis
  - Ependymoma: occurs in ependymal cells that line the ventricles / spinal canal
  - Medulloblastoma: MC primary malignant in children (long term survival with tx = 70%)
- **MC source of intracranial mets:** lung, breast, kidney, GI tract
- **Tx:** complete superficial removal of tumor with radiation / chemotherapy
  - Steroids to help reduce cerebral edema, anticonvulsants

Meningitis
- **Classic triad:** fever >38C, nuchal rigidity, headache
- **P/e:** kernig’s sign (neck pain with knee extension), brudzinski’s sign (leg raise with bent neck)
- **Aseptic:** usually viral; negative blood cultures
- **Bacterial:** community acquired, usually s. pneumo / n. meningitidis (both gram positive diplococci) – likely if pt has a rash
  - Neonates = e. coli / s. agalactiae
  - >50-60 = listeria / cryptococcus neoformans
  - Hospital acquired: staph / aerobic gram negative
- **Dx:** lumbar puncture → must first check for increased intracranial pressure (check for papilledema!!) get a CT if unsure
  - **Bacterial:** increased protein, decreased glucose (bacteria eat glucose); markedly increased opening pressure
  - **Viral:** normal pressure, increased WBC (lymphocytes)
Tx:
  o Aseptic: symptomatic or IV acyclovir for HSV
  o Bacterial: dexamethasone + empiric IV abx (cephalosporin, vanco, penicillins)
  o Household contacts: treat with rifampin, cipro, Levaquin, azithromycin, ceftriaxone

Multiple Sclerosis
  • Autoinflammatory disease → demyelination, neuronal loss, scarring of white matter in the brain / spinal cord
  o Destruction of myelinated axons in central nervous system
  • Usually present with visual disturbances often over many years
  • MC problems: sensory loss, optic neuritis, weakness, paresthesias
  • Lhermitte’s sign: electrical shock sensation in limbs / torso brought on by flexion of neck
  • Relapsing-remitting = MC type (85%) – episodic flare ups over days / weeks between periods of neurologic stability; during attacks: new sx present and existing ones worsen; complete recovery / residual deficits may ensue following each out
  • Dx: MRI looking for plaques/ criteria:
    o 2 episodes / attacks of sx
    o 2 different areas of the CNS involved
  • Tx: steroids (for acute attacks), interferon betas (avonex)
    o PT, symptomatic treatment for fatigue, urge incontinence, muscle spasm

Myasthenia Gravis
  • Ptosis, diplopia, blurred vision, progressive proximal muscle weakness worse at end of day, dysphagia, dysarthria
  • Antibodies to acetylcholine receptors at neuromuscular junction
  • Dx: ice pack test / tensilon test; serologic can be performed for acetylcholine receptor ab, EMG
  • Tx: pyridostigmine (acetylcholinesterase inhibitor) = first line; plasmapheresis and IVIG can be used in crisis
    o Myasthenic crisis: neuromuscular respiratory failure from dysphagia / aspiration (treat with plasma exchange, IVIG)
  • Associated with thymoma

Peripheral Neuropathies
  • Symmetric distal sensory loss, burning pain, weakness
  • Slow onset: stocking glove → DM, uremia
  • Fast onset: drugs
  • Ascending: Guillain barre
  • Hereditary: motor and sensory loss, loss of reflexes, hammer toes → stork leg deformity → charcot-marie-tooth
  • Dx: clinical; aided by electromyography / nerve conduction studies
  • Tx: gabapentin, amitriptyline, topiramate, tramadol, NSAIDs
    o May use low dose narcotics sparingly for breakthrough pain

Status Epilepticus
  • s/s: > or equal to 5 min continuous seizure activity or more than one 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 regimen of someone with seizure disorder
  • Tx:
    o Place in left lateral decubitus position (suppressed gag reflex → prone to aspiration of gastric contents)
      • IV route is preferred
    o Watchful waiting for auto-correction of acidosis once seizure activity is controlled

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

Syncope
• MC causes: vasovagal, idiopathic
• Red flags: syncope during exertion, multiple recurrences in short time, heart murmur / structural heart disease, old age, significant injury during syncope, family hx of unexpected death / exertional / unexplained recurrent syncope
• Usually from insufficient cerebral blood flow / from benign causes
• Less common = cardiac arrhythmia
• Dx: ECG, glucose, pulse ox, echo, tilt table, CNS imaging = rare
• Tx: fix underlying cause

**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
• 10% of TIA patients will have a stroke within 90 days
• Tx: Aspirin + dipyridamole 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

**UROLOGY / RENAL**

**Acid base disturbances**

Average values "24/7 40/40"
- 24 (HCO3, base) / 7.40 (pH) / 40 (CO2, acid)

**Three Step Approach to Acid Base Disorders**
- Look at your PH (7.35-7.45 is normal)
  - < 7.35 = acidosis
  - > 7.45 = alkalosis
- Next look at your PCO2 is it normal low or high (35-45 normal)
  - ↑ CO2 and ↓ PH = respiratory acidosis
  - ↓ CO2 and ↑ PH = respiratory alkalosis
  - If you don’t see a change in the CO2 in relation to the PH then take a look at the HCO3
- Finally, look at the HCO3 is it normal low or high (20-26 normal)
  - ↓ HCO3 and ↓ PH = metabolic acidosis
  - ↑ HCO3 and ↑ PH = metabolic alkalosis

<table>
<thead>
<tr>
<th>Table comparing types of acid base disorders:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type</strong></td>
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<tr>
<td>-------------------</td>
</tr>
<tr>
<td>Respiratory Acidosis</td>
</tr>
<tr>
<td>Respiratory Alkalosis</td>
</tr>
</tbody>
</table>
| Metabolic Acidosis | PH 7.30, Normal PCO2 40, Low Bicarb 16 | Need to calculate anion gap: Anion Gap = Na – (Cl + HCO3-) = 10-16

**Increased ion gap (>16):** Addition of hydrogen ions (lactic acidosis (think metformin), diabetic ketoacidosis, aspirin overdose)
- **MUDPILES:**
  - Methanol
  - Uremia
  - Diabetic Ketoacidosis
  - Paraldehyde
  - Infection
  - Lactic Acidosis
  - Ethylene Glycol
  - Salicylates

**Low anion gap (<16):** Loss of bicarbonate (think diarrhea, pancreatic or biliary drainage, renal tubular acidosis)

| Metabolic Alkalosis | PH 7.52, Normal PCO2 40, High Bicarb | Loss of hydrogen (**vomiting**), bulimia, overdose of antacids, addition of bicarbonate (hyperalimentation therapy)

## Acute renal failure

- Rapid but usually reversible reduction in renal excretory function sufficient to cause azotemia
- **Causes:** ATN, interstitial nephritis, glomerulonephritis
  - Azotemia: retention of nitrogenous waste
  - Uremia: symptomatic azotemia, with n/v/lethargy
  - Acute: sudden, hours/days and is reversible
  - Chronic: progressive, irreversible
  - Oliguria: urine output <400ml/day
- **Dx:** CBC, BUN, Cr, electrolytes (Ca, phosphate), UA, postvoid residual bladder volume
- **Tx:** depends on cause
- **3 mechanisms:**
  - Prerenal: perfusion (50%) – kidney working find but things that perfuse it aren’t
    - Ex: volume loss, heart failure, loss of peripheral vascular resistance (sepsis / anesthesia)
    - Pt. weak, decreased urine output, dizziness, sunken eyes, tachy, orthostatic
    - Fractional excretion of sodium is normal
    - Urine specific gravity >1.030, Bun/Cr >20, urine osm >500
    - Tx: fluids, cardiac support, treat shock
  - Renal: glomerular, tubular, interstitial
    - RC case = glomerulonephritis
    - WBC cast = pyelonephritis
    - Muddy casts = ATN
    - Hyaline casts = normal
    - Waxy = chronic renal disease
    - Urine specific gravity <1.010, BUN/Cr <10, urine osm <300
  - Post-renal: obstructive – most likely prostate
    - Usually low / no urine output
    - Place foley cath to find source of obstruction; renal US to look for tumor / hydronephrosis
- **ATN:** from kidney ischemia / toxins; UA shows muddy brown casts
  - Damaged tubules means can’t concentrate urine = high FENa
  - Prerenal failure is MC cause
  - Drugs: amp B, cisplatin, aminoglycosides, NSAIDs, ACE
  - Ischemic: dehydration, shock, sepsis
  - **Fraction excretion of sodium >2% + muddy, pigmented granular casts + high urine osm**
• Interstitial nephritis
  o Immune-mediated response
    ▪ Drugs: PCN, sulfa, NMSAIDs, phenytoin
    ▪ **US: WBC casts + eos + hematuria**
  o Dx: renal biopsy, discontinue offending drug, steroids, dialysis if needed, usually self-limiting
• Glomerulonephritis: IGA nephropathy, postinfectious, membranoproliferative
  o UA: oliguria, hematuria, RBC casts
  o Causes: group A strep, IGA, anti-GBM, ANCA
  o Post-strep glomerulonephritis = NC infectious cause of acute glomerulonephritis: either from strep pharyngitis or strep skin infection (impetigo) → hematuria, HTN, periorbital edema
    ▪ Dx: proteinuria + RBC in urine; usually caused by group A beta-hemolytic strep

**End Stage Renal Disease**

• Complete loss of kidney function for >3mo; kidneys functioning at 10-15% normal capacity
  o Stage 1: GFR >90 + persistent albuminuria or known structural / hereditary renal dz
  o Stage 2: GFR 60-89
  o Stage 3: GFR 30-59
  o Stage 4: GFR15-29
  o Stage 5: GFR <15
• Kidneys can’t regulate their own electrolytes → excess Na/K and decreased Mg
• Decreased PTH, vitamin D → weak bones
• Decreased erythropoietin → tells body to make RBCs → anemia
• Too much renin → increased bp
  o Decreased bp → renin release → increased angiotensin → aldosterone release → kidneys retain sodium and excrete potassium → hold on to water and increase bp
  o Dx: GFR <15, low epo (normochromic, normocytic anemia), metabolic acidosis (increased K, phosphate, decreased Na, bicarb, C), high PTH, high phosphate, low calcium, waxy casts with low urine flow
• Tx: dialysis and kidney transplant
  o Pericarditis = urgent dialysis
  o Indications for dialysis: abnormalities in acid-base balance, electrolyte disturbances, volume overload, dialyzable toxins, uremia
  o Manage comorbitidies: hemoglobin 11-2 with EPO, iron, antipletlet, diet, ACE/ARB to slow progression of renal dysfunction, loop diuretics, pneumococcal vaccine

**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, RUBBERY 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

**Bladder Cancer**

• transitional cell = MC (3x F>M)
• s/sx: painless hematuria in a smoker
• dx: cystoscopy = gold standard
• tx: endoscopic resection q3mo (recurrent / multiples lesions treated with intravesical chemo)

**Epididymitis**

• acquired by retrograde spread of organisms through vas deferens
  o <35 = chlamydia and gonocci = MC organism
  o >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

**Erectile Dysfunction**

- Inability to achieve and maintain erection
  - r/f: atherosclerosis, medications (antihypertensives), hematologic (sickle cell), hx of pelvic surgery / perianal trauma, alcohol abuse, low testosterone, hypogonadism, hypothyroid, congenital penile curvature
  - major organic causes: vascular disorders, neurologic disorder, psychological, hormonal, drugs
  - MC is atherosclerosis of cavernous arteries of penis (smoking / diabetes)

**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

**Hydrocele**

- mass of fluid-filled congenital remnants of tunica vaginalis; painless scrotal swelling
  - + transillumination
- Dx: scrotal US
- Tx: usually close within 1st year of life; may require elective repair

**Hydronephrosis**

- “water inside the kidney” – distention / dilation of renal pelvis and calyces, usually from obstruction of urine from kidney; can → atrophy
- Dx: CMP shows impaired kidney function (elevated urea / Cr) or electrolyte imbalance – hyponatremia, hyperchloremic metabolic acidosis
  - PE: palpable abdominal flank mass
Intravenous urogram, US, CT, MRI

• Tx: remove obstruction and drain urine ie nephrostomy tube, sten, urinary catheter, suprapubic catheter

• Na <135

Hypovolemia

• Inadequate fluid intake / excess water loss: no thirst, fluid loss, urinary loss, GI loss, burns, diuretics, osmotic diuresis (hyperglycemia), sodium excess, diabetes insipidus

Dx: Na >145

• DI:
  o Neurogenic (central) – caused by deficient secretion of vasopressin (ADH = anti-pe hormone) from posterior pituitary
  o Nephrogenic: kidneys unresponsive to normal vasopressin levels → inherited x-linked or from lithium / renal disease
  o Low urine sodium and polyuria
  o Urine osm <250 despite hypernatremia

• Treat underlying cause and treat accordingly (isotonic saline / LR); dialysis if Na >200

Beware with rapid fluid correction –> pulmonary

Nephrotic Syndrome

• Urinary excretion >3g protein in 24 hour urine + edema + hypoalbuminemia, fatty casts with “maltese cross” sign, hyperlipidemia, lipiduria, oval fat bodies

• MC causes: membranous nephropathy, minimal change disease, focal segmental glomerulosclerosis (obese pt, heroin, HIV+black males)

• MC secondary cause: lupus, diabetes, preeclampsia

Dx: Urinary excretion >3g protein in 24 hour urine, serologic testing and renal biopsy, serum albumin often <2.5

• Tx: treat causative disorder and angiotensin inhibition, Na restriction, diuretic +/- statin
  o Minimal change: prednisone, ACE-s
  o Membranous nephropathy: steroids, cyclophosphamide, ACE-Is
  o Focal segmental: ACE-I + prednisone

Polycystic Kidney Disease

• Growth of numerous cysts in kidneys made of epithelial cells from renal tubules; can → kidney failure / ESRD

• One of the MC human genetic disorders (autosomal dominant)

• s/sx: >30yo, +FH, abdominal mass
  o young patient, HTN + flank pain
  o 10% have rain aneurysms (worry when they complain of worst headache of life)
  o Cardiovascular abnormalities: mitral valve prolapse, LVH

• Dx: ultrasound (shows fluid-filled cysts); CT shows large renal size and multiple thin-walled cysts
  o Anemia, UA = proteinuria, hematuria, pyuria, bacteriuria

• Tx: no cure; treatment is supportive
  o ACE-I/ARB for HTN, treat infections with abx, dialysis / transplant with renal insufficiency

Prostate Cancer

• s/sx: urinary retention, decrease in urine stream/strength, back pain (metastatic disease)

• RF: old age (>80), family hx; age = most important risk factor

• Screening: all men 50yo; 40 yo with 1st degree FH or AA with discussion of risks / benefits
  o 45-49 Yrs with retest at 50 with level <0.7 + annual / biannual retesting with level >1.0
  o 50-70yo with normal DRE and PSA <3 → every 1-2 years

• dx: DRE = hard, nodular, enlarged, asymmetric prostate; dx made by needle core biopsy (transrectal US guided bx)
  o PSA >4 = further work-up with US + needle biopsy; PSA > 10 = suggests CA → bone scan to r/o mets

• Tx: radical prostatectomy – complication = erectile dysfunction
  o With mets: androgen deprivation therapy (leuprolide) – if no meds then castration
  o Monitor PSA – should be <0.1

Prostatitis

• ascending infection of gram-negative rods into prostatic ducts

• features:
• acute: sudden onset high fever, chills, low back / perineal pain
• chronic: variable – asymptomatic → acute symptomatology
• all forms present with irritative bladder sx (frequency, urgency, dysuria) and some obstruction
• prostate = swollen / tender
• avoid vigorous prostate exam in case of septicemia
• dx: UA = pyuria; possible hematuria / bacteriuria
  • prostatic fluid = leukocytosis, culture typically positive for E.coli in acute infections
    • chronic usually has enterococcus
• tx:
  • hospitalization in acute- may need parenteral fluoroquinolones
  • uncomplicated: cipro 500mg bid or levo 500mg qd 2-6 eweks 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 effective 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 parenchyma 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 interstitium 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

**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;
  • calcium > uric acid>cystine>struvite
• features: asx until inflammation / complete or partial ureteral obstruction develops
  • 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)
  • UA = microscopic / gross hematuria – leukocytes / crystals
  • CT without contrast can detect stones as small as 1mm
  • Plain film can identify radiopaque stones
  • Renal U/s: can identify stones in kidney, proximal ureter, or UVJ
• Tx: size indicates management:
  • <5mm: likely to pass on own; lots of fluid; strain urine; adequate analgesics
  • 5-10mm: not likely to pass spontaneously; increased fluid and analgesics; elective lithotripsy / ureteroscopy with stone basket extraction
    • Refer to urology with a 9 mm mid-ureteral stone
  • >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)

**Renal Cell Carcinoma**
- Triad: flank pain, hematuria, palpable abdominal/renal mass
  - Renal clear cell = MC (80); transitional cell = second MC
  - r/f: smoking
- dx: ultrasound or CT then biopsy
- tx: surgery with radical nephrectomy = curative

**Renal Vascular Disease**
- renal artery stenosis: narrowing of one or both renal arteries; MC caused atherosclerosis / fibromuscular dysplasia
  - narrowing of artery = impeded blood flow to kidney → renovascular HTN
- presentation: age <30 with HTN or HTN with CAD / PVD, or HTN resistant to 3+ drugs
  - if on ACE-I and develops acute renal failure or sharp rise in BUN/Cr → think renal artery stenosis
- dx: ultrasound = first imaging in age <60, renal arteriography = gold standard
  - may hear renal artery bruit on auscultation

**Testicular Torsion**
- twisting of spermatic cord → compromised blood flow + ischemia (SURGICAL EMERGENCY!)
- s/sx: often after vigorous activity, minor trauma; usually pospubertal boys (age 10-20), asymmetric high riding testicle "bell clapper deformity", negative phren’s sign, loss of cremasteric reflex, more common in pt with hx of cryptorchidism
- dx: testicular doppler = best initial test; radionuclide scan demonstrates decreased uptake in affected testes = gold standard
- tx: surgical emergency → 6 hour time frame for repair with best outcomes
  - followed by elective surgery on contralateral testes which is also at risk for torsion

**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
  - often appear following sexual intercourse in women
- exam usually unremarkable – sometimes suprapubic tenderness
- dx: UA = pyuria, bacteriuria, +/- hematuria
  - urine culture positive for offending organism
  - imaging only warranted 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)

**Varicocele**
- “bag of worms” superior to testicle; formation of venous varicosity within the spermatic vein
- Cystic testicular mass of varicose veins – dilation worse when pt is upright or with Valsalve – decreases in size with elevation of scrotum or supine position
- Dx: scrotal ultrasound = negative transillumination → chronic, nontender mass that does not transilluminate is seen
- Tx: may require surgery if painful or cause of infertility

**CRITICAL CARE**

**Acute abdomen**
- Renal
  - CC: colicky right sided flank pain, nausea, vomiting, hematuria, CVA tenderness
- **Pancreas:**
  - CC: dull epigastric pain that radiates to the back
  - Workup: CT abdomen, CBC, electrolytes, amylase, lipase, AST, ALT, bilirubin, alk phos, U/S abdomen
  - Ddx: pancreatitis, pancreatic cancer, peptic ulcer disease, cholecystitis/choledocholithiasis

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

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

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

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

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

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

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**Adrenal Insufficiency**

- **Primary (Addison’s disease):** autoimmune, infections, disease of adrenal gland → decrease in cortisol secretion
  - Adrenal gland destruction causing lack of cortisol and aldosterone secretion (usually autoimmune)
    - Autoimmune (70%), infectious (tuberculosis), vascular (thrombosis / hemorrhage), metastatic, medications (rifampin, barbiturates, phenytoin, ketoconazole)
  - Dx: increased ACTH, decreased cortisol, decreased aldosterone

- **Secondary:** pituitary adenoma or discontinuation of steroid – pituitary failure!
  - **Exogenous steroid use = MC, Hypopituitarism**
  - Dx: decreased ACTH, decreased cortisol, normal aldosterone

- Adrenal crisis = acute adrenal insufficiency
- **Dx:**
  - 8AM serum cortisol and plasma ACTH alone with ACTH stimulation test
    - High ACTH, low cortisol = primary
    - Low ACTH, low cortisol = secondary
  - CRH stimulation test: differentiates between causes of adrenal insufficiency
    - Primary/addison’s (adrenal): high ACTH, low cortisol
    - Secondary (pituitary): low ACTH, low cortisol
  - Adrenal autoantibodies can be assessed; CXR for TB (CT of adrenals)
- Autoimmune: atrophied adrenals
- TB / granulomas: enlarged adrenals + calcification
- Bilateral adrenal hyperplasia = genetic enzyme defect

**Tx:**
- Addison’s: cortisol replacement therapy + androgen replacement
  - Glucocorticoid + mineralocorticoid $\rightarrow$ hydrocortisone = 1st line, fludrocortisone for primary addison’s disease only
- Secondary: cause = focus of treatment (pituitary adenoma resection, wean steroid therapy slowly)

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

**Melena**
- melena: black tarry stool; upper GI bleed (>50ccs blood to have melena from upper GI bleed)
- **DDX:** gastric CA, duodenal ulcers, portal HTN with esophageal varices, erosive esophagitis, Mallory-weiss syndrome
- **Labs:** CBC, CMP, LFTs, coags (microcytic = chronic bleed; normocytic = acute bleed)
- **dx:** EGD; blood, stool, breathe test for H. pylori
- **tx:** ocreatide + somatostatin: vasoactive drug; sclerotherapy decreases risk for rebleeding in pt with esophageal varices

**Hematochezia**
- **BRBPR**
- **DDx:** hemorrhoids, anal fissures, polyyps, proctitis, rectal ulcers, colorectal CA, diverticular bleed = bigvolume
- **Labs:** CBC, CMP, liver test, coags (microcytic = chronic bleed; normocytic = acute bleed)
- **Dx:** colonoscopy, enteroscopy, barium XR, radionuclide scanning, angiography, laparotomy

Hematochezia + melena tx: endoscopic thermal probe, endoscopic clips, endoscopic injection, angiographic dmbolizaiton, endoscopic intravascular cryanoacrylate injection, band ligation
- Transfuse Hgb <9 in high-risk and <7 in low-risk

Approach to GI bleed: 2 large bore IVs, labs (CBC, CMP, LFTs, coags), fluids, type and cross

**Acute Glaucoma**
- **Open-angle:** MC type; pt will be AA, asymptomatic, diagnosed during routine; slow process
  - Impaired aqueous outflow through diseased trabecular meshwork causing increased IOP $\rightarrow$ gradual increase in pressure and progressive visual field loss
- **Acute: OPHTHALMOLOGIC EMERGENCY:** occurs when iris dilates and pushes against lens of eyes, disrupting flow of aqueous humor into anterior chamber; pressure in posterior chamber then pushes peripheral iris forward and blocks the angle
  - Acute, severe pain, unilateral, decreased visual acuity, HA, n/v
  - **Triad:** injected conjunctiva, cloudy cornea, fixed, dilated pupil
  - (irits presents similarly but it’s a small constricted pupil)
- **Dx:** tonometry showing increased intraocular pressure (may show cupping of optic nerve) $\rightarrow$ GONIOSCOPHY
  - **Pressure >21** = concerning; cup to disc ration >0.5 = suggestive
  - Peripheral field testing and optic disc changes confirm diagnosis in normal pressure glaucoma
- **Tx:**
  - Acute narrow angel: IV Acetazolamide = 1st line, topical beta blocker, miotics / cholinergics, peripheral iridotomy = definitive tx (punch hole in iris)
Chronic open angle: prostaglandins analogs = 1st line (increase outflow aqueous humors); topical beta blocker (decrease production aqueous humor); trabeculoplasty (open trabecular meshwork)

**Acute Respiratory Distress Syndrome**
- Underlying abnormality: increased permeability of alveolar capillary membranes → protein-rich pulmonary edema
- Settings:
  1. Sepsis (MC)
  2. Severe multiple trauma
  3. Aspiration of gastric contents (alcoholics), toxic inhalation, near drowning
- Dx: CXR shows air bronchograms (when infiltrate surrounds peripheral bronchi and is establishing lung consolidation)
- Tx: identify and manage underlying precipitations and secondary conditions
  1. Tracheal intubation with lowest PEEP to maintain PaO2 above 60mmHg or SaO2 above 90% in pt with ARDS

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

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

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

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

**Unstable angina**
- **Previously stable and predictable symptoms of angina that are now more frequent, increasing or present at rest**
  - Chronic angina—increasing in frequency, duration, or intensity of pain
  - New-onset angina—severe and worsening
  - **Angina at rest**
  - **O2 demand unchanged**, supply decreased, secondary to low resting coronary flow

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

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

Angina is chest pain or discomfort attributed to myocardial ischemia, which occurs when myocardial oxygen demand exceeds oxygen supply. Physical exertion, mental stress, tachycardia, hypertension, and ventricular hypertrophy increase oxygen demand, and can therefore lead to myocardial ischemia. Patients with angina classically present with chest pressure, tightness, or heaviness in the middle or left side of the chest that is preceded by exertion and relieved by rest. The pain is usually difficult to locate, and patients usually indicate a diffuse area of discomfort. Angina usually lasts for five to fifteen minutes. Pain that lasts longer than that may be indicative of acute coronary syndrome. An electrocardiogram obtained when chest discomfort is present may reveal J point or ST segment depression indicative of subendocardial ischemia, but the ECG may also be normal.

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

Nonexertional chest pain similar to unstable angina
• Preservation of exercise capacity
• Look for a history of smoking (#1 risk factor) or cocaine abuse
• Pain is cyclical (mostly occur in morning hours, no correlation to cardiac workload)

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

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

Cardiac Arrest

NSTEMI: Myocardial necrosis without acute ST segment elevation or Q waves
• EKG: STD, t wave inversion or both; coronary artery not completely blocked; subendocardial infarct
• Cardiac markers elevated:
  o troponin = most sensitive; appears at 2-4 hours, peaks 12-24 hours, lasts 7-10 days
  o CK/CK-MB: appears at 4-6 hours, peaks at 12-24, returns in 48-72 hours
  o Myoglobin (Mb): used less; appears at 1-4 hours, peak 12 hours, return to baseline 24 hours
• Tx: beta blocker + NTH + aspirin + clopidogrel + heparin + ACE-I + statin + reperfusion
  o Reperfusion via percutaneous coronary intervention
  o Less time-sensitive than STEMI

STEMI: myoccardial necrosis WITH ST changes/ q waves
• Coronary artery completely blocked; full thickness of myocardial wall involved
• ECG: ST elevation, possible q waves
  o Anterior wall: q waves / STE in I, AVL, V2-V6
Inferior: II, III, AVF
Lateral: lateral (I, aVL, V5-V6) + reciprocal STD in inferio leads (III and aVF)
Posterior: STD in V1 – V3

- Tx: BB + NTG + aspirin and clopidogrel + heparin + ACE-I + statin + reperfusion
  - Aspirin and clopidogrel are given at once
  - TIME SENSITIVE!!!! Immediate coronary angiography and primary PCI IN 90 minutes!!!!!
  - Thrombolytic therapy within first 3 hours if PCI not available

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 cather 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 recurrrent 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 exists, 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

**Heart Failure**

- Syndrome of ventricular dysfunction – can be involved together or separately
  - LV failure → SOB + fatigue
  - RV failure → peripheral and abdominal fluid accumulation
- Systolic: S3 – rapid ventricular filling during early diastole
- Dilated thin-walled weak left ventricle (often from CAD / heart attack → muscle dies)
  - s/sx: dyspnea, PND, orthopnea, rales, crackles, displaced own and to the left apical impulse
  - EF <40%
  - Tx: ACE-I, BB, loop diuretic
  - Acute worsening: O2 + ACE + stop BB + start nitro and double dose diuretic IV (when stable → BB + PO loop diuretic)
- **Diastolic**: S4 – hypertrophic thick walled LV with impaired relaxation
  - Increases age 55, common in pt with HTN (often forget to take meds)
  - s/sx: dyspnea and rales with apical heave / lift
  - EF = normal
  - Tx: ACE-I + BB/CCB (do not use diuretics in stable chronic diastolic failure)
  - **NEVER USE DIGOXIN FOR DIASTOLIC**
  - Acute = same as systolic (ACE_- + loop IV + NTG + O2)
- **Right heart failure** (right ventricle)
  - Cause: pulmonary HTN (right heart can’t pump blood to lungs – MC cause right heart failure = left heart failure)
  - s/sx: JVD, leg edema
  - dx: echo and doppler; gold = right heart cardiac catheterization
  - treat underlying condition
  - high output cardiac failure: caused by increased metabolic demand (higher than heart can pump)
    - hyperthyroid, severe anemia, beriberi / thiamine deficiency
    - tachycardia → systolic failure bc heart tires, dilates, weakens
    - tx: like heart failure and acute CHF, treat underlying condition

### Cardiac Tamponade
- fluid between pericardial sac + heart → distant heart sounds, distended jugular veins, decreased arterial pressure
- beck’s triad: hypotension, muffled heart sounds, elevated neck veins (JVD)
- pulsus paradoxus: drop 10mmHg systolic pressure on inspiration, narrow pulse pressure
- dx: EKG shows electrical alternans (consecutive normally-conducted QRS complexes alternate in height) and low voltage QRS
  - DXR: water bottle heart (heart shaped like canteen)
- Tx: IV fluids to increase preload and prevent RV collapse
  - Pericardiocentesis = therapeutic
  - Balloon pericardiectomy and pericardial window may be warranted in cases of decompensation

### Diabetic Ketoacidosis
- Diabetic with history of: infection, ischemia (cardiac, mesenteric), infarction, ignorance (poor control), intoxication
- s/sx: abdominal pain, vomiting, fatigue
- PE: fruity smelling breath, dehydration, AMS
- Labs: hyperglycemia, ketonemia, anion gap metabolic acidosis
- Tx: IVF + insulin infusion
- Corrected sodium add 1.6mEq for each 100mg/dL in serum glucose

### Hypertensive Urgency
- Systolic >180 or diastolic >100 without target-organ damage

### Hypertensive Crisis
- Lower insulin infusion to 0.02 – 0.06 U/kg/h
- Change IV fluid to 5% dextrose with 0.45% NaCl at 150-200 mL/h
- Keep glucose between 150-200 mg/dL, until DKA resolved
- Monitor intake and output
- Supplement K+ at 20-30 mEq until K+ >3.3
- Higher insulin infusion
- Increase IV fluid to 3% dextrose with 0.45% NaCl at 150-200 mL/h
- Lower insulin infusion to 0.02 – 0.06 U/kg/h
- Change IV fluid to 5% dextrose with 0.45% NaCl at 150-200 mL/h
- Keep glucose between 200-300 mg/dL, until DKA resolved
- Monitor intake and output
- Healty 
- Supplement K+ at 4 - 5 mEq
• Immediate BP reduction not required but pt should be started on 2-drug oral combination and close eval
• Clonidine = drug of choice

**Hypertensive Emergency**
• SBP >180 and/or DBP >120 with signs of damage to target organ: encephalopathy, nephropathy, intracranial hemorrhage, aortic dissection, pulmonary edema, unstable angina, MI
• BP must be reduced within 1 hour
• Tx: sodium nitroprusside = drug of choice
  o Potential for developing cyanide toxicity → treat with sodium thiosulfate which combines with cyanide ion to form thiocyanate (nontoxic)
  o Nitro should be avoided in setting of renal failure due to risk of cyanate and thiocyanate toxicity
• Admit to ICU – goal 20-25% reduction in mean arterial pressure in 1-2 hours; decrease 10% in first hour, additional 15% in next 2-3 hours using IV agents

**Malignant HTN:**
• Diastolic reading >140mmHg with papilledema and either encephalopathy or nephropathy
• If untreated → progressive renal failure
• Can be caused by MAOI + food (cheese, sausage, red wine)
• Hydralazine = drug of choice

For all:
• Dx: BP, ECG, UA, ophthalmic exam, serum BUN and Cr

**Pericardial Effusion**
• Same sx as acute pericarditis except signs of fluid build up around heart – low voltage QRS complex, electrical alternans, distant heart sounds
• Dx: EKG shows **low voltage QRS along with electrical alternans**
  o Echo: increased pericardial fluid
  o Radiograph: water bottle heart
• Tx: treat underlying cause – pericardiocentesis if effusion is large
  o Pericardial window if recurrent

**Pericarditis**
• Inflammation of pericardial sac often → pericardial effusion
• Pleuritic chest pain: worse when supine and inspiration; alleviated when pt leans forward
• Dressler’s syndrome: pericarditis 2-5 days after acute MI
• Causes: idiopathic, SLE, uremia, viral (coxsackie), TB, RA, neoplasms, drugs, radiation, scleroderma, MI, open heart surgery, radiotherapy
• Dx:
  o pericardial friction rub heard best with pt upright and leaning forward
  o diffuse, STE in precordial leads
• tx: treat underlying cause
  o NSAIDs, aspirin 7-14 days; abx to treat bacterial endocarditis; pericardiocentesis with tamponade, head at 45 degrees, pericardial window for drainage

**Pneumothorax**
• Accumulation of air in pleural space
• s/sx: acute onset ipsilateral chest pain and dyspnea with decreased tactile fremitus, deviated trachea, hyperresonance, diminished breath sounds
• can be spontaneous or traumatic
  o primary: occurs in absence of underlying disease (tall, thin males age 10-30 at greatest risk)
  o secondary: in presence of underlying disease (COPD, asthma, cystic fibrosis, interstitial lung disease)
• dx: tension pneumothorax = mediastinal shift to contralateral side and impaired ventilation
  o XR = pleural air; ABG shows hypoxemia
• Tx:
  o small - <15% diameter of hemithorax will resolve spontaneously without need for chest tube placement
  o large - >15% diameter and symptomatic pneumothoraces, chest tube placement performed
  o serial XR every 24 hours until resolved
• tension = medical emergency!!! – large bore needles to allow air out of chest; chest tube for decompression
Pulmonary Embolism

- arises from thrombi in systemic venous circulation or right side of heart, from tumors that invade venous circulation or other
- >90% from clots in deep veins of lower extremities (homan’s sign – dorsiflexion foot = calf pain)
- r/f: virchow’s triad – hypercoaguable state, trauma, venostasis (surgery, cancer, oral contraceptives, pregnancy, smoking)
- dx: spiral CT = initial method of identifying
  - ABG = respiratory alkalosis secondary to hyperventilation
  - EKG: S1Q3T3 (rare); non-specific ST wave changes
  - CXR: Westermark sign or Hampton hump (triangular or rounded pleural base infiltrate adjacent to hilum)
  - VQ scan: perfusion defects with normal ventilation (normal VQ rules out PE; abnormal – non-specific)
  - D-dimer
  - Pulmonary angiography = gold standard definitive
- Tx: heparin = anticoagulant of choice or warfarin (INR 2-3)
  - Duration: minimum of warfarin 3 months with reversible risk factor
  - Unprovoked: warfarin recommended for at least 6 months then reevaluate
  - 2 episodes unprovoked, long term with warfarin

Seizures

Focal:
1. retained awareness – consciousness maintained → aka simple partial; no alteration in consciousness
2. loss of awareness – consciousness impaired → aka complex parital; altered consciousness, automatisms (lip smacking); present with post-ictal state (confusion / memory loss) which differential from absence seizures
3. tx: phenytoin, carbamazepine = drugs of choice

Generalized: widespread seizure activity in left and right hemispheres of brain; start midbrain or brainstem and spread to both cortices
1. Absence seizures (aka petit mal): brief impairment of consciousness with abrupt beginning and end; pt has no memory of these and people often miss them
2. Tonic-clonic (aka grand mal): convulsive
   a. Bilaterally symmetric and without focal onset; begins with sudden loss of consciousness; tonic phase (stiff and rigid 10-60seconds); clonic = convulsions / limb jerking; postictal = confused
3. Atonic: drop attacks – looks like syncope, sudden loss muscle tone
4. Clonic: lose control of body functions and begin jerking in various parts of body – may lose consciousness temporarily followed by confusion
5. Tonic: extreme rigidity → immediate LOC, no clonic phase
6. Myoclonic: muscle jerking, occurs in morning, no tonic phase

Additional:
1. Febrile: temp >38F, >6mo, <5years, absence CNS infection / inflammation
2. Infantile spasm: type of epilepsy but don’t fit into category of focal or generalized
3. Psychogenic non-epileptic seizures (PNES): not due to epilepsy but may look similar

Status Epilepticus: single epileptic seizure lasting >5min or 2+ seizures in 5 minute period without person returning to normal between them
1. Convulsive: regular pattern of contraction and extension of arms / legs
2. Non convulsive: complex partial status epilepticus and absence status epilepticus
3. Tx: benzos (lorazepam) and then phenytoin given

SIRS / SEPSIS

- Sepsis: toxic inflammatory condition from microb spread from a focus of infection → triggers systemic inflammatory response (SIRS)
- SIRS: at least 2 of 4: temp <36C or >38C, HR >90, RR >20, WBC <4 or >12
- qSOFA: new or worsened mentation, RR >22, systolic BP <100
- gram positive shock: exotoxins leading to fluid loss (staph / strep)
- gram negative shock: endotoxins: e.coli, klebsiella, proteus, psuedomonas
- dx: respiratory alkalosis with metabolic acidosis, neutropenia/neutrophilia + increased bands, thrombocytopenia, coag studies (possible DIC panel)
- any neonate with 100.4F should get full work-up: CBC, UA, LP
• **Shock**

<table>
<thead>
<tr>
<th>System</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Very mild hemorrhage &lt; 15% vol loss</td>
<td>Mild hemorrhage 15% - 30% vol loss</td>
<td>Moderate hemorrhage 30% - 40% vol loss</td>
<td>Severe hemorrhage ≥ 40% vol loss</td>
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<tr>
<td>Cardiovascular</td>
<td>HR normal or mildly increased</td>
<td>Mild tachycardia</td>
<td>Significant tachycardia</td>
<td>Severe tachycardia</td>
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<tr>
<td>Pulses</td>
<td>Normal</td>
<td>Peripheral pulses may be diminished</td>
<td>Threaded peripheral pulses</td>
<td>Threaded central pulse</td>
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<tr>
<td>pH</td>
<td>Normal</td>
<td>Normal</td>
<td>Mild to moderate acidemia</td>
<td>Significant acidemia</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Normal</td>
<td>Tachypnea</td>
<td>Moderate tachypnea</td>
<td>Severe tachypnea</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Normal/Increased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>CNS</td>
<td>Slightly anxious</td>
<td>Irritable, confused</td>
<td>Irritable or lethargic</td>
<td>Coma</td>
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<td>Urine output</td>
<td>Normal, increased specific gravity</td>
<td>Oliguria, increased specific gravity</td>
<td>Oliguria, increased SUN</td>
<td>Anuria</td>
</tr>
<tr>
<td>Skin</td>
<td>Warm, pink</td>
<td>Cool extremities, mottling</td>
<td>Cool extremities, mottling or pallor</td>
<td>Cold extremities, pallor or cyanosis</td>
</tr>
<tr>
<td>Capillary refill</td>
<td>Brisk</td>
<td>Delayed</td>
<td>Prolonged</td>
<td>Prolonged</td>
</tr>
</tbody>
</table>

- **Thyroid Storm**

- **hyperthyroid state + acute event**
- **r/f:** surgery, trauma, infection, acute iodine load, parturition
- **s/sx:** heat intolerance, palpitations, weight loss tachycardia, anxiety, jaundice
  - jaundice = hepatic tissue hypoxia due to increased peripheral consumption of oxygen
- **clinical:** tachycardia HR >140, heart failure, hypotension, dysrhythmia (afib), hyperpyrexia (104F-106F), agitation, psychosis, coma; hyperreflexia, goiter, exophthalmos, pretible edema; **MC caused by grave’s disease**
- **labs:** free t4/t3, TSH → low TSH and high free T4
- **tx:** beta blocker, thionamides, iodine, hydrocortisone, bile acid sequestrants
  - propylthiouracil – inhibits conversion of thyroxine to triiodothyronine
  - methimazole or PTU (PTU if pregnant)

**HEMATOLOGY**

- **Acute / Chronic Lymphocytic Leukemia**
  - **ALL:** child + LAD + bone pain + bleeding + fever + >20% blasts
    - **Tx:** highly responsive to **chemo**; in relapse → bone marrow transplant
  - **CLL:** middle aged pt – often asymptomatic, fatigue, LAD, splenomegaly
    - **Dx:** SMUDGE cells on peripheral smear, mature lymphoctyes
    - **Tx:** observation, if lymphs >100K or symptomatic → **chemo**

- **Acute / Chronic Myelogenous Leukemia**
  - **AML:** auer rods + >20% blasts in adults >50yo
    - **s/sx:** anemia, nosebleeds, bruising thrombocytopenia, neutropenia, splenomegaly, gingival hyperplasia, bone pain
    - **tx:** combination chemo, bone marrow transplant
  - **CML:** WBC >100K + hyperuricemia + adult >50yo
    - 70% asymptomatic until pt has blastic crisis (acute leukemia)
    - **Dx:** Philadelphia chromosome (translocation chromosome 9 and 22), splenomegaly
    - **Tx:** Gleevec (imatinib) which makes this chronic dz state

**Anemia of Chronic Disease**
• Multifactorial; often with iron deficiency
  • Dx: usually needs presence of chronic infection, inflammation, cancer, microcytic / normocytic anemia, and values for serum transferrin receptor and serum ferritin that are between those typical for iron deficiency and sideroblastic anemia
• Normal/normocytic: decreased MCV, decreased TIBC, increased ferritin 9bc not being transported / utilized)
  o Some transferring converted to lactoferrin which can’t transport ferritin → decreased TIBC
  o Increased ferritin bc not being transported to bone marrow
• MC causes: chronic renal failure and anemia from connective tissue disorder: RA, SLE, HIV, CA, cirrhosis, chronic infection
• Anemia of renal failure: erythropoiesis impaired bc of decreases in EPO production and marrow responsiveness to EPO
  o Renal insufficiency + decreased serum EPO
• Dx: decreased MCV, decreased TIBC, increased ferritin
  o Decreased serum EPO in anemia of renal failure
  o CBC and serum iron, ferritin, transferrin, transferrin receptor
• Tx: treat underlying disorder, recombinant EPO and iron supplements
  o Treat with EPO analog (epogen, Procrit) if Hgb <10 and stop when Hgb >11 d/t increased chance MI / stroke

**Clotting Factor Disorders**
• Proteins that respond in cascade to form fibrin strands that strengthen platelet plug
• Von Willebrand: MC genetic bleeding disorder, autosomal dominant
  o Decreased vWF and decreased factor VIII; vWF found in plasma, platelets, walls of blood vessels; when factor missing / defective, platelets can’t adhere to the vessel wall at site of injury so bleeding doesn’t stop as quickly as it should
  o Presentation: excessive bleeding after cut / increased menstrual bleeding – no hemarthrosis (small amounts of superficial bleeding); hx of bleeding problems – bruise easily, bleed excessively
  o Tx: DDAVP (desmopressin) or transfusion of concentrated blood clotting factors containing vWF
• Hemophilia: X linked recessive = usually in males
  o Hemarthrosis, bruising, bleeding, increased PTT, normal PT and platelets, normal bleeding time decreased factor VIII or IX on assay
  o Dx: functional assay for factor VIII or IX to confirm diagnosis
  o Tx: replacement of factor VIII or IX
    ▪ Hemophilia A: decreased clotting factor VIII – 80% of cases
    ▪ Hemophilia B (Christmas disease): decreased clotting factor IX

**G6PD deficiency**
• Common in blacks that results in hemolysis after acute illness / intake of drugs (antimalarials, analgesics, sulfonamides, dapson) / fava beans
• G6PD deficiency reduces energy available to maintain integrity of RBC membrane, which shortens RBC survival
• Oxidative stress (infection / drug) + AA male + Heinz bodies
  o X-linked recessive, damaged hemoglobin (G6PD protects RC membranes) cause Heinz bodies and bite cells on smear
  o Look for trigger event: antimalarials, sulfa drugs, ASA, febrile illness → causes RBC breakdown
• Dx: G6PD assay: Heinz bodies and bite cells
  o Hemolysis labs: increased indirect bilirubin, decreased haptoglobin, increased LDH
• Tx: avoid triggers + supportive care (transfusion rarely needed)
  o Avoid Bactrim!!

**Hypercoagulable States**
• Pneumonic PVCs!!!:
  o Platelets (too many >1 million) or overactive (TTP, heparin-induced thrombocytopenia, HUS, HELLP)
  o Vascular injury: plaque, trauma, burns
  o Clotting factors: anticlotting factors protein C, protein S, antithrombin II deficient / not working
  o Stasis / Surgery
• Virchow triad: stasis, hypercoagulable state, vascular injury
• Acquired hypercoagulable states: malignancy, pregnancy, nephrotic syndrome, ingestion of meds (estrogen), immobilization, myeloproliferative dz, US/Crohn’s, behcet’s syndrome, polycythemia vera, intravascular devices, DIC, hyperlipidemia, antiphospholipid syndrome
• Heparin can cause HIT, causing decrease platelets followed by platelet activation causing clotting and infarction
• Lupus anticoagulants: IgM or IgG immunoglobulin is seen in 5-10% of pt with SLE but MC without lupus or in those taking phenothiazines

**Factor V Leiden (MC):** procoagulant clotting factor – amplifies production of thrombin → clot formation
  o Mutated factor V resistant to breakdown by activated protein C → hypercoagulability
  o Increased DVT and PE especially in young patients
  o Dx: activated protein C resistance assay; normal PT/PTT
  o Tx: LMWH bridge to warfarin; long term antithrombotic therapy not recommended

• **Protein C Deficiency:** vitamin K dependent anticoagulant liver protein that stimulates fibrinolysis and clot lysis (inactivates factor V and VIII) – potentiated by protein S
  o Increased risk recurrent DVT / PE; may have family hx
  o Dx: protein C / S functional assay: decreased protein C / S activity levels
  o Tx: heparin and oral anticoagulation for life

• **Protein S Deficiency:** vitamin K dependent that is a cofactor for activated protein C, which inactivates procoagulant factors Va and VIIIa → reducing thrombin generation
  o VTE = risk
  o Dx: protein C or S functional assay: decreased protein C or S activity levels
  o Tx: heparin / oral anticoagulation for life

• **Antithrombin III Deficiency:** recurrent venous thrombosis and PE, repetitive intrauterine fetal death (IUFD)
  o Antithrombin III = natural anticoagulant; inhibits thrombin (IIa), Xa and other proteases; potentiated by Heparin
  o Associated with VTE; first episode 20-30yo
  o Asymptomatic pt require anticoagulation only before surgical procedures
  o Pt with thrombotic events reuiqre high dose IV heparin then oral anticoagulation indefinitely

• **Antiphospholipid Antibody Syndrome:** autoimmune; often associated with SLE; characterized by thromboses and recurrent spontaneous abortions
  o Autoantibodies react against platelet membranes, activating endothelial cells and platelets → complement mediated thrombosis
  o Dx: lupus anticoagulant, anticardiolipin, DRVVT test, prolonged PTT
  o Tx: high dose IV heparin with thrombotic events then oral anticoagulation indefinitely

• **Idiopathic Thromobcytopenic Purpura**
  - Autoimmune ab reaction to platelets → splenic platelet destruction after acute infection – chronic in adults; acute / self-limited in kids
  - Primary ITP: no underlying condition
  - Secondary ITP: underlying condition i.e. HIV, HCV, SLE, CLL
  - Presentation: 1-2 weeks after viral infection
  - Dx: isolated thrombocytopenia and otherwise normal CBC and peripheral blood smear
    o Primary: isolated thrombocytopenia <100,000 without a cause
    o Secondary: isolated (<100,000) with an underlying cause (ie HIV)
    o Positive direct coombs test
  - Tx: observation in pt with platelet count >30,000 and no bleeding
    o Steroid with <30,000
    o IVIG with <30,000 with CI to steroids, refractory to steroid tx or bleeding / high risk bleeding
    o Splenectomy = second line tx with refractory

• **Iron Deficiency Anemia**
  - Decreased MCV (microcytic), increased TIBC, decreased ferritin (low iron stories), decreased MCH (hypochromic) – microcytic hypochromic; MC cause microcytic anemia
  - MC cause of anemia and usually from blood loss
In men: chronic occult bleeding; women = menses
• s/sx: fatigue, palpitations, SOB, weakness, HA, tinnitus, koilonychia (nail), atrophic glossitis (tongue), angular cheilitis
• dx:
  o CBC: low reticulocyte count, high RDW
  o Iron studies: decreased serum iron, ferritin, transferrin saturation; increased TIBC, ferritin <15 (diagnostic)
  o H&H: <13.5 & 39 for men; <12 & 37 for women
  o Peripheral smear: poikilocytes; rarely bone marrow examination; hemoccult if indicated
• Tx: ferrous sulfate 3mg/kg once or twice daily between meals with juice (not milk)
  o Ferrous fumarate 100–200mg/day in 2–3 doses; ferrous gluconate 3–6mg/kg/day in 3 doses
  § s/e: gray staining / teeth (liquid preps); GI upset / constipation
• SIX WEEKS TO CORRECT; SIX MONTHS TO REPLETE IRON STORIES; RECHECK BLOOD COUNTS EVERY 3 MO X 1 YEAR
  • other causes decreased MCV: lead poisoning, sideroblastic anemia, basophilic stippling, thalassemia
    o tx = EDTA

**Lymphoma**

Hodgkin’s: HIGHLY CURABLE – painless LAD + reed-sternberg + bimodal age (20s/50s) + B sx
  o Bimodal; peaks 20s and 50s
  o Reed-sternberg cells
  o Painless LAD; contiguous spread to local lymph nodes
  o B sx common: fever, weight loss, night sweats
  o Associated with EBV
  o Excellent 5 year cure rate
  o Dx: CXR to check for mediastinal adenopathy
  o Tx: chemo, radiation, highly curable

Non-Hodgkin’s: HIV pt + IG sx + painless LAD
  o >50 yo; increased risk immunosuppression
  o B cells and T cells
  o Peripheral, multiple nodes; non-contiguous extranodal spread (GI and skin = MC)
  o B sx not common
  o Not associated with EBV
  o Variable cure rate
  o s/sx: SOB, intussusception, bowel obstruction, abdominal masses
  o tx: rituximab, chemo, variable course

**Multiple Myeloma**

cancer of monoclonal plasma cells; MC primary tumor of bone/bone marrow in pt >50yo
• s/sx: bone pain (low back / ribs) + infection; less common: renal failure, spinal cord compression, hyperviscosity syndrome
• dx: serum protein electrophoresis with monoclonal M protein spike
  o UA = Ig light chains (bence jones protein)
  o Peripheral blood smear: RBC rouleaux formation
  o Bone marrow aspiration: “fried-egg” appearance
  o Hypercalcemia
  o Prolonged bleeding time
  o Imaging: punched out lytic bone lesions on XR
• Tx: bone marrow transplant = definitive
  o Melphalan, steroids, thalidomide, bortezomib

**Sickle Cell Anemia**

Sickled cells on peripheral smear + HgbS on hemoglobin electrophoresis
• Chronic hemolytic anemia occurs almost exclusively in blacks
• Sickle-cell shaped RBCs clog capillaries causing organ ischemia (crises)
• Very high retic count can be used to monitor event (decreases as pt improves)
• Dx: HgbS on hemoglobin electrophoresis; sickled cells, polychromasia, reticulocytosis, howel jolly bodies, mildly increased WBCs, Hgb 8-10, Hct 20-30
  o RBCs = normochromic, normocytic
  o Hb SS = disease
  o Hb SA = trait
Two parents with sickle cell trait = 1 in 4 chance of having child with Hb SS

- Tx: high flow O2, pain control during crisis, supportive transfusion when Hgb < 6
- Infection, bone marrow aplasia, lung involvement can develop acutely and be fatal; normocytic hemolytic anemia = characteristic

**Thalassemia**

- Unbalanced Hb synthesis caused by decreased production of one globin polypeptide chain (beta, alpha,)
- Abnormal formation of hemoglobin; hypochromic and microcytic
- Decreased MCV, normal TIBC, positive Hb electrophoresis \(\rightarrow\) nucleated erythroblasts, target cells, small, pale RBCs, punctate / diffuse basophilia
- Iron deficiency \(\rightarrow\) RBC low; thalassemia – normal to high
- B: decreased B chains – pt are normal at birth but sx at 6 mo old
  - Dx: microcytic, normal TIBC, normal ferritin, + Hgb electrophoresis (decreased HgbA)
  - Tx: minor = no care; major: weekly blood transfusions, iron chelating, splenectomy, bone marrow transplant
- Alpha: decreased alpha polypeptide chain; 2 defects = mild to moderate anemia no sx; 3-4 genes = more severe; in all 4 genes = lethal in utero (fetalis hydrops)
  - Dx: microcytic, normal TIBC, normal ferritin, + Hgb electrophoresis (decreased HgbA)
  - Target and teardrop cells; basophilic stippling
  - Tx: mild = no tx; moderate: folate, avoid oxidative stress; severe: weekly blood transfusion, iron chelating agents, splenectomy, bone marrow transplant
- Hgb electrophoresis confirms diagnosis

**Thrombotic Thrombocytopenic Purpura**

- TTP: decreased platelets + anemia + schistocytes
- Purpura + FAT RN \(\rightarrow\) fever, anemia, thrombocytopenia, renal failure, neurological sx
  - Triggers: drugs (quinine, ticlopidine, clopidogrel, cyclosporine), SLE, infections, AIDS, malignancies
  - Dx: CBC normal except low platelets, schistocytes (RBC fragments) on smear (-) coombs test
- Hemolytic uremic syndrome: decreased platelets + anemia + renal failure (associated with E.coli O157 – children with epoure to E.coli (ground beef) + severe kidney problems
- TTP vs ITP: ITP = insidious and chronic from TTP which is acute febrile disease with multi-organ thrombosis
- Tx: plasmapheresis (plasma exchange)
  - adults with TTP also give steroids
  - HUS refractory to plasma exchange / steroids \(\rightarrow\) eculizumab

**Vitamin B / Folate Deficiency**

- Folate: decreased folate, increased MCV (macrocytic); no neurological sx; common – from inadequate intake, malabsorption, drugs
  - causes megaloblastic anemia; maternal deficiency \(\rightarrow\) neural tube birth defects
  - dx: CBC and serum B12/folate levels (folate <3)
    - normal MMA levels may differentiate folate from B12 deficiency (MMA levels rise in B12 but not folate)
  - tx: folate 400-1000ug po once/daily (normal = 400 per day)
    - have to rule out B12 if folate deficiency is present
- Vitamin B12: macrocytic + hypersegmented neutrophils, neuropathy (los proprioception and decreased vitratory sense), increased MMA in early B12 loss
  - can be malabsorptive (ETOH, gastric bypass) or secondary to decreased intrinsic factor (pernicious anemia + schilling test + decreased intrinsic factor
  - dx: increased MCV and hypersegmented neutrophils on blood smear
    - normal folate, increased MMA, increased homocysteine
  - tx: IM B12 in pernicious or sublingual

**INFECTIOUS DISEASE**

**Botulism**

- caused by clostridium botulinum from canned-food products / honey in infants
  - wait at least until 12 mo for infants to have honey
- sx: diplopia, dry mouth, dysphagia, dysphonia, muscle weakness \(\rightarrow\) respiratory paralysis
- dx: toxin assays, electromyography \(\rightarrow\) see toxin in serum or stool by isolating organisms from stool
- tx: supportive care; activated charcoal sometimes helpful, equine heptavalent antitoxin= benefit if given >72hr after sx onset
- exposure to moist heat at 120C for 30min kills spores but toxins are destroyed by heat 80C for 30min

Candidiasis
- diffuse erythema, “beefy red” with sharp, marginated border with pinpoint satellite pustules at edge of erythema; favors skinfolds/creases; commonly associated with immunocompromised states (HIV, cancer, diabetes)
- types: vulvovaginal, skin/nail, oral thrush
- dx: KOH prep $\rightarrow$ budding yeast, hyphae, pseudohyphae
- tx:
  - vaginal: miconazole cream or fluconazole 150mg single dose
  - oropharyngeal: nystatin suspension
  - esophagitis: fluconazole
  - pregnancy: topical x7days rather than oral -azole

Chlamydia
- MC STI; caused by chlaymdia trachomatis
- s/sx: mucopurulent cervical discharge, multiple sex partners, cervical motion tenderness, painful / frequent urination
  - men: urethritis, bartholinitis, cervicitis, dysuria, abnormal discharge, post-coital bleeding
- dx: NAAT = gold standard
- tx: azithromycin 1gpo x1 or doxy BID x 7 days + ceftriaxone 250mg x 1 to cover gonorrhea

Cholera
- from Vibrio cholera (gram negative, oxidase positive, comma-shaped bacteria $\rightarrow$ life threatening, rice water diarrhea
- from water / seafood, usually in endemic areas; usually free of cholerae within 2 weeks of cessation diarrhea; chronic biliary tract carriers
- dx: confirmed by stool culture; rapid dipstick testing can be used in public health setting without access to labs
- tx: fluid replacement (mild); doxy, azithro, Bactrim, cirpo; adequate hydration

Cryptococcosis
- caused by fungus cryptococcus neoformans (AIDS / immunocompromised states) – considered AIDS-defining illness – found in pigeon/bird droppings and transmitted through inhalation
  - can lead to meningoencephalitis / pneumonia
- dx: with CSF and serum serology $\rightarrow$ india ink may be positive; pleocytosis (lymphocytes), increased opening pressure, increased protein, decreased glucose
  - CT or MRI indicated if you suspect cryptococcoma
- Tx: amp B + flucytosine for 2 weeks followed by fluconazole for 10 weeks
  - Prophylaxis if CD4 <100 with fluconazole

Cytomegalovirus infections
- In herpesvirus family; HHV 5; can cause infections similar to mononucleosis but no severe pharyngitis; can cause inflammation of retina especially with CD4 <50; can cause severe systemic dz in infants (TORCH)
- Dx: tissue biopsy (owl’s eye inclusions); culture = difficulty; antigens can be detected in blood, urine, CSF via PCR
- Tx: ganciclovir, foscarinet, cidofovir; initial IV loading followed by maintenance therapy

Diphtheria
- Gram + bacilli Corynebacterium diphtheriae
- Sx: pseudomembranous pharyngitis, transmitted through respiratory excretions / inhalation; fever, nasopharyngeal sx, pseudomembranes: friable gray/white membranes on pharynx that bleeds if scarped + bull neck: neck swelling 2/2 enlarged cervical LAD
- dx: characteristic membrane, low grade fever, pharyngitis, cervical LAD, palatal parylsis, stridor
  - PCR for rapid detection, culture to confirm, serial EKGs and cardiac enzymes
- Tx: diphtehria antitoxin + erythromycin or penicillin x2 weeks
  - Post exposure antibiotics: all close contacts examined for 7 days
    - Asymptomatic = macrolide abx or penicillin injection
    - Vaccine (DTap) given at 2,4,6, 15-18 mo and booster at 4-6yo

Epstein-Barr Virus
• “mono” triad – fever + LAD + pharyngitis
  Incubation = 30-50 days, transmission via oropharyngeal secretions / saliva
  s/sx: fever, LAD, sore throat, Maculopapular rash if treated with ampicillin (80% pt); splenomegaly / risk splenic rupture
  dx: atypical lymphocytes, monospot test – appears in first 2 weeks of illness, usually slow decline over 6 mo

Gonococcal Infections
• gram negative diplococci: women = often ax but can → PID; men = yellow, creamy profuse and purulent discharge
  sequelae: gonococcal pharyngitis: sore throat; septic arthritis, PID, fitz-hugh-curtis dyrome
  dx: NAAT assay of discharge or urine
  tx: CDC recommends DUAL THERAPY → 250mg IM ceftriaxone (gonorrhea) + 1g oral azithromycin (chlamydia)
    o always empirically treat chlamydia with azithro or doxy
    o check other STIs and treat partners
    o think of gonococcal pharyngitis in anyone with persistent pharyngitis and take samples for culture

Herpes Simplex
• sx: grouped vesicles on erythematos base that burn/sting; HSV 1 = oral; HSV 2 = genital
  HSV 1 – oral lesions; HSV 2 – genital, HHV 3 – VZV (zoster), HHV 4 – EBV (mono); HHV 5 – CMV, HHV 6 – roseolovirus; HHV 7 (similar to HHV 6 and not classified); HHV 8 = kaposi’s sarcoma
  After all initial infections they remain latent; transmission usually requires intimate contact
  Dx: mucocutaneous infections = clinical but do viral cluture, PCR, antigen detection
    o Herpes zoster: tzanck prep + for multinucleated giant cells

Histoplasmosis
• AIDS defining illness, associated with soil contained birds and bat droppings in Mississippi / Ohio River Valleys
  Highest risk = CD4 < 100
  s/s: fever, multiorgan failure, fulminant disease, septic shock, death; ax in most cases
  dx: culture = gold standard; incrased alk phos and LDH, + blood cultures if disseminated
  tx: itraconazole orally for weeks to months recommended
    o amp B if can’t tolerate / fail itraconazole; don’t administer antifungal prophylaxis with itraconazole to prevent primary infection

HIV
• CD4 count <200, recurrent severe and life-threatening opportunistic infections / malignancies
  Dx: ELISA: if positive, then confirm with western blot
    o Rapid: blood or sleive; western blot = confirmatory
    o HIV RNA viral load can be positive in window period, used to monitor infectivity and tx effectiveness in pt dx with HIV
  Tx: combo therapy based on CDS count and viral load
    o HAART initiatin for pt with CD4 <350 or viral load by PCR-RNA >55,000
      ▪ NNRTI + 2NRTI or PI + 2NRTI
      ▪ INTI + 2 NRTIS
    o PEP started within 72 hours of exposure; chance of getting from needle stick is <0.3%; can be transmitted through breastmilk – should be on ART throughout pregnancy
• All CD4 counts: tuberculosis
  <250 = coccidioidomycosis: perform annual IgG and IgM serologic screening + fluconazole therapy if newly positive
  <200 = pneumocystis: Bactrim or dapsone if can’t do Bactrim
  <150 = histoplasmosis – don’t administer antifungal prophylaxis with itraconazole
  <100 = toxoplasmosis, cryptococcus: suppressive therapy with Bactrim with CD4 <100 and positive IgG – prevention not recommended
  <50 = MAC (mycobacterium acvium complexes) – no prophylaxis

Influenza
• Caused by orthomyxovirus → fever, coryza, cough, HA, malaise – strains A,B,C; complications in young, old, immunocompromised
• Everyone >6mo should receive annual influenza vaccine
  o Avoid vaccination: severe egg allergy, previous reaction, guilain barre within 6 week of previous vaccinaition, GBS in the past 6 weeks, <6 mo old, avoid FluMis in pt with asthma
• Dx: rapid antigen test in clinic, rapid serology test more accurate
- CXR: primary flu pneumonia will show bilateral diffuse infiltrates
- Tx: Antiretroviral tx in <48 hours – Tamiflu (oseltamivir)
  - Indications for tx: hospitalized, outpatient with severe/progressive illness, outpt at high risk for complications (immunocompromised, pt with chronic medical conditions, >65 yo, pregnant women / 2 weeks post partum)

**Lyme Disease**

- Caused by borrelia burgdorferi – that’s spread by deer tick
  - Early localized: 7-10 days after bite (erythema migrans rash = bullseye)
  - Early disseminated: 1-12 weeks after bite: MSK, flu-like syndrome, malaise, fatigue, chills, fever, HA, stiff neck
  - Late: persistent synovitis and arthritis
- Dx: ELISA testing positive by 3rd week
- Tx: doxycycline / amoxicillin 10-21 days started immediately after diagnosis
  - Prophylaxis: doxy 200mg x 1 dose within 72 hours if Ixodes tick
  - Doxy avoided in children <8 yo – should received amoxicillin or cefuroximine instead
- Late lyme: IV therapy (ceftriaxone, cefotaxime, penicillin G x28 days)

**Parasitic Infection**

- Helminth: intestinal;
  - Nematodes: cause GI sx + cough; tx with mebendazole
  - Pinworm: anal pruritus in child in morning, scotch tape test
  - Roundworm (ascaris lumbricoides) abdominal sx
  - Cestodes: tapeworm – GI sx and weight loss – tx with praziquantel
  - Trematodes: fluikes, avian and mammal schistosomes
  - Dx: in all – eosinophilia may be present
  - Tx:
    - Pinworm + roundworm = mebendazole
    - Tapeworm + fluke = praziquantel
- Malaria: chills, fever, sweats (fever every 3 days) – transmitted by anopheles mosquito; splenomegaly after >4days of sx
  - Dx: giemsa stain peripheral smear – parasites in RBCs, thrombocytopenia, increased LDH
  - Txz: chloroquine, mefloquine
- Pinworms: enterobiasis vermicularia – MC sx = itching in anal area (difficult sleep)
  - Perianal itching at night; eggs cling to fingers (transmitted to others either directly or food/surfaces); eggs can thrive 2-3 weeks on inanimate object
  - Dx: scotch tape test; can see eggs under microscopy
  - Tx: albendazole, mebendazole
- Toxoplasmosis: encephalitis + chorioretinitis + intracranial calcification in AIDS pt CD4 <100
  - Pregnant female with exposure ot cat feces; part of TORCH
  - Dx: CT brain shows ring-enhancing lesions
  - Tx: prophylaxis for all HIV pt with CD4 <100 with Bactrim
- Trichomoniasis: sexually active woman, copious malodorous green/yello “frothy” discharge
  - Strawberry cervix, motile flagellated protozoa
  - Tx: metronidazole 2g po x 1 dose

**Pertussis**

- Highly contagious gram neg bacteria: Bordetella pertussis – consider pertussis in adults with cough >2 weeks
- Dx: nasopharyngeal secretions; culture = standard
- Tx: macrolide – clarithromycin or azithromycin + supportive care (steroids +/- beta2 agonist)
- Vaccine: 5 doses DTaO vaccine (2, 4, 6, 15-18 mo; 4-6 years); during pregnancy 27-36 weeks

**Pneumocystis (PCP)**

- Yeast-like fungus transmitted by aerosol route and causes NO disease in immunocompetent pt; usually CD4<200
- Dx: CXR (diffuse, bilateral perihilar infiltrates), pulse ox, histopathologic confirmation, elevated LDH
- Tx: Bactrim and steroids (pentamidine for allergy); prophylaxis with daily Bactrim for high risk CD4 <200

**Rabies**

- Rapidly progressive CNS infection caused by RNA rhabdovirus affective mammals/humans
  - s/s: hydrophobia (can’t swallow water), pharyngeal spasm, aerophobia, hyperactivity
• transmission via dogs, raccoons, skunks, bats, fox, coyote
• incubation 1-3 mo; 100% fatal
• dx: negri bodies = pathognomonic and found in brain of dead animals (animals observed 7-10 days before being given back to owner)
• tx: rabies vaccine not 100% preventative; once symptoms occur patients rarely survive
  o post-exposure prophylaxis vaccine on day 0,3,7, 14 + rabies immune globulin ½ injected into wound and ½ given IM

Rocky Mountain Spotted Fever

• Rickettsia rickettsiae: dog tick
• s/sx: 2-4 days after bite = fever, chills, myalgias, HA
  o red maculopapular rash on wrists and ankles then spreads centrally over 2-3 days
• dx: made by skin biopsy; Rumpel-Leede phenomenon = petechiae formation after bp cuff inflation
• tx: doxycycline 200mg po once followed by 100mg bid until pt improves, afibrile 24-48 hr, and received tx at least 7 days
  o chloramphenicol = 2nd line
  o no effective vaccine

Salmonellosis

• typhoid and enteric fever: salmonella typhi – flu-like bacterial infection → fever, GI sx, HA, transmitted via fecal-oral
  o pea soup diarrhea, rose spots, MC in developing world
• gastroenteritis: improperly handled food that’s contaminated by animal or human fecal material; can also be acquired via fecal-oral – usually egg yolk
• dx: blood, stool, urine cultures
  o increased WBC, low platelets
• tx: ceftriaxone and sometimes fluoroquinolone or azithromycin

Shigellosis

• gram negative bacteria → watery diarrhea / dysentery (frequent and painful passage) → usually with blood, pus, mucus
• s/sx: abrupt diarrhea, lower abdominal cramps, tenesmus, fever, chills, anorexia, HA, malaise
  o stool mixed with blood / mucus; tender abdomen
  o HLA-B27 may get reactive arthritis
  o Often spread in crowded areas (daycare) – transmission via person-to-person contact and contaminated foods / water
• Dx: stool positive for leukocytes and RBCs; culture yields shigella spp; sigmoidoscopy: inflamed engorged mucosa, punctate lesions, ulcers
• Tx: fluid replacement + Bactrim (amoxicillin not effective); do not use anti-diarrheals (prolong illness)

Syphilis

• Spirochete treponema pallidum – 3 phases:
  o Primary (3-90 days after exposure) \:\; painless chancre → persists 3-6 weeks
  o Secondary (4-10 weeks): rash on palms and soles or condyloma lata
  o Tertiary (latent – 3-15 years): widespread systemic involvement, permanent CNS changes, gummas
  o Congential: hutchinson teeth (notches), saddle-nose deformity, TORCH
• Dx: VDRL, RPR, ICE → treponemal antibody-absorption test = confirmatory
  o May cause false positive: IVDU, lyme, pneumonia, malaria, pregnancy, SLE, TB
  o LP performed in pt suspect of having neurosyphilis with no contraindication
• Tx: IM benzathine penicillin for primary and secondary disease; IV penicillin G for congenital / late dz
  o Aspirin / tylenol (supportive care) for Jarisch Herxheimer reaction – occurs within first 24 hours after being treated for spirochetal infection (fever, chills, malaise, HA, LAD< hyperventilation, hypotension, worsening of lesions)
    • Treatment with abx → sudden release of bacterial products → incrcased fever / constitutional sx

Tetanus

• Results from toxin by anaerobic bacteria clostridium tetani → rigid muscles / involuntary spasm + exaggerated reflexes
• Puncture wounds = most susceptible; incubation period = 5-15 weeks
• s/s: pain and tingling at site of inoculation → spasticity of muscles nearby; jaw and neck stiffness, dysphagia, irritability are common; hyperreflexia and muscle spasm develop esp in jaw; painful tonic convulsions and asphyxia if untreated
• Dx: can sometimes grow in culture but don’t rely on culture to diagnose tetanus
• Tx:
  o Post-exposure prophylaxis: vaccination (Td, Tdap, TT) – clean wound and vaccination history out of date / unkonw
Hyperimmune human globulin neutralizes toxin
- Metronidazole or penicillin to eradicate toxin producing organism
- Spasmolytic drugs
  - Prevention: vaccine – 5 doses starting at 2 mo of age; Td booster every 10 years; prophylaxis for burns / open fractures

**Toxoplasmosis**
- Parasitic protozoan that causes disease toxoplasmosis; part of TORCH
- Triad: encephalitis, choreioretinitis, intracranial calcifications in AIDS pt with CD4 < 100
- Dx: CT brain shows ring enhancing lesions; ELISA positive for anti-toxoplasma IgG and IgM
- Tx: prophylaxis all HIV pt with CD4 <100
  - Most effective regimen in immunocompetent pt is pyrimethamine + sulfadiazine

**Tuberculosis**
- Mycobacterium tuberculosis – transmitted through respiratory droplets
- S/sx: fever, night sweats, anorexia, weight loss
- Dx: tuberculin skin test (TST) or interferon – gamma release assays IGRAs
  - PPD rules: >5: at high risk, CXR with evident, immunocompromised HIV / drugs, steroids daily, or close contact with pt with infectious TB
  - >10: some risk factors, IVDU, recent immigrants from high prevalence area, renal insufficiency, prison, homeless shelter, diabetes, head/neck cancer, gastrectomy / jejunostomy bypass surgery
  - >15 = no risk factors
- Diagnosis with sputum for AFB smears and cluttures – have to be 3 afb negative
  - NAAT helps diagnosis etter and sooner
  - XR: cavitary lesions, ghn complexes in apex of lungs, caseating granulomas
  - Military TB = spread outside lungs à potts dz (TB in spine); scrofula (TB to cervical lymph nodes)
- Tx: start meperic tx in those who likely have it
  - PPD positive + CXR negative: latent TB à isoniazid x9mo (+ B6 to prevent neuropathy)
  - PPD + CXR positive = active: quad therapy (RIPE): rifampin, isoniazid, pyrazinamide, ethambutol – all = hepatotoxic
    - **Four drugs x8 weeks (RIPE) then two drugs x16 weeks (RI)**
      - Rifampin: orange body fluids, hepatitis
      - Isoniazid: peripheral neuropathy (B6 = pyridoxine 25-50mg/day)
      - Pyrazinamide: hyperuricemia (gout)
      - Ethambutol: optic neuritis, re-green blindness
  - Prophylaxis household members: isoniazid x1 year
  - Monitor serum creatinine; take meds on empty stomach since food can reduce absorption, watch for hepatotoxicity, aware of drug interactions especially with HIV meds

**Varicella Zoster Virus**
- Transmitted via respiratory droplets 10-20 days incubation
  - Varicella (chicken pox) = primary infection: dew drops on rose petal – start on face spreads down
  - Herpes simplex (shingles) – varicella reactivion along dermatome
    - Zoster ophthalmicus: involves CCN V, keratoconjunctivitis
    - Zoster oticus (ramsay-hung): facial nerve (CN VIII) otalgia, lesions on ear, auditory canal/TM, facial palsy
    - Postherpetic neuralgia: pain >3mo, paresthesias, decreased sensation
- Dx: clinical; serology / fluorescent microscopy will confirm diagnosis
  - Zoster can be idetifed via tzanck smear with visualization of multinucleated giant cells
- Tx:
  - Chicken pox: symptomatic, acyclovir in special populations
  - Shingles: acyclovir, valacyclovir for 72 hours to prevent post-herpetic neuralgia
  - Zoster ophthalmicus: oral antivirals +/- acyclovir ophthalmic
  - Zoster oticus (ramsay-hung): oral acyclovir and steroids
  - Postherpetic neuralgia; gabapentin or TCA, topical lidocaine gel, capsicin
- Herpes zoster vaccine is live attenuated – recommended for immunocompetent adults >60 yo