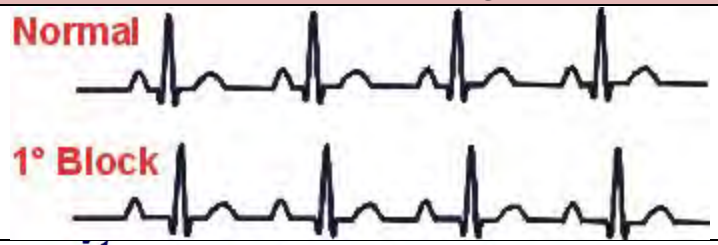
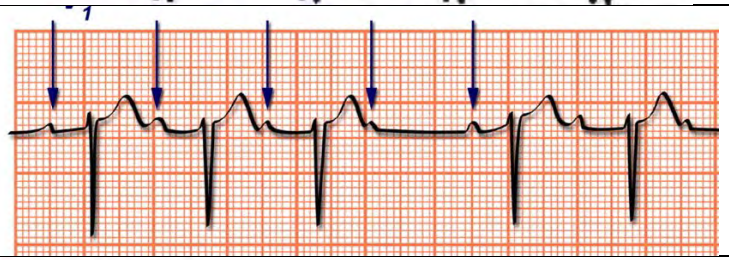
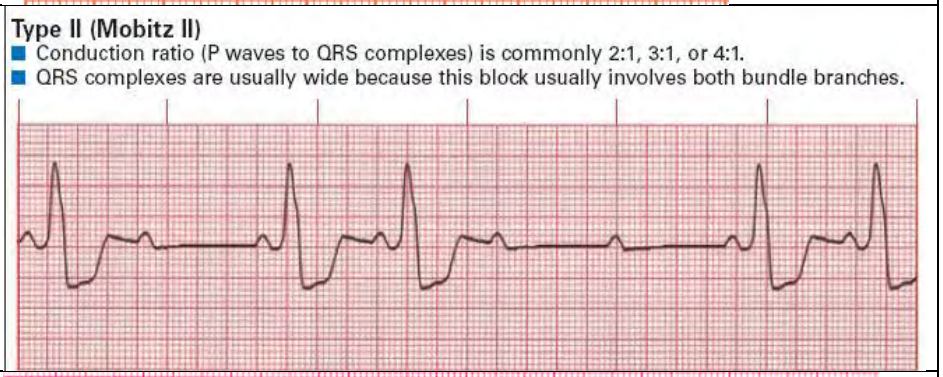
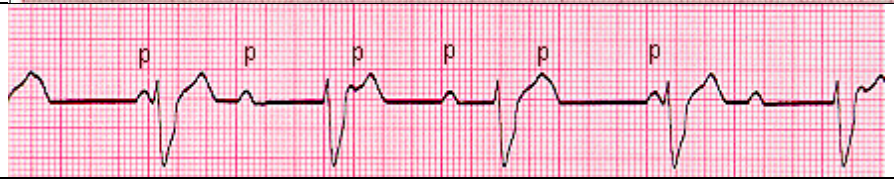
CARDIOLOGY			
Antithrombotic Therapies			
Anticoagulants: for treatment of venous clots or risk of such as factor V Leiden disorder, other clotting disorders, post PE, post DVT	**Vitamin K antagonists**	Warfarin (Coumadin)	-Impairs hepatic synthesis of thrombin, 7, 9, and 10 -Interferes with both clotting and anticoagulation = need to use another med for first 5 days of therapy -Must consume consistent vit K -Pregnancy X -Monitor with INR and PT twice weekly until stable, then every 4-6 weeks
		Jantoven	-Rarely used, usually only if there is a warfarin allergy
		Marvan	
		Waran	
		Anisindione (Miradon)	
	Heparin		-IV or injection -Short half-life of 1 hour -Monitored with aPTT, platelets for HIT -Protamine antidote
	LMWH	Ardeparin (Normiflo)	-Inhibit factors 10a and thrombin -Injections can be done at home -Useful as bridge therapy from warfarin prior to surgery -Monitor aPTT and watch platelets initially for HIT, then no monitoring needed once goal is reached? -Safe in pregnancy
		Dalteparin (Fragmin)	
		Danaparoid (Orgarin)	
		Enoxaparin (Lovenox)	
		Tinzaparin (Innohep)	
	Heparinoids	Fondaparinux (Arixtra)	-Direct 10a inhibitor -Only anticoagulant that does not affect thrombin
		Rivaroxaban (Xarelto)	
	Direct thrombin inhibitors	Dabigatran (Pradaxa)	-Monitor aPTT
		Lepirudin (Refludan)	
		Bivalirudin (Angiomax)	
Antiplatelets: used for arterial clots or risk of such as stroke, TIA, atherosclerosis, CAD, MI, angina, PVD, post PCI, post CABG, a-fib	**COX inhibitors**	Aspirin	-Blocks thromboxane A-2 = only 1 platelet pathway blocked = weak antiplatelet -Only NSAID where antiplatelet activity lasts for days rather than hours
	ADP receptor inhibitors	Ticlopidine (Ticlid)	-No monitoring needed -May ease migraines
		Clopidogrel (Plavix)	-Needs monitoring during initiation due to risk of blood count abnormalities
		Ticagrelor (Brilinta)	
		Prasugrel (Effient)	
	PPD inhibitors	Cilostazol (Pletal)	-Contraindicated in CHF -May be useful in PVD as it widens leg arteries -Pregnancy X
	Glycoprotein IIB/IIIA inhibitors	Abciximab (ReoPro)	-IV only
		Eptifibatide (Integrilin)	
		Tirofiban (Aggrastat)	
		Defibrotide	
	Adenosine reuptake inhibitors	Dipyridamole (Persantine)	-Strong treatments for prevention of recurrent stroke

CARDIOMYOPATHIES					
-A group of diseases of the myocardium associated with mechanical or electrical dysfunction that usually exhibit ventricular hypertrophy or dilation -Current major society definitions of cardiomyopathies exclude heart disease secondary to CV disorders such as HTN, CAD, or valvular disease -Etiologies may be genetic, inflammatory, metabolic, toxic, or idiopathic					
Type	**Info**	**Signs & Symptoms**	**Workup**	**Management**	**Prognosis**
Dilated Cardiomyopathy: dilation and impaired contraction of one or both ventricles	-Common etiologies: viral, genetic, alcoholism -Systolic dysfunction	-CHF -Arrhythmias -Sudden death -Exercise intolerance -Fatigue or weakness -Dyspnea		-Treat CHF symptoms -ICDs -Eval for transplant	
Hypertrophic Cardiomyopathy: disorganized hypertrophy of left ventricle and occasionally right ventricle	-Caused by genetic mutations -Diastolic dysfunction -Usually asymptomatic until childhood or adolescence -Athletes with underlying HOCM at greater risk for lethal arrhythmia during exertion -May have abnormal SAM movement of mitral valve	-Varied presentation, may be asymptomatic -CHF -DOE: the most common sx -Orthopnea and PND -Exertional chest pain -Atypical chest pain -Syncope and presyncope -Palpitations -Postural hypotension -Fatigue -Edema -Arrhythmias -Harsh crescendo systolic murmur ± mitral regurg -S4 -Displaced apical impulse or thrill -Sudden death -Stroke	-Differential: athlete's heart (physiologic LVH), HTN, aortic stenosis -Valsalva will increase HCM murmur and decrease aortic stenosis murmur -EKG: prominent Q waves, P wave abnormalities, LAD -Echo -Holter monitor -Exercise stress test -Screen relatives	-β-blockers to reduce O2 demand -CCB to reduce contractility and improve diastolic relaxation -Pacer or AICD -Surgical myectomy, mitral valve surgery, or ethanol ablation to destroy thickened septum	-Annual mortality of 1% -May progress to dilated cardiomyopathy
Restrictive Cardiomyopathy: diastolic dysfunction → normal contractility but rigid and stiff ventricular walls	-Etiologies: scleroderma, amyloidosis, genetic, HOCM, DM, chemo, HIV -Uncommon in US	-R CHF as pulmonary pressures must increase to deliver blood	-Differential: constrictive pericarditis		
Arrhythmogenic Right Ventricular Cardiomyopathy/Dysplasia: RV wall replaced with fibrous tissue	-Genetic cause	-Ventricular arrhythmias			
Unclassified Cardiomyopathies	-Includes stress-induced cardiomyopathy and left ventricular noncompaction				

ARRHYTHMIAS AND CONDUCTION DISORDERS				
Atrioventricular Block				
Type		**Signs & Symptoms/Info/Workup**	**Management**	**EKG**
First degree		-Asymptomatic -EKG showing lengthened PR interval -Determine site of block using EKG findings, atropine, exercise, or vagal maneuvers	-Treat reversible causes such as ischemia, increased vagal tone, or meds -Pacemaker usually not recommended	
Second degree	**Wenckebach (Mobitz type I)**	-Typically asymptomatic -EKG shows progressive PR prolongation for several beats prior to nonconducted P wave -Beats classically occur in ratios of 3:2, 4:3, or 5:4 -Can be a result of inferior MI	-Treat reversible causes such as ischemia, increased vagal tone, or meds -Pacemaker if there is symptomatic bradycardia	
	Mobitz type II	-May be asymptomatic or have signs of hypoperfusion or HF -PR interval remains unchanged prior to a nonconducted P wave	-Treat reversible causes such as ischemia, increased vagal tone, or meds -Most patients will require a pacemaker	
Third degree		-May have dizziness, presyncope, syncope, v-tach, v-fib, worsening HF, or angina -P waves don't correlate to QRS -Escape rhythm takes over for QRS (junctional or ventricular)		

Bundle Branch Block

-Occurs when block in left or right BB delays depolarization to a ventricle
-Variation is "intermittent Mobitz" where there is a RBBB or LBBB plus intermittent BBB of opposite side → can progress to 3° AV block

Causes
-Structural heart disease: cor pulmonale, pulmonary embolism, MI or ischemia (both branches receives blood supply from LAD), myocarditis, HTN, congenital heart disease
-Iatrogenic: R heart cath, ethanol ablation

Differential
-Any BBB: ventricular rhythm or ventricular pacing
-RBBB: Brugada syndrome

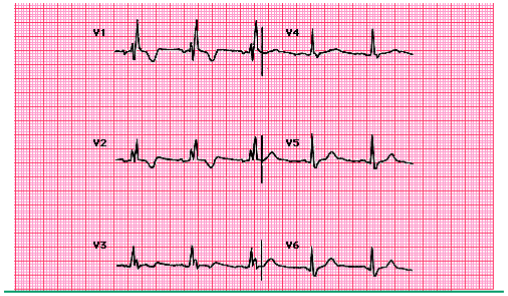
BBB and acute MI
-LBBB interferes with dx of ventricular hypertrophy, myocardial ischemia, and acute MI because of associated Q waves (early ventricular depolarization is affected) as well as ST changes (ventricular repolarization is affected)
-RBBB usually does not interfere with dx of Q wave MI (early ventricular depolarization is not affected) but can inflict ST segment changes

Workup
-RBBB: if asymptomatic and no other evidence of cardiac disease, no further w/u indicated
-LBBB needs further w/u for cardiac cause

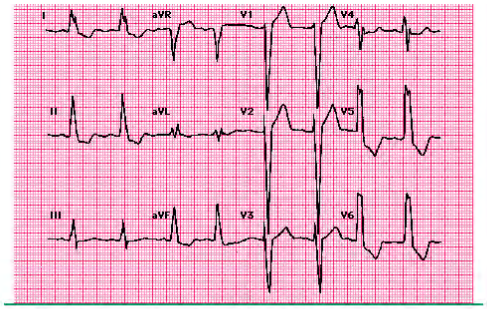
Management
-Permanent pacemaker insertion for symptomatic BBB or progression to AV block

Prognosis
-LBBB in older individuals associated with increased mortality

EKG
-Joined QRS's or "rabbit ears"
-May have accompanying ST or T wave change due to altered sequence of repolarization
-RBBB will be prominent on R heart leads (V1 and V2)
-LBBB will be prominent on L heart leads (V5 and V6)



Electrocardiogram showing characteristic changes in the precordial leads in common RBBB. The asynchronous activation of the two ventricles increases the QRS duration (0.13 sec). The terminal forces are rightward and anterior due the delayed activation of the right ventricle, resulting in an rsR' pattern in the anterior-posterior lead V1 and a wide negative S wave in the left-right lead V6 (and, not shown, in lead I).



Electrocardiogram in typical complete LBBB. The asynchronous activation of the two ventricles increases the QRS duration (0.16 second in this example). The abnormal initial vector results in loss of "normal" septal forces as manifested by absence of q waves in leads I, aVL, and V6. The late activation of the left ventricle prolongs the dominant leftward progression of the middle and terminal forces, leading to a positive and widened R wave in the lateral leads. Both the ST segment and T wave vectors are opposite in direction from the QRS, a "secondary" repolarization abnormality.

Sick Sinus Syndrome

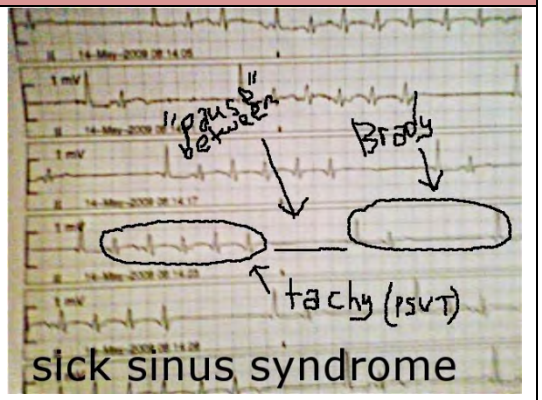
-A combination of unhealthy SA that stops pacing intermittently + unresponsive supraventricular foci
-Seen in the elderly with heart disease and in kids with congenital and acquired heart disease after corrective cardiac surgery

Causes
-SA node tissue becomes replaced with fibrous tissue
-Compromised blood supply to SA node (atherosclerosis, inflammation, emboli)
-Lyme disease
-Drugs causing depressed SA node function: β-blocker, clonidine, methyldopa, digitalis, Li, amiodarone

Signs & Symptoms
-Lightheadedness
-Presyncope or syncope
-DOE
-Worsening angina
-Palpitations

Management
-May eventually need pacemaker

Workup
-Appears as sinus bradycardia
-May also see intermittent SVT → bradycardia-tachycardia syndrome



Ventricular Tachycardia

Torsades de pointes is a polymorphic form of VT

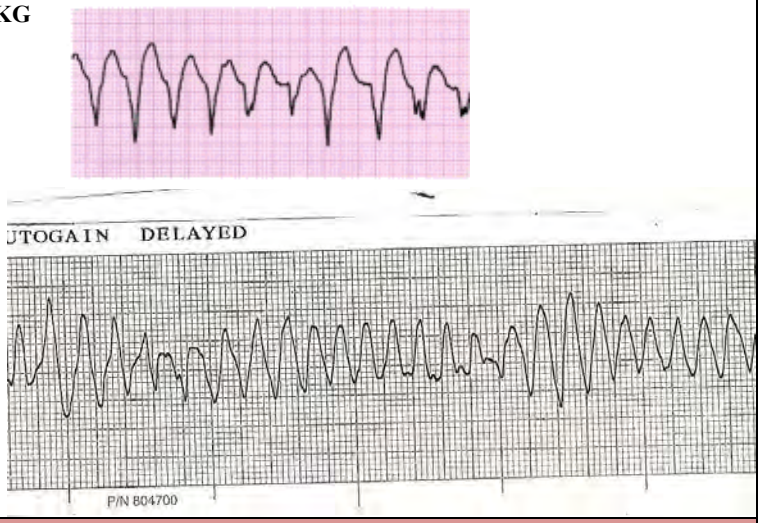
Ventricular flutter is a rapid (240-280) unstable form of VT that can deteriorate to VF

Causes
-Electrolyte imbalances
-Acid/base abnormalities
-Hypoxemia
-MI
-Drugs

Signs & symptoms
-Can remain alert and stable with short runs
-Prolonged runs → hypotension, myocardial ischemia, syncope, chest pain, dyspnea
-Sudden cardiac death

Management
-Torsades: remove offending med, use anti-arrhythmics
-Treat if > 30 s with antiarrhythmics (amiodarone, lidocaine, procainamide)
-Cardioversion if pt remains unstable

EKG

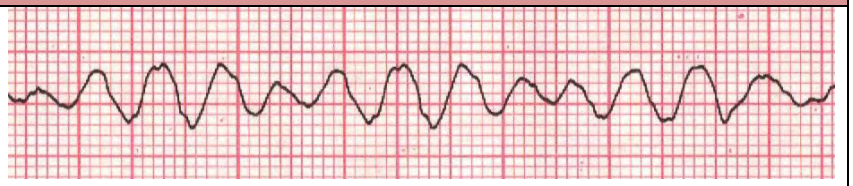


Ventricular Fibrillation

Causes
-Underlying ischemia or LV dysfunction

Signs & Symptoms
-Can remain alert and stable with short runs
-Prolonged runs → hypotension, myocardial ischemia, syncope, chest pain, dyspnea
-Sudden cardiac death

Management
-Treat underlying cause
-Electric defibrillation



Atrial Fibrillation

-Associations: valvular disease, dilated cardiomyopathy, ASD, HTN, CAD, thyrotoxicosis, alcohol excess, pericarditis, chest trauma, OSA, pulmonary disease, stimulants
-2/3 of pts will spontaneously convert within 24 hours of 1st episode

Acute management
-Hemodynamically stable → rate and rhythm control, discuss cardioversion (with warfarin for several weeks before and after if this has been going on > 48 hours)
-Hemodynamically unstable (HR > 150 or symptomatic) → ER for cardioversion

Subsequent management
-Cardioversion w/ antiarrhythmic maintenance vs. rate control w/ long-term anticoagulation
-Rate control with β-blockers, CCB, and/or digoxin
-Assess risk using CHADS2
-Anticoagulation with warfarin to an INR of 2-3 to prevent stroke (unless low risk)
-New drug dabigatran does not require INR monitoring

$CHADS_2$ criteria	Points
Previous stroke or TIA	2
Age ≥ 75 years	1
Hypertension	1
Diabetes mellitus	1
Heart failure	1

Stroke risk score	Recommended therapy
High 2–6	Warfarin (INR 2–3)
Moderate 1	Warfarin or aspirin
Low 0	Aspirin 100–300 mg daily

Atrial Flutter

-Single irritable atrial focus fires rapidly with every 2-3 flutters reaching AV node that is not refractory → QRS production

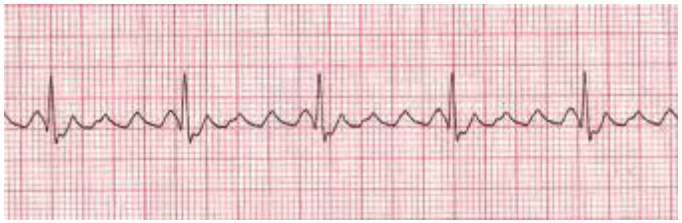
Causes
-Mitral valve disease
-Post cardiac surgery
-Pericardial disease
-Prior heart surgery
-Acute or chronic pulmonary disease

Signs & symptoms
-Palpitations
-Fatigue
-Lightheadedness
-Mild SOB

Workup
-Can use vagal maneuvers to inhibit AV node and get clearer picture of flutters

Management
-Pharmacologic rate control (diltiazem or verapamil): more difficult than in afib
-Cardioversion in pts in whom pharmacologic rate control is ineffective or poorly tolerated or who have hemodynamic instability
-Consider radiofrequency ablation s/p cardioversion to prevent recurrence
-Pts with persistent atrial flutter should be considered for anticoagulation in the same manner as afib

EKG
-Sawtooth flutter pattern
-Atrial rate ~300



Supraventricular Tachycardia (SVT)

Paroxysmal SVT = AV node reentrant tachycardia = SVT with abrupt onset and termination

Causes
-Reentry
-Automaticity

Differential
-Atrial tachycardia: will see spiked P' waves
-Multifocal atrial tachycardia
-Afib or aflutter with rapid ventricular response
-Sinus node reentrant tachycardia
-AV node reentrant tachycardia
-AV reentrant tachycardia
-AV junctional tachycardia (junctional ectopic tachycardia): may have wider QRS due to aberrant ventricular contraction, inverted P waves from retrograde depolarization
-Nonparoxysmal junctional tachycardia
-Ventricular tachycardia: can look similar to SVT
-Sinus tachycardia: will usually be < 150 bpm while SVT is usually > 150 bpm
***Consider dx of Wolf-Parkinson-White syndrome with rapid ventricular response of SVT (accessory Bundle of Kent pathway aids conduction)
→ If you can't distinguish atrial from junctional tachycardia (can't find the P waves), just call it SVT

Signs & symptoms
-Pounding heart
-SOB
-Chest pain
-Dizziness
-Loss of consciousness

Management
-Vagal maneuvers to inhibit AV node
-Chemical cardioversion with adenosine (short AV node block)
-Prevention or rate control with diltiazem, verapamil, or metoprolol (or sotalol or amiodarone if AV node is not involved)
-Synchronized cardioversion
-Radiofrequency ablation for recurrent SVT

EKG
-Rate 150-250
-Regular rhythm

Premature Beats

-Occurs when an irritable focus spontaneously fires
-Some are not serious while some are warning signs

Causes
-Irritable atrial or junctional focus: epi, caffeine, amphetamines, cocaine, ↑ sympathetic stimulation, digitalis toxicity, EtOH, hyperthyroidism, stretch, hypoxia

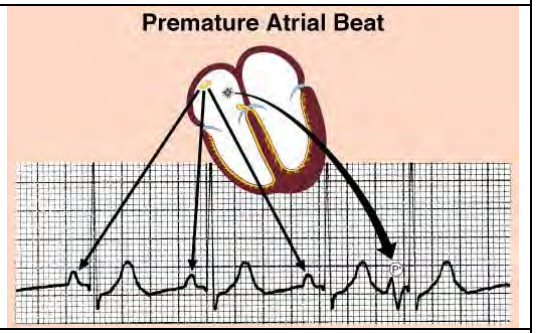
Management
-Treat underlying cause
-May need antiarrhythmics

Premature atrial beat (Premature atrial contraction or PAC)

-P' that is sooner than expected and may be inverted depending on origin
-Wide QRS due to aberrant contraction from semi-refractory bundle branch OR absence of QRS following P' if totally refractory & reset of AV node

Atrial bigeminy = when focus fires prematurely at end of each normal beat

Atrial trigeminy = when focus fires prematurely at end of every 2 normal beats

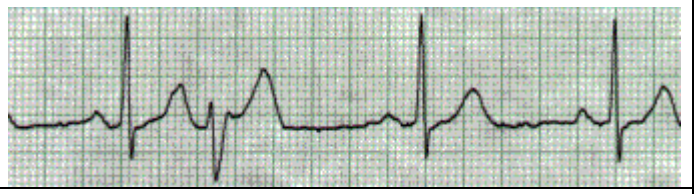


Premature junctional beat

-Premature depolarization of ventricles may retrograde depolarize the atria → inverted P' waves before, after, or within the QRS & reset of SA node
-Wider QRS because ventricular bundle branch may still be refractory

Junctional bigeminy = when focus fires prematurely at end of each normal beat

Junctional trigeminy = when focus fires prematurely at end of every 2 normal beats



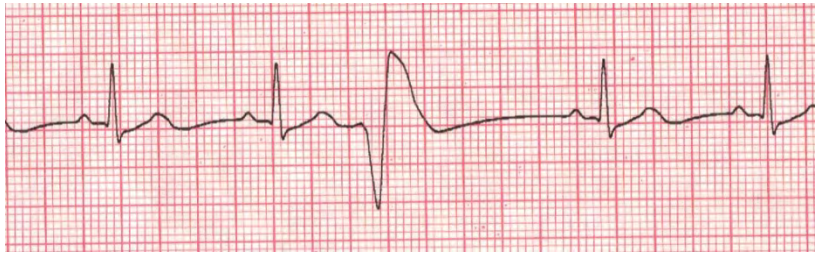
Premature ventricular contraction (PVC)

-Considered pathological if > 6 PVCs/min (esp if they look the same) or if PVCs occur in runs (geminy)
-Early warning sign of hypoxia
-3+ successive PVCs are considered ventricular tachycardia
-Severe hypoxia (MI) can cause multifocal PVCs
-PVC on a T wave is bad ("R on T")
-Differential: ventricular parasystole (ectopic ventricular focus pokes through normal sinus rhythm

-Occurs early in cardiac cycle
-Wide, deep, opposite polarity QRS
-Does not affect SA node

Ventricular bigeminy = when focus fires prematurely at end of each normal beat

Ventricular trigeminy = when focus fires prematurely at end of every 2 normal beats



Ventricular quadrigeminy = when focus fires prematurely at end of every 3 normal beats

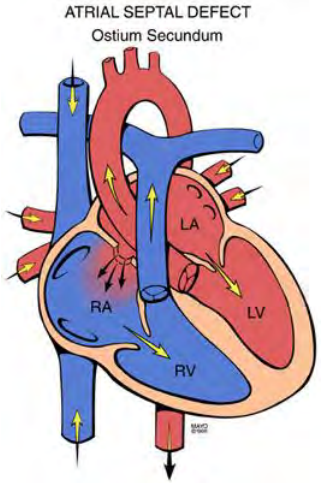
CONGENITAL HEART DISEASE

-Acyanotic = left-to-right
-Cyanotic = right-to-left
-All left-to-right shunts have the potential to revert to right-to-left shunts due to increasing pulmonary congestion (Eisenmenger's syndrome)

Investigation of suspected heart defect
-Most cases are diagnosed prenatally by US screening @ 16-20 weeks
-Some defects don't emerge until several days or weeks have passed since birth due to transition of circulation → adult levels of pulmonary vascular resistance
-Neonate will usually have symptoms within 24 hours

Atrial Septal Defect

-Acyanotic

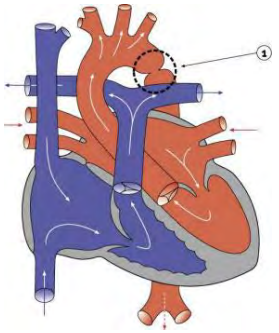


Signs & symptoms
-May be asymptomatic unless there are other defects
-R heart failure
-Pulmonary edema
-Increased pulmonary vasculature
-Midsystolic pulmonary flow or ejection murmur accompanied by a fixed split S2

Management
-Refer to pediatric cards for echo
-Surgical repair at age 2-3 for most
-Small defects in boys don't need closure if RV size is normal

Coarctation of the Aorta

-Obstructive



Signs & symptoms
-Poor perfusion to LEs → diminished femoral pulses, cyanosis, cardiogenic shock, cold extremities, claudication
-Association with Turner's syndrome, Shone's syndrome, and bicuspid aortic valve

Workup
-Measure BPs on all 4 extremities → HTN in UEs with low or unattainable BP in LEs
-Refer for echo

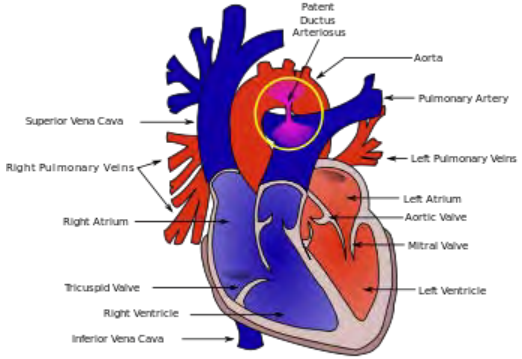
Management
-Reopen truncus arteriosus within 4 days of birth with prostaglandins

Patent Ductus Arteriosus

-Acyanotic

Signs & symptoms
-Harsh continuous machine murmur
-Usually asymptomatic
-May have exertional dyspnea or heart failure

Management
-Refer to pediatric cards for echo and for meds to make ductal tissue regress or surgical repair



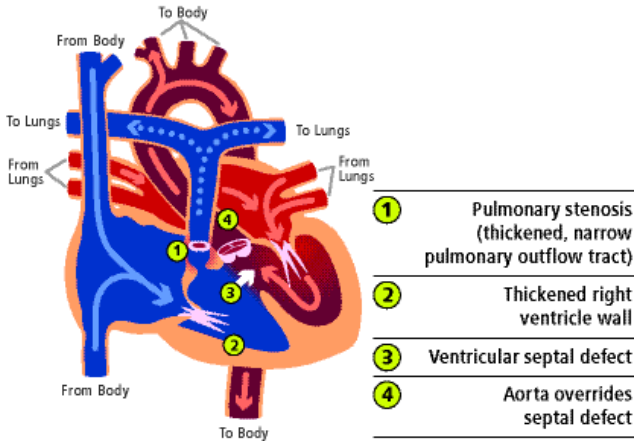
Tetralogy of Fallot

-The most common cyanotic heart defect
-Pulmonary stenosis → RV hypertrophy, overriding aorta, VSD
-VSD may be right-to-left or left-to-right

Signs & symptoms
-Progressive
-May appear healthy and pink at birth
-Cyanotic "tet spells" where child turns blue, squats to valsalva
-Harsh systolic ejection murmur
-May also have right aortic arch, Down's or DeGeorge's syndrome

Management
-Surgical correction in early infancy

Complications
-Brain abscess
-Stroke
-CNS injury

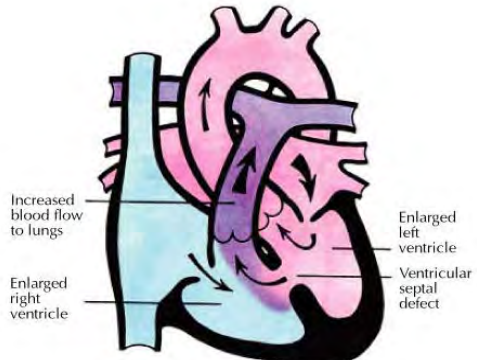


Ventricular Septal Defect

-The most commonly diagnosed congenital heart defect
-May be single or multiple
-May be associated with other lesions

Signs & symptoms
-Holosystolic murmur
-May have thrill or diastolic rumble
-Heart failure
-Down's syndrome association

Management
-Most will get smaller and disappear on their own
-Surgical repair indicated for intractable CHF, failure to thrive

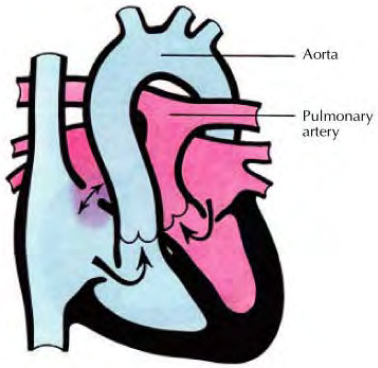


Transposition of the Great Vessels

-Cyanotic
-Aorta and pulmonary trunk are switched so that deoxygenated blood gets pumped through the aorta to systemic circulation while the oxygenated blood gets pumped through the pulmonary artery back through the lungs
-Coexisting left-to-right shunt must also be present for life ex utero

Signs & symptoms
-Severe cyanosis at birth
-Loud S2

Management
-Requires arterial switch for long-term survival



HEART FAILURE

Chronic Congestive Heart Failure

-Most often a result of ischemic heart disease (systolic HF) → myocardial remodeling
-Other causes: bad valves, HTN (diastolic HF), myocarditis, pericarditis, alcoholism (R HF), substance abuse, COPD or other lung disease (R HF)
-Usually associated with low cardiac output but can be high

Acute/flash pulmonary edema with acute MI, severe illness, PE, HTN, end stage valvular disease

Beware **acute HF** with massive MI, tachyarrhythmias, or endocarditis with valve rupture: severe SOB, cool skin, diaphoresis, AMS, pallor, cyanosis

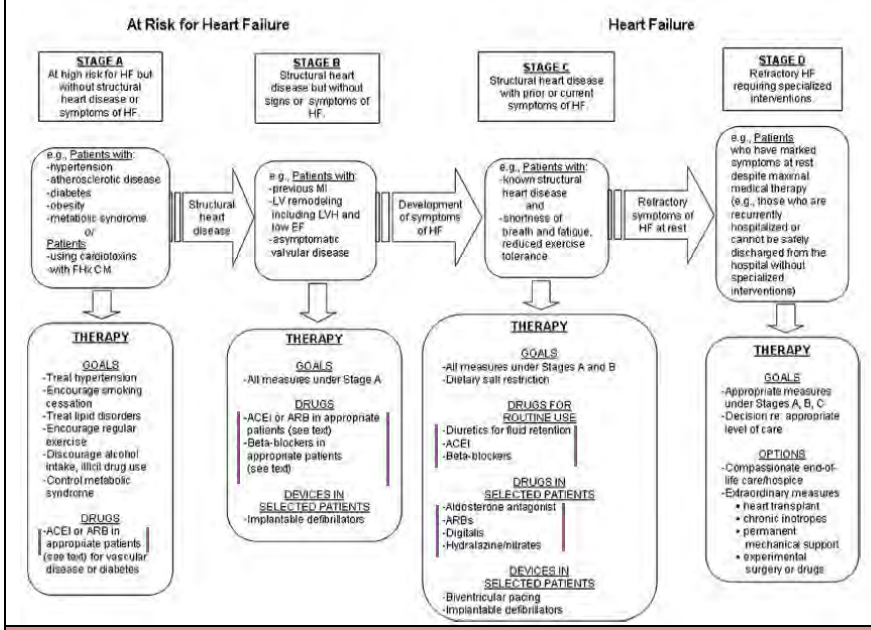
Decompensated HF with new or worsening sx, new murmur, pt is "cold and wet", CHF "decision rule" predicts 30 day mortality, avoid β-blockers

Signs & symptoms
-R CHF → ascites, + hepatojugular reflex, weight gain, JVD, hepatomegaly, edema, abdominal distension, mostly clear lungs with dullness at the bases, ↑ JVP with hepatojugular reflux, tricuspid regurg, peripheral edema
-L CHF (mostly heart and lung sx) → dyspnea, cough, S3 or S4, crackles, wheezes, dullness at bases, frothy or pink sputum, pulse alternans (alternating strong-weak pulse), palpitations, fatigue, diaphoresis, displaced PMI, mitral regurg, pulmonary edema, orthopnea, PND
→ But research shows there are no hard and fast physical exam differentiations for R vs L CHF; all of these s/s can overlap!

Workup
-Referral for echo
-EKG for LVH
-Stress test
-CXR for pulmonary edema (Kerley B lines)
-Labs: BNP, CBC, CMP, fasting glucose, lipids
-Cardiovascular MRI can help distinguish ischemic heart disease from cardiomyopathy

Other Management
-Salt restriction, daily weights
-Avoid NSAIDs, CCBs
-ATP III recommends giving aspirin to reduce prothrombotic state
-Exercise training program for stable NYHA class II to III
-Devices: AICD, intra-aortic pump, LVAD

Class	Description
1	• No limitation of physical activity • Physical activity does not cause fatigue, palpitation or shortness of breath
2	• Slight limitation of physical activity • Comfortable at rest, but physical activity results in fatigue, palpitations or shortness of breath
3-A	• Limitation of physical activity • Comfortable at rest, but ordinary activity causes fatigue, palpitations or shortness of breath
3-B	• Significant limitation of physical activity • Comfortable at rest, but minimal activity causes fatigue, palpitation or shortness of breath
4	• Unable to carry on any physical activity without discomfort • Symptoms of heart failure at rest



Drugs with proven mortality benefit

-Note that there is only evidence for systolic HF for these drugs; use with diastolic CHF is not yet substantiated

-ACEI or ARB

-Hydralazine + isosorbide dinitrate: most beneficial for black man as an add on therapy to those already on β-blocker and ACEI that are still symptomatic

-β-blockers

-Aldosterone antagonists

Prognosis

-Diet and prescription noncompliance are a major cause of hospital readmission for CHF

-Main causes of death are arrhythmia and progressive pump failure

Acute Congestive Heart Failure

-The development of acute dyspnea associated with elevated left sided filling pressures ± pulmonary edema

Differential

-PE

-Pneumonia

-Asthma

Causes

-New onset HF (MI, valvular dz, afib and other arrhythmias, cardiotoxic agents, RV pacing dyssynchrony)

-Exacerbation of chronic CHF (dietary indiscretion, medication nonadherence, iatrogenic volume overload, drug effects)

-Severe HTN

-Renal failure

-Anemia

-Hypo or hyperthyroid

-Infection

-PE

-Uncontrolled DM

Signs & symptoms

-Tachypnea with use of accessory muscles

-Wheezing ("cardiac asthma")

-Tachycardia

-HTN

-Many have new or changed murmur of S3 or S4

-Elevated JVD if R sided filling pressures are ↑

-Peripheral edema with chronic CHF

-Flash pulmonary edema

Workup

-ACS workup: enzymes, EKGs

-BNP (most helpful in comparison to previous)

-CXR

-ABG if respiratory distress present

-BMP

-Echo to assess cardiac fcn

Management

-Treat underlying cause

-Supplemental O2: nonrebreather if necessary

-Diuresis with IV loop diuretic

-Vasodilators

-Bipap if respiratory failure present

-Severe symptoms → IV + inotropes, mechanical cardiac assistance, ultrafiltration

-Hold ACEI and β-blockers until hemodynamically stable or unless acute CHF is mild

-Maintain aldosterone antagonist therapy or begin prior to discharge if not already on

HYPERTENSION

Hypertension

-95% of cases are essential hypertension
-Secondary cause workup: renal disease, renal stenosis, aortic coarctation, hyperaldosteronism from tumor or hyperplasia, Cushing's, pheochromocytoma, OSA
-May hear S4 from forceful atrial contraction

JNC- 7 classifications and initial treatment of HTN
-Pre-HTN is 120/80 to 139/89 → lifestyle changes
-HTN stage I is 140/90 to 159/99 → thiazide (or loop if renal pt)
-HTN stage II is >160/>100 → thiazide + 2nd agent

Hypertensive urgency = stable or no end organ damage, no raised ICP
-May have SOB, headache, BPs usually > 220/110
-Tx is to lower BP in clinic slowly over several hours (≤160/100) with labetalol, clonidine, captopril
-Close follow-up

Hypertensive emergency = rapidly progressing end organ damage

Monitoring HTN:
-Annual urine microalbumin
-Annual BMP
-Annual lipids
-Baseline EKG, look for LVH

Pharmacologic treatment options: single agents only lower by 10-20 mm Hg, may need a 2nd agents	
Thiazides: HCTZ, chlorthalidone	-DOC for HTN but can't use once CrCl < 30 -Ca sparing = good for osteoporosis pts -Need to check BMP before and after starting -Chlorthalidone has the most evidence
Loops: furosemide	
K-sparing: spironolactone, eplerenone	Not very potent
ACEIs: benazepril, enalapril, lisinopril	-Cough -Can cause renal failure = need to monitor BMP 1 week and 1 month after starting and periodically after that, STOP if serum Cr ↑ by 30% -Ok to use in patients with no renal function left -Pregnancy D
ARBs: irbesartan, losartan, olmesartan, valsartan	-Same AEs as ACEIs and also pregnancy D
CCBs: dihydropyridine (nifedipine, amlodipine) and non-dihydropyridine (verapamil and diltiazem)	-Useful in the elderly -FDA warning about amlodipine, verapamil, and diltiazem use with simvastatin -Contraindicated in heart failure
Other direct vasodilators: hydralazine, minoxidil	
α-blockers	-Clonidine: only for refractory HTN due to risk of falls -Methyldopa: DOC for HTN in pregnancy
β-blockers	-Questionable role in the treatment of essential HTN unless patient also has CHF or MI -Need strict β-1 blockers for asthma/COPD patients so that bronchial relaxation is not blocked (atenolol or metoprolol) -Propranolol and labetalol block at multiple sites -Can mask signs of hypoglycemia -Contraindications: heart block

Preferred drug classes for initial treatment of HTN with comorbid conditions

Diabetes	**MI**	**CAD**	**CHF**	**Pregnancy**	**Older patients**	**CKD**	**Recurrent stroke prevention**
Thiazide β-blocker ACEI/ARB CCB	β-blocker ACEI AA	Thiazide β-blocker ACEI CCB	Thiazide β-blocker ACEI/ARB AA	Clonidine Methyldopa	CCB	ACEI/ARB	Thiazide ACEI

Malignant Hypertension (Hypertensive Emergency)

-HTN with signs of acute end-organ damage

Causes
-Longstanding uncontrolled HTN
-Self d/c of HTN meds
-Acute aortic dissection
-Post-CABG
-Acute MI
-Unstable angina
-Eclampsia
-Head trauma or burns

Signs & Symptoms
-BP ~ 220/140
-Retinopathy: blurred vision, retinal hemorrhages or exudates, papilledema
-HA, confusion, n/v, seizures
-Acute CHF
-Renal: AKI, oliguria

Management
-Goal is lower DBP by 10% first hour, then 15% next 3-12 hours (otherwise could cause ischemic stroke or MI)
-IV nitroprusside is the fastest acting
-Labetalol also a good option for most cases
-Underlying CAD → nitroglycerin
-Switch to outpatient oral therapy with goal of lowering DBP to 85-90 over 2-3 months

HYPOTENSION

Hypotension

Causes
-Low stroke vol: dehydration, hemorrhage, vomiting, diarrhea, burns, 3rd spacing, pneumothorax, PE, cardiac tamponade, myocardial ischemia or cardiomyopathy, aortic stenosis or insufficiency, mitral regurg, aortic dissection, ventricular septum or free wall rupture
-Abnormal HR: brady or tachy
-Low systemic vascular resistance: sepsis, adrenal insufficiency, anaphylaxis, vasodilating drugs, neurogenic shock
-Orthostatic/postural: antipsychotics, diuretics, ACEI, vasodilators, methyldopa, polyneuropathy, Parkinson's
-Postprandial: a result of autonomic dysfunction

Signs & Symptoms
-SBP <90 or >30 below baseline
-AMS
-Cyanosis
-Oliguria
-Cool, clammy extremities
-Generalized weakness
-Presyncope or syncope

Workup
-Echo if suspecting cardiogenic shock

Management
-Treat underlying cause
-IVF
-Pressors: dopamine, dobutamine
-Intra-aortic balloon pump

Cardiogenic Shock

Causes	Signs & Symptoms	Workup	Management
-Acute MI -End-stage CHF -Tachyarrhythmia -Acute mitral or aortic regurg -Aortic or mitral stenosis -Traumatic cardiac injury -Myocarditis	-Hypotension -AMS -Cyanosis -Oliguria -Cool, clammy extremities -Elevated JVD -Peripheral edema	-Echo to assess wall motion and function	-Supplemental O2 -Optimize heart rate and rhythm: β-blockers, antiarrhythmics -Optimize volume status: fluids vs diuretics -Reduce afterload with vasodilator -Improve pump function: dobutamine to ↑ CO, consider device like intra-aortic balloon pump or ventricular assist device

CORONARY HEART DISEASE

Coronary Artery Disease

-Risk of developing CAD for 40 year olds in the US is 49% for men and 32% for women
-Risk factors: age, males, FH, sedentary lifestyle, tobacco, HTN, DM, ↑ lipids

Classification
-Class I = no limitations or symptoms with normal activity
-Class II = slight limitations and normal activity results in symptoms
-Class III = marked limitation and minimal activity results in symptoms
-Class IV = symptoms persist with minimal activity and rest

Screening
-Consider stress test in asymptomatic pts with multiple risk factors

Signs & Symptoms
-Angina
-SOB
-Sudden cardiac death is the first symptom in 15%

Chest pain differential
-Atherosclerosis
-Vasospasm from cocaine or stimulants
-Prinzmetal's angina: women under 50
-Coronary artery or aortic dissection
-Congenital abnormality
-Aortic stenosis
-HCM
-Coronary thrombus or embolus
-Non-cardiac: costochondritis (reproducible on palpation), intercostal shingles, cervical or thoracic spine disease (reproducible with specific movements of the head or neck, causes paresthesias), PUD, GERD, cholecystitis, PE, pneumonia, pneumothorax (dyspnea)

Workup
-PE findings: S4, arterial bruits, abnormal funduscopic exam, corneal arcus, xanthelasma, tendinous xanthoma, CHF, murmurs
-EKG
-Refer for stress test if pt has low to intermediate probability of CAD
-Refer for cardiac cath if pt has high probability of CAD

Management of new disease or worsening symptoms
-Referral to cardiology
-ER via ambulance if EKG shows new ischemic changes: ST depression or elevation, inverted T waves or there is hemodynamic instability

Management of stable disease
-LDL goal <100 or <70
-β-blocker (proven mortality benefit), CCB, statin, clopidogrel, nitrates PRN
-New drug ranolazine for refractory chest pain
-PCP visits every 6 months: annual CBC to check for anemia, annual lipids, FBG
-Cardiologist every 1-2 years
-Consider early revascularization for significant narrowing of LAD, left main CAD, LVEF < 30%, or large area of myocardium at risk

Acute Myocardial Infarction

Signs & symptoms
-Sudden onset chest pain, nausea, vomiting, diaphoresis, SOB
-Jaw, neck, scapular, throat, or arm pain
-DOE
-Chest pain > 30 min not responsive to NG
-Hypovolemia
-HTN or hypotension
-Tachy or bradycardia
-S3 or S4
-Signs of CHF
-Systolic murmurs
-Friction rub if day 2 or later
-Change from stable angina to ACS = angina at rest, new onset angina that markedly limits activity, more frequent angina, long duration angina, or angina occurring with less exertion than previous
-Remember that women, the elderly, and diabetics may have atypical presentations

Workup
-Obtain 12 lead within 10 min of arrival and repeat every 10 minutes if initial EKG is not diagnostic (1st EKG is negative 40% of the time)
-Look for early peaked T waves, ST elevation, Q waves, J point elevation
-NSTEMI does not have EKG changes because the infarction is in an electrically silent area
-Presence of LBBB or pacing spikes makes EKG difficult to interpret in the setting of MI!
-Emergent cardiac consult for patients with cardiogenic shock, left heart failure, or sustained ventricular tachyarrhythmia
-Electrolytes, coagulation studies, H/H
-Serial troponins: specific cardiac damage marker, including damage from defibrillation, arrhythmias, cardiac procedures, CHF, vasospasms, PE, myocarditis; elevation begins within 1 hour and remains ↑ for 5-14 days
-CK: found in skeletal muscle throughout the body; shows up in 1-6 hours and lasts up to 1.5 days
-CK-MB: cardiac specific CK; shows up in 2 hours and declines after 24-72 hours
-Coronary angiography to determine location of lesion

Emergent Management (any ACS)
-Oxygen: only if sats < 90% or resp distress)
-Aspirin + NG
-Morphine for continued chest pain despite NG
-Treat HF if present with NG, furosemide
-Give β-blocker if HF is not present in order to reduce myocardial oxygen demand
-Begin 80 mg atorvastatin for pts not already on
-Echo to determine cardiac function

Additional STEMI Treatment
-Antiplatelet and anticoagulant therapy for all patients (in addition to aspirin)
-Emergent stent if < 3 hours from symptom onset
-Alternative is lytic therapy if not contraindicated, symptoms < 12 hours, and PCI unavailable within 90-120 minutes
-CABG rarely performed during acute MI

Additional NSTEMI Treatment
-Antiplatelet therapy for all patients (in addition to aspirin; clopidogrel, ticagrelor, etc.)
-Anticoagulant therapy for all patients (heparin)
-Invasive intervention based on presence of high risk factors (recurrent angina at rest, elevated troponin, ST depression, high risk stress test result, EF < 40%, hemodynamic instability, sustained VT, recent PCI, prior CABG, TIMI score)
-Glycoprotein IIA/IIIB inhibitor in addition to all other meds for a subset of select pts who will undergo early PCI

Treatment of Cocaine-Related ACS
-Benzos every 15 minutes PRN
-DON'T give β-blockers

Post-ACS Treatment
-Continue drugs used during hospitalization: β-blocker, statin, ASA, SL nitrates PRN (and possibly PO as well), aldosterone antagonist & ACEI (if DM, HF, LV EF < 40%, or HTN), clopidogrel if intervention was done
-Pre- or postdischarge stress test (depending on whether or not intervention was done)

Prognosis
-33% are fatal, with most deaths caused by v-fib
-Complications: CHF, RV infarction, ventricular rupture, arrhythmias, mural embolus, stroke, pericarditis, postinfarction angina
-For USA and NSTEMIs, can estimate 14-day risk of death, recurrent MI, or need for urgent revascularization using TIMI score (Skyscape)

TIMI RISK SCORE for UA/NSTEMI

HISTORICAL	POINTS
Age ≥ 65	1
≥ 3 CAD risk factors (FHx, HTN, ↑ chol, DM, active smoker)	1
Known CAD (stenosis ≥ 50%)	1
ASA use in past 7 days	1
PRESENTATION	
Recent (≤24H) severe angina	1
↑ cardiac markers	1
ST deviation ≥ 0.5 mm	1
RISK SCORE = Total Points (0 - 7)	

RISK SCORE	RISK OF CARDIAC EVENTS (%) BY 14 DAYS IN TIMI 11B* DEATH OR MI	DEATH, MI OR URGENT REVASC
0/1	3	5
2	3	8
3	5	13
4	7	20
5	12	26
6/7	19	41

*Entry criteria UA or NSTEMI defined as ischemic pain at rest within past 24H, with evidence of CAD (ST segment deviation or +marker)

For more info go to www.timi.org

Antman et al JAMA 2000; 284: 835 - 842

Angina Pectoris

Differential

-AMI
-Syndrome X: myocardial ischemia in the setting of normal coronary arteries; due to disease of coronary microvasculature
-Prinzmetal's angina: caused by coronary vasospasm; usually at rest; typically in younger women; pain relieved by nitrates; dx via angiography with injection of provocative agents
-Pericarditis
-Aortic dissection
-PE
-Tension pneumothorax
-Esophageal rupture
-Pneumonia
-Pleuritis
-Bronchitis
-GERD
-PUD
-Biliary disease
-Pancreatitis
-Cervical or thoracic disk disease, thoracic outlet syndrome
-Costochondritis
-Anxiety or panic attack
-Shingles

Stable Angina	Unstable Angina
Signs & symptoms -Gradual onset chest pain due to myocardial ischemia that occurs predictably and reproducibly on exertion -May also have SOB -Relieved by rest or NG -Usually lasts 2-5 minutes -Diffuse discomfort **Workup** -Stress test -Coronary angiography if needed **Management** -Goal is to relieve symptoms and prevent future cardiac events -Nitrates and β-blockers are initial DOCs -CCB for refractory symptoms -Exercise -Daily aspirin -CV risk reduction: BP control, smoking cessation, statins, weight reduction, glycemic control -Periodic reevaluation with EKGs -Revascularization therapy an option for select candidates	**Signs & symptoms** -Chest pain refractory to NG treatment or chest pain at rest or nocturnally -May be associated with SOB, nausea, diaphoresis **Management** -Treat like other ACS: admission, MOANS, serial troponins, EKGs -Stabilize -Antiplatelet therapy and possible reperfusion for select patients - β-blockers -Statins -ACEI with DM, HF, LV EF < 40%, or HTN -CV risk reduction

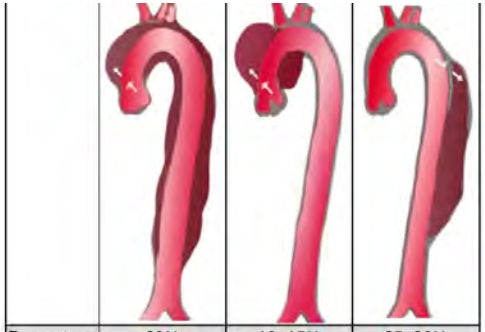
VASCULAR DISEASE

Aortic Dissection

-Occurs when tear in the inner wall of the aorta causes blood to flow between the wall layers → creation of false lumen
-Acute or chronic
-Usually in ascending aorta
-Typically in men 60-70
-Debakey and Stanford classifications

Risk Factors
-Connective tissue disorders
-Bicuspid aortic valve
-Coarctation of the aorta
-HTN

Causes
-Usually a result of HTN
-Increased risk in pregnancy, connective tissue disease, bicuspid aortic valve, aortic coarctation



Percentage	60%	10–15%	25–30%
Type	DeBakey I	DeBakey II	DeBakey III
	Stanford A (Proximal)		Stanford B (Distal)

Signs & Symptoms
-Sudden onset of "ripping" retrosternal and back pain
-HTN
-Hypovolemia
-Syncope
-Shock
-Pulse discrepancies
-Cardiac tamponade
-May have focal neuro deficits or CVA due to poor perfusion of the brain

Workup
-EKG may show infarct pattern or LVH
-CXR will show widened mediastinum, L sided pleural effusion
-Bedside TEE test of choice, CT if unavailable

Management
-Achieve hypotension and bradycardia with β-blocker and nitroprusside
-Surgical repair for Stanford type A
-Stanford type B admitted to ICU for medical management: morphine for pain control, β-blockers and nitroprusside

Prognosis
-Greater than 20% intra-op mortality
-50% mortality within 10 years of all hospital survivors

VASCULAR DISEASE

Aortic Aneurysm

-Occurs when blood collects between the aortic vessel layers, with true aneurysms involving all 3 layers (intima, media, adventitia)
-Most commonly occurs below the kidney

Risk Factors
-Smoking
-HTN
-Hyperlipidemia
-Male
-Atherosclerosis
-FH

Differential
-Pseudoaneurysm: a collection of blood and connective tissue located outside of the vessel wall

Prognosis
-Can spontaneously rupture
-Post-op complications: MI, reduced blood flow to LEs from emboli, AKI, mesenteric or spinal cord ischemia, device migration or endoleak with graft placement

Abdominal Aortic Aneurysm

-Normally AA is 2 cm, becomes aneurysmal when > 3 cm
-Caused by atherosclerosis and inflammation
-Categorized based on morphology
-Usually infrarenal in location

Screening
-USPSTF recommends US screen in all men 65-75 who have ever smoked
-May also want to screen women with cardio risk factors and anyone > 50 with a FH

Signs & Symptoms
-Usually asymptomatic and discovered incidentally on abdominal exam
-Abdominal or back pain
-May have signs of limb ischemia

Workup
-Abdominal US for diagnosis
-Abdominal CT for further characterization and measurement

Management
-Surgical repair indicated when > 5 cm; may be endovascular (stent) or open graft repair; endovascular has lower short-term mortality/morbidity but open repairs have better long-term outcome
-Watchful reimaging and risk reduction if < 5 cm: smoking cessation
-Consider elective nonrepair and cessation of surveillance imaging if life expectancy is < 2 years

Thoracic Aortic Aneurysm

-Further classified as ascending, descending, or arch
-Ascending thoracic AA usually due to elastin degradation, which can be normal aging or accelerated by HTN, connective tissue disorder, RA, or bicuspid aortic valve
-Descending thoracic aneurysm is caused by atherosclerosis
-Arch aneurysm seen in trauma or deceleration injuries

Signs & Symptoms
-Aortic insufficiency symptoms from dilation of valve
-CHF
-Compression of SVC by enlarging aorta → SVC syndrome
-Tracheal deviation
-Cough
-Hemoptysis
-Dysphagia
-Hoarseness
-Steady, deep, severe substernal, back, or neck pain

Workup
-CXR for widened mediastinum, enlarged aortic knob, tracheal displacement
-MRI or CTA are test of choice for characterization and dx
-Echo

Management
-BP control, β-blockers preferred
-Re-image with CT or MRI every 6 mos
-Surgical management is risky and complicated = rarely done, need to weigh risk of rupture
-Surgical repair indicated for thoracic AA ≥ 6 cm, rapid expansion of aneurysm, or symptomatic aneurysm

Prognosis
-Less likely to spontaneously rupture than AAA

Arterial Embolism (Acute Arterial Occlusion)

Causes
-Embolism or thrombus lodges or forms in a usually already-diseased vessel

Risk factors
-Afib
-Valvular disease
-Prosthetic device
-Ischemic disease
-Trauma
-Hypercoagulable disorder

Signs & symptoms
-5 P's of critical limb ischemia: pain, pallor, pulselessness, poikilothermia (cold), paresthesias/paralysis

Workup

	Viable	Threatened	Nonviable
Pain	Mild	Severe	Variable
Capillary refill	Intact	Delayed	Absent
Motor deficit	None	Partial	Complete
Sensory deficit	None	Partial	Complete
Arterial Doppler	Audible	Inaudible	Inaudible
Venous Doppler	Audible	Audible	Inaudible
Treatment	Urgent work-up	Emergency surgery	Amputation

Management
-Heparin drip
-Thrombolytics
-Surgical emergency, requires correction via thrombectomy or bypass within 6 hours of symptom onset or amputation will be necessary

Prognosis
-With intervention limb loss approaches 30% and overall mortality of 20% due to underlying cardiopulmonary disease

Giant Cell Arteritis (Temporal Arteritis)

-Rheumatic disease, most often affects med-large head & neck vessels
-Usually in white patients over 50
-Often co-exists with polymyalgia rheumatica

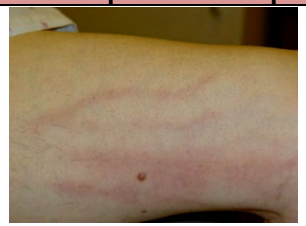
Signs & symptoms
-Weight loss
-Night sweats
-Fever
-Jaw claudication
-Temple tenderness
-Vision loss with pale optic disc
-New headache
-Scalp tenderness

Workup
-Arterial biopsy
-↑ ESR or CRP

Management
-Immediate steroids while awaiting biopsy results in order to prevent blindness
-Monitor for thoracic aortic aneurysm (increased risk)

Thrombophlebitis of Superficial Veins

-Usually along a recent IV or PICC site (commonly the saphenous)
-May be spontaneous in pregnancy or postpartum or with varicose veins or trauma
-Can be associated with malignancy
-20% of cases will also have DVT
-May not see significant swelling
-Linear rather than circular



Disposition
-If any fever or chills, progressive→ ER
-Stable and localized → heat, NSAIDs

Prognosis
-Inflammatory rxn subsides in 1-2 weeks
-20% mortality if septic

Deep Venous Thrombosis

Signs & Symptoms
-Palpable cord
-Calf pain
-Ipsilateral edema, warmth, tenderness, erythema

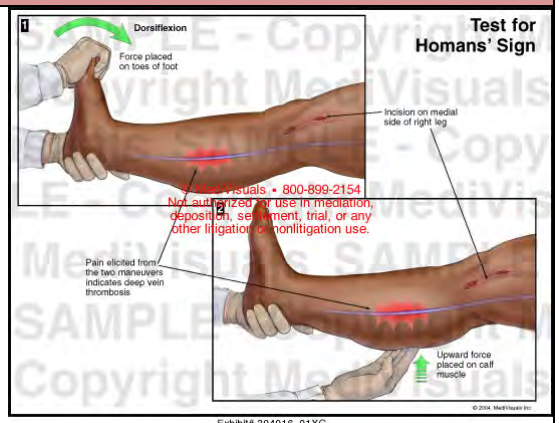
Workup
-Homan's is only + 50% of the time
-Determine probability with Well's criteria → < 2 indicates unlikely, > 6 highly likely
-Further investigation using D-dimer
-US for at least moderate Well's score

Management
-Immediate anticoagulation with heparin, LMWH, or fondaparinux
-Lytics or thrombectomy for select cases
-3 months of anticoagulation for initial distal DVT or consider IVC filter if not a good candidate

Table 5. Wells et al Clinical Model For Predicting Pretest Probability For DVT[24]

Clinical Characteristic	Score
Active cancer (patient receiving treatment for cancer within previous the 6 months or currently receiving palliative treatment)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recent bedridden for greater than 3 days or major surgery within the previous 12 weeks requiring general or regional anesthesia	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling at least 3 cm larger than that on the asymptomatic leg (measured 10 cm below tibial tuberosity)	1
Pitting edema confined to the symptomatic leg	1
Collateral superficial veins (nonvaricose)	1
Previously documented deep-vein thrombosis	1
Alternative diagnosis at least as likely as deep-vein thrombosis	-2

A total score of two or higher indicates that the probability of deep-vein thrombosis is likely; a total score of less than two indicates that the probability of deep-vein thrombosis is unlikely. In patients with symptoms in both legs, use the more symptomatic leg.



Peripheral Vascular Disease

-Risk factors: smoker, DM, HTN, ↑ lipids, obesity

Signs & symptoms
-LE disease: claudication (ppt by exercise, relieved by rest) in butt, hip, thigh, calf, or foot; diminished peripheral pulses; femoral bruits; nighttime pain from ischemia
-UE disease: difference in systolic BPs between aroms, arm pain with exertion, dizziness during arm exertion (subclavian steal syndrome)
-Cool skin or abnormal skin color
-Poor hair growth
-Ulceration or tissue necrosis
-Signs of atherosclerosis elsewhere in the body

Workup
-Ankle/brachial index: PVD if <0.9, will have intermittent claudication if <0.7, pain at risk if <0.4, impending necrosis if <0.1

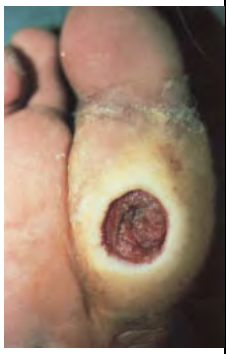
Management
-Smoking cessation
-Walking program
-Antiplatelet therapy
-Revascularization if necessary via open surgery or stent
-BP, lipid, sugar control

Leg Ulcers

Risk factors
-Poor circulation, venous insufficiency, disorders of clotting, diabetes, sickle cell, neuropathy, renal failure, HTN, lymphedema, inflammatory skin diseases, smoking, genetics, malignancy, meds

Diabetic (Neurotrophic) Ulcers

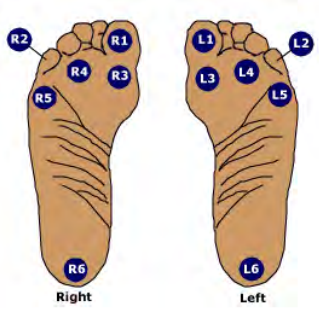
Screening
-Recommended annually with visual examination and monofilament test (checks most common sites of ulceration)



Signs & symptoms
-Ulcers with punched-out borders with calloused surrounding skin
-Underlying neuropathy

Workup
-ABIs to r/o PAD

Management
-Comprehensive assessment of ulcer and patient's overall medical condition
-Classification of wound at each follow-up
-Debridement, local wound care, pressure relief, infection control, and proper dressing selection
-Negative pressure wound therapy following debridement after infection, necrosis, or amputation
-Revascularization for critical wound ischemia

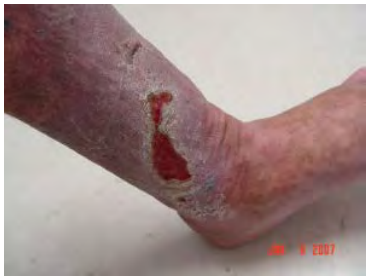


Chronic Venous Insufficiency Ulcers

-Classified by CEAP system, which helps distinguish initial disease severity as well as changes over time
-Varicose veins in the absence of skin changes are NOT chronic venous insufficiency!

Risk factors
-Advancing age, FH, increased BMI, smoking, h/o LE trauma, prior DVT, pregnancy

Signs & symptoms
-C/o tired, heavy legs, leg pain, or leg swelling
-Telangiectasias, reticular veins, and varicose veins
-Edema, inflammation, pruritic dermatitis
-Ulcers with irregularly shaped borders along the medial ankle or saphenous veins that are tender, shallow, exudative, and have a base of granulation tissue
-Skin discoloration or redness, may appear shiny or tight
-20% of symptomatic patients will have no visible clinical signs

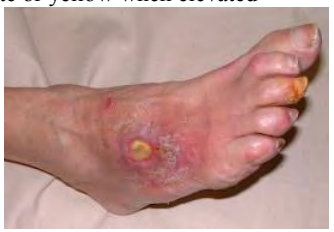


Workup
-ABIs to r/o PAD
-Duplex US to eval degree of venous obstruction or reflux

Management
-Leg elevation, exercises, and graduated compression stockings
-SCDs for patients refractory to stockings
-Horse chestnut seed extract for patients who can't tolerate or are noncompliant with compression therapy
-Aspirin therapy to accelerate healing of ulcers
-Invasive options: sclerotherapy, laster therapy, endovenous ablation techniques, vein stripping
-Skin moisturizers
-Wound debridement PRN
-Barrier creams to protect adjacent skin
-Selection of proper wound dressing
-NOT effective: topical antibiotics, debriding enzymes, growth factors, or honey
-Compression bandages for severe edema, weeping, eczema, or ulceration
-Aspirin therapy accelerates healing
-Referral to subspecialty for slowly healing ulcers, persistent dermatitis, or recurrent cellulitis

Arterial Insufficiency (Ischemic) Ulcers

Signs and symptoms
-PAD symptoms: pain and claudication with walking that is relieved by rest (however may have more pain with leg elevation in severe disease)
-Ulcers are usually on the feet at points of friction and appear punched-out
-Feet will turn red when dangled and pale white or yellow when elevated



Varicose Veins

-Usually occur in the saphenous veins

Causes
-Incompetent valves from damage or venous dilation
-AV fistula
-Congenital venous malformations

Signs & Symptoms
-Dull or aching pain in legs that is worse after standing
-Pruritus
-May have h/o DVT
-Brownish thinning of the skin above the ankles

Differential
-Claudication
-Superficial thrombophlebitis
-Arthritis
-Peripheral neuropathy

Management
-Compression stockings
-Leg elevation
-Venous ablation
-Sclerotherapy
-Great saphenous vein stripping

Prognosis
-Complication of thrombophlebitis

VALVULAR DISEASE

***Stenosis causes HYPERTROPHY while regurgitation causes DILATION

Aortic Stenosis (Aortic Valve Stenosis)

-Causes obstruction → ↑ LV pressure → LVH → CHF
-Normal valve is 3-4 cm^2

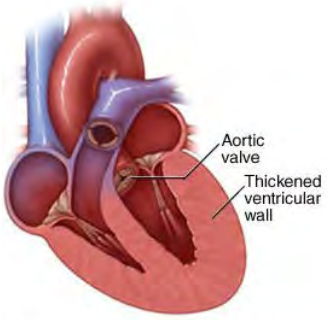
Etiologies
-Age-related calcification (inflammation + lipids)
-Bicuspid aortic valve
-Rheumatic fever

Signs & symptoms
-May have asymptomatic murmur early in disease: harsh systolic < > murmur at RUSB, with radiation to the carotids bilaterally
-Late: DOE, SOB, angina, syncope, CHF, PND, orthopnea, carotid pulsus parvus et tardus
-Sudden cardiac death
-Arrhythmias
-Endocarditis
-Bleeding predisposition

Workup
-EKG for LVH
-CXR for cardiomegaly
-Echo is diagnostic

Management
-Proven benefit with statins
-Valve replacement if symptomatic
-Aortic balloon valvotomy as bridge to surgery or for palliation

Prognosis
-High risk of sudden death without valve replacement (life expectancy of 2-3 years)



Heart with Aortic Valve Stenosis

Aortic Regurgitation (Aortic Insufficiency)

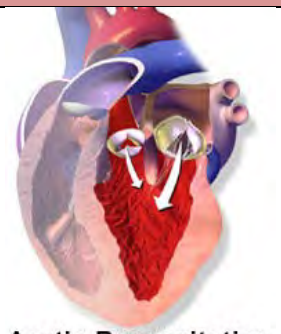
-Causes increased afterload in LV → LV dilation → increased end-diastolic pressure in LV → backup into pulmonary circulation

Causes
-Congenital: bicuspid or unicuspid aortic valve
-Infectious: rheumatic fever or infective endocarditis
-Inflammatory: SLE or RA
-Aortic root disease: Marfan, syphilis, ankylosing spondylitis, aortic dissection, trauma

Signs & Symptoms
-Acute: flash pulmonary edema
-Chronic: left-sided CHF, increased pulse pressure
-Classic murmur is soft blowing < diastolic murmur
-May have S3 gallop
-Austin Flint murmur: mid-diastolic, low frequency murmur at the apex from regurgitant flow competing with inflow from left atrium
-DeMusset sign: head bob with each heartbeat
-Water hammer pulse (Corrigan pulse): radial and carotid pulses are abrupt and distensive with fast collapse
-Traube sign (pistol shot femoral): booming S1 and S2 over femoral artery
-Muller sign: systolic pulsations of the uvula

Workup
-EKG for LVH
-CXR for cardiomegaly
-Echo is diagnostic

Management
-Acute: nitroprusside and emergent aortic valve replacement
-Vasodilators (ACEI or ARB or hydralazine + nitrates) to reduce afterload
-Endocarditis prophylaxis in certain patients
-Aortic valve replacement if having symptoms of CHF, hemodynamic compromise, or EF < 55%


Aortic Regurgitation

Mitral Stenosis

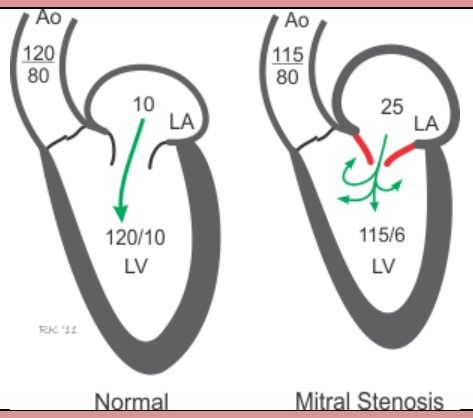
-Causes elevated LA pressure → LA hypertrophy → transmission of high pressures to pulmonary vasculature → pulmonary edema → possible R-sided CHF

Etiologies
-Most commonly due to rheumatic heart disease
-Congenital malformation
-Connective tissue disease

Signs & symptoms
-Dyspnea is commonly the only symptom
-A-fib from disruption of electrical conduction in hypertrophied tissue
-Fatigue
-Apex murmur: loud S1 with post S2 opening snap, followed by low-pitched rumble
-RV heave with progression to pulmonary HTN
-Pregnancy may bring on symptoms

Workup
-Echo to stage

Management
-If asymptomatic, only watchful waiting
-HTN management
-Afib management
-β-blockers to prevent pulmonary edema
-Anticoagulation for any embolic event
-Follow with echoes every 1-5 years depending on stage
-Mitral valve replacement or balloon valvuloplasty



Mitral Regurgitation (Mitral Insufficiency)

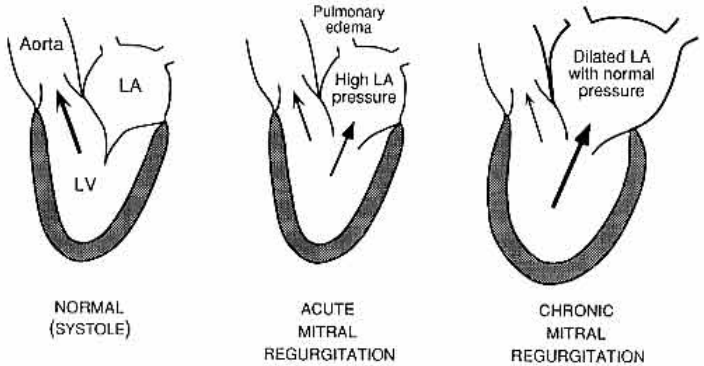
-Causes increased afterload in LA → dilation of LA to accommodate → increased end-diastolic pressure in LA → backup into pulmonary circulation

Causes
-Weakening of connective tissue
-Mitral valve prolapse
-Ischemic LV function post MI
-Dilated cardiomyopathy
-Rheumatic fever
-Papillary muscle dysfunction or rupture
-Mitral annulus calcification
-Bacterial endocarditis

Signs & symptoms
-Acute: flash pulmonary edema, cardiogenic shock
-Chronic: progressive L-sided CHF, afib
-Holosystolic murmur at apex with radiation to axilla with severity correlated to duration rather than intensity (↑ with valsalva, ↓ with squatting)
-↑ JVD
-Laterally displaced apical impulse

Workup
-EKG for LVH
-Echo
-Cardiac cath to grade severity

Management
-ACEI to reduce afterload
-Diuretics
-Digoxin
-Endocarditis prophylaxis
-Surgical repair if acute



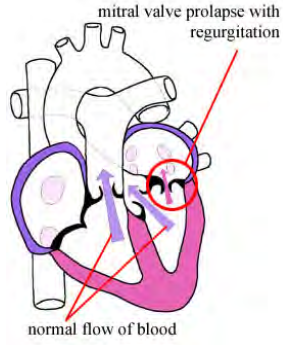
Mitral Valve Prolapse

-Displacement of an abnormally thickened mitral valve leaflet into the LA during systole → elongated chordae tendinae and mitral regurg
-More common in women

Risk factors
-Collagen vascular disease: lupus, RA, ankylosing spondylitis, Ehlers-Danlos, Marfan's

Signs & symptoms
-Usual presents in early adulthood
-May be asymptomatic or have fatigue, atypical chest pain, palpitations, anxiety disorder, postural orthostasis
-Rarely progresses to mitral regurg
-Mid-systolic click

Management
-Initial → refer to cardiology
-β-blockers for palpitations
-Aspirin for clot risk
-Endocarditis prophylaxis no longer recommended
-Periodic echos if there is progression to mitral regurg
-Valve repair vs. replacement if severe



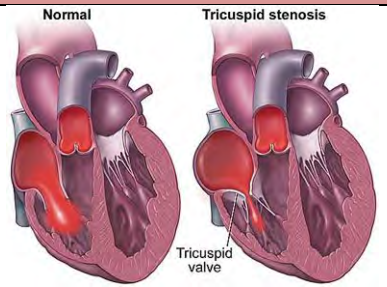
Tricuspid Stenosis

-Results in elevated RA pressures → edema, hepatosplenomegaly, ascites

Causes
-Usually due to rheumatic disease

Signs & Symptoms
-Fatigue and weakness
-Soft, high-pitched diastolic murmur that is ↑ with inspiration
-Visible hepatojugular reflux
-Rarely an isolated disease, usually will see signs of mitral or aortic defects

Management
-Balloon valvuloplasty or valve replacement if symptomatic



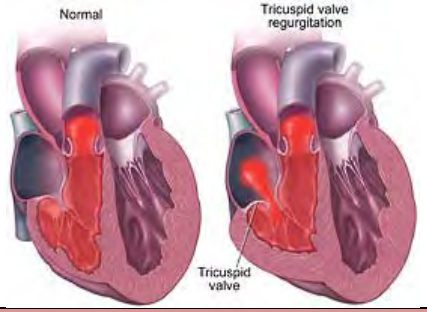
Tricuspid Regurgitation (Tricuspid Insufficiency)

-Results in leakage into the RA during systole
-Can be normal if present in small amounts

Causes
-Ebstein's anomaly (displacement of valve towards apex)
-Rheumatic disease
-Carcinoid
-Endocarditis

Signs & Symptoms
-Symptoms of RV failure: anasarca, ↑ JVD with hepatojugular reflux, pulsatile liver
-Holosystolic murmur that is ↑ with inspiration
-Afib

Management
-Treated only if severe
-Diuretics for R-sided CHF
-Digoxin for arrhythmias
-Treat pulmonary HTN
-Surgical repair better outcome than replacement



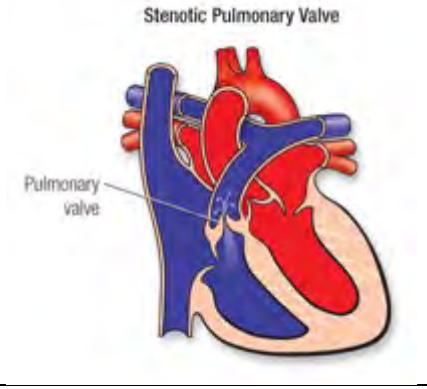
Pulmonary Stenosis (Pulmonic Valve Stenosis)

Causes
-Most commonly congenital
-Rheumatic fever
-Complication of arrhythmia ablation procedure

Signs & Symptoms
-DOE
-Fatigue
-Presyncope
-Cyanosis
-↑ JVP
-Split S2 with soft P2
-Ejection click followed by < > systolic murmur
-May hear S4

Workup
-Echo to look for doming of pulmonic valve during systole and grade severity

Management
-Based on Doppler gradient obtained during echo
-Balloon valvulotomy



Pulmonary Regurgitation (Pulmonic Valve Regurgitation)

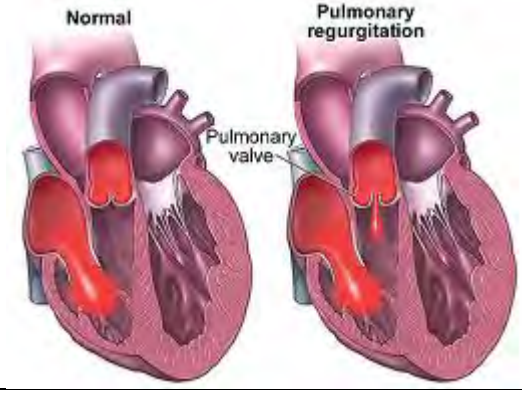
-Results in backward flow of blood into the RV during diastole

Causes
-Dilated annulus from pulmonary HTN
-Connective tissue disorder
-Infection endocarditis
-Surgical complication
-Congenital malformation
-Syphilis
-Carcinoid
-Rheumatic fever

Signs & Symptoms
-Can be tolerated asymptomatically for many years if it is the only defect
-Palpable RV heave
-Low-pitched < > diastolic murmur
-RV failure symptoms if pulmonary HTN present

Workup
-Echo

Management
-Valve replacement



OTHER HEART DISEASES

Bacterial Endocarditis

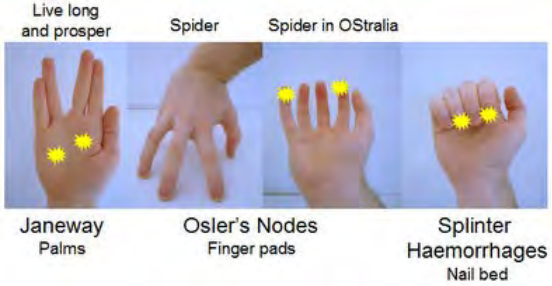
-Mostly affects the elderly

Agents
-*Staph aureus* is most common
-Viridans strep
-Enterococci

Risk factors: IVDU, prosthetic heart valve, structural heart disease, prior endocarditis

Prevention
-New guidelines from AHA suggest only prophylaxing the highest risk groups prior to procedures likely to result in bacteremia: prosthetic heart valves, h/o endocarditis, unrepaired cyanotic congenital defect, repaired congenital defect with prosthetic material, cardiac valvulopathy in a transplanted heart
-Usual AB is amoxicillin 2 g 30-60 min prior to procedure

Signs & symptoms
-New regurgitant murmur
-HF
-Evidence of embolic events
-Peripheral lesions (petechiae, splinter hemorrhages, Roth spots, etc)
-Fever



Workup
-Blood cultures x 3
-EKG
-Echo
→ Use Duke criteria to determine probability

Management
-Prosthetic valves may need to be replaced
-Empiric therapy initiated with IV vanco, gentamicin, and either cefepime or a carbapenem; subsequent AB therapy targeted to culture results
-Treat for at least 6 weeks

Prognosis
-Complications are common: heart failure, stroke and other embolic events, perivalvular abscess, pericarditis, fistulas, aortic valve dissection, meningitis or encephalitis
-In-hospital mortality rate of 18-23%
-6 month mortality rate of 22-27%

Acute Pericarditis

Causes
-Viral: Coxsackie virus B, hep B, CMV
-Bacterial: *Staph, Strep,* TB
-Post-MI
-Drugs (procainamide)
-Malignancy mets
-SLE or other collagen vascular disease

Signs & Symptoms
-Chest pain that is worse with deep breathing, cough, or lying down
-Pericardial friction rub
-Cardiac tamponade: hypotension, tachycardia, DOE, distended neck veins, narrow pulse pressure, pulsus paradoxus, indistinct heart sounds

Workup
-EKG shows diffuse ST elevation without reciprocal lead depression
-Cardiac enzymes are –
-Echo shows pericardial effusion

Management
-Treat underlying cause
-NSAIDs, may need steroids
-Pericardiocentesis if tamponade or large effusion present

Sequelae
-May develop constrictive pericarditis, a thickening and fibrosis of the pericardium
-May develop cardiac tamponade from accumulation of effusion fluid

Cardiac Tamponade

Causes	Signs & Symptoms	Management
-Pericarditis -Trauma -Post-heart surgery -Myocardial rupture -Hypothyroidism	-Hypotension, tachycardia, DOE, distended neck veins, narrow pulse pressure, pulsus paradoxus, indistinct heart sounds	-Pericardiocentesis

PULMONARY SYSTEM

INFECTIOUS DISORDERS

Acute Bronchitis

Acute bronchitis = less than 3 weeks -Almost always VIRAL! even if sputum is purulent and you hear wheezes and rhonchi -Only consider bacterial if pt does not get better	**Treatment is supportive** -Fever → antipyretics -Wheezing or cough with activity → short-acting bronchodilator (albuterol) -Antitussives → dextromethorphan has the best evidence (Robitussin, Vick's DayQuil Cough, codeine/guaifenesin (Cheratussin AC – may need Rx in NC), benzonatate (Tessalon – Rx required, comes in capsules), chlorpheniramine/hydrocodone (Tussionex – may need Rx in NC) -Peds over 2 → children's Robitussin or Triaminic Long-Acting **Post-tussive emesis** → concern for pertussis → give azithromycin if high suspicion and isolate for 5 days	**Differential** -Asthma -Allergic rhinitis -Pneumonia -Pertussis -CHF -GERD -Meds -Environmental exposure
Chronic bronchitis = more than 3 months each year for at least 2 years, all other causes excluded -Think smokers, COPD	**Treat as exacerbation** if there is a change in sputum color or amount from baseline, deterioration in respiratory function or increased dyspnea -Antibiotics: Augmentin, cephalosporins, macrolides, respiratory FQ (not cipro, *S. pneumo* resistance)	

Acute Bronchiolitis

-Research definition = first episode of wheezing in a child younger than 12 to 24 months who has physical findings of a viral respiratory infection and has no other explanation for the wheezing, such as pneumonia or atopy -Broader definition = an illness in children <2 years of age characterized by wheezing and airway obstruction due to primary infection or reinfection with a viral or bacterial pathogen, resulting in inflammation of the small airways/bronchioles -Mostly in infants < 2 months -Prophylaxis with Synagis given to high risk infants during first RSV season	**Agents** -Usually RSV -Rhinovirus -Human metapneumovirus -Influenza -Parainfluenza -Adenovirus **Signs & symptoms** -Concomitant URI -Conjunctivitis or OM -Wheezing, tachypnea, retractions, crackles	**Differential** -Asthma -Foreign body **Workup** -Diagnosis is usually clinical -CXR showing hyperinflation, interstitial pneumonitis, infiltrates -ELISA for RSV available **Management** -Supportive -Humidifier -Oxygen if needed for severe disease -Bronchodilators or steroids for select patients

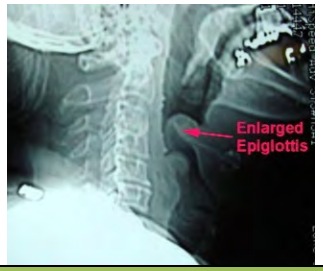
Acute Epiglottitis

Agents
-*H. flu*
-*Strep pneumo or Strep pyogenes*
-*Staph aureus*
-Trauma

Signs & symptoms
-Abrupt onset of high fever, sore throat, stridor, dysphagia, drooling, trismus
-Sitting child that won't lie down, head leaning forward (sniffing or tripod position)

Differential
-Croup
-Peritonsillar abscess
-Foreign body
-Diptheria

Workup
-Lateral x-ray for "thumb sign"



Management
-Send to ED for inpatient management and antibiotics as any manipulation of glottis could result in airway obstruction

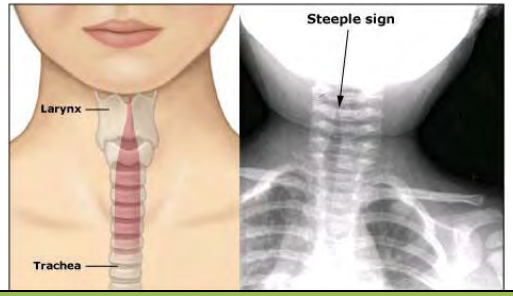
Croup

Agents
-Usually parainfluenza virus
-RSV
-Human metapneumovirus

Signs & symptoms
-Average child is 18 months of age
-Stridor, hoarseness, barking seal cough, low-grade fever
-Rales, rhonchi, wheezing
-Symptoms worse at night

Differential
-Epiglottitis
-Neoplasm
-Bacterial tracheitis
-Pharyngeal abscess
-Foreign body

Workup
-CXR showing "steeple sign"



Management
-Supportive: cool mist humidifier
-Send to ED for inhaled epinephrine if severe or if there is stridor at rest
-Steroids

Influenza

Influenza vs. common cold
-Flu = abrupt onset (sx worsen over 3-6 hours) with fever > 101.5, severe myalgias, headache, malaise, painful dry cough, sore throat, rhinitis
-Cold = slow, insidious onset, usually no headache or chills, sore throat, stuffy nose, sneezing, mild aches

Prevention
-Inactive vaccine starting at 6 months (first-time vaccination in kids under 9 requires 2 doses)
-Live vaccine if 2-49 and healthy (warning: viral shedding)

Workup
-Nasopharyngeal swab (may be done just for epidemiologic purposes)

Treatment
-Antipyretic/analgesic
-Albuterol neb
-Ipratropium inhaler (Atrovent) for secretions
-Consider steroids
-Consider antivirals (oseltamivir, zanamivir) for influenza A or B only with hospitalization, severe or progressive disease, age under 2 or over 65, and for outbreak control in institutions or health care workers, AND MUST BE within 48 hours of start of symptoms to help at all

Sequelae
Secondary *Staph aureus* pneumonia may follow

Pertussis

Prevention
-Dtap vaccine series for kids
-Tdap vaccination for adults to protect kids

Signs & symptoms
-Initial: cold-like; rhinorrhea, lacrimation, dry cough with episodes of severe cough, low-grade fever; post-tussive emesis
-Paroxysmal stage: coughing becomes more severe and may persist up to 10 weeks at this stage; paroxysmal whooping may be heard
-Convalescent stage: coughing diminishes as patient recovers and disappears over 2-3 weeks but may recur with subsequent URIs

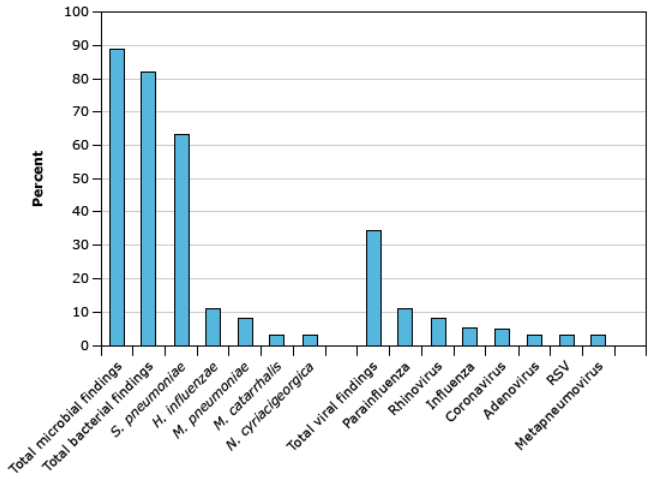
Workup
-*Bordetella* culture or PCR from nasopharyngeal swab

Management
-Macrolides are DOC
-Septra is an alternative

Prognosis
-May be infectious for several weeks if untreated

Pneumonia

Microbial etiology of community-acquired pneumonia in patients who underwent comprehensive testing



Prevention with pneumococcal vaccination	
23 valent (Pneumovax)	-Adults over 65 -Persons aged 19-64 years with chronic cardiovascular disease (including CHF and cardiomyopathy), chronic pulmonary disease (including asthma and COPD), DM, alcoholism, chronic liver disease (including cirrhosis), CSF leak, cochlear implant, cigarette smoking - Persons aged 19-64 years who are residents of nursing homes or long-term care facilities -Singe revaccination recommended if adult was < 65 and it was more than 5 years ago when they got it, and in immunocompromised 5 years after initial dose
13 valent	-Adults who are immunocompromised (should get 23 valent also, but not at same time) -Routine for all kids under 5 -Kids 6-18 who have sickle cell disease, HIV or other immunocompromising conditions, cochlear implant, or CSF leak
7 valent	-No longer being used

Signs & symptoms
-Rigors, sweats, fever or subnormal temp, cough ± sputum, dyspnea, pleuritic chest pain, fatigue, myalgias, abdominal pain, anorexia, headache, AMS
-Pleural effusion: pulmonary consolidation, crackles, dullness to percussion, ↓ breath sounds

Outpatient

Workup
-CXR: may lag behind PE findings! -Urine test for *Legionella*
-CBC, BMP

CXR Findings
-Can't tell explicitly viral vs pneumonia by patterns (old myth!)
-Lobar pneumonia: suggests *Strep pneumo, H flu, Legionella*
-Patchy infiltrates in multiple lung areas (bronchopneumonia): suggests *Staph aureus*, gram negs, atypicals, viruses
-Fine dense granular infiltrates (interstitial pneumonia): suggests influenza, CMV, PCP
-Lung abscess: suggests anaerobes
-Nodular lesions suggests fungal

Management
-CAP → macrolide
-Underlying comorbidity (higher risk = need to cover resistant *Strep pneumo*, enterics, *Moraxella*, anaerobes) → antipneumococcal FQ like levo, or macrolide + β-lactam (cefpodoxime, cefuroxime, amox HD, ceftriaxone)

Disposition
-Use PORT score or CURB-65 to estimate risk (QX Calculate app) and determine outpatient vs inpatient
-ER if RR > 30, HR > 125, SBP < 90, comorbidities

Prognosis
-Fever clears after 2-4 days of treatment
-CXR clears after 30 days (up to 6 mos if elderly)

Inpatient

HAP = pneumonia appearing > 48 hours after admission, or PNA in a recently hospitalized pt

HCAP = PNA in non-hospitalized pt that has had extensive healthcare contact (group home, SNF, IV therapy, HD, etc)

VAP = ventilator-associated pneumonia

Additional Workup
-ICU or EtOH or pleural effusion→ blood culture, sputum culture, *Legionella* & pneumococcal antigen testing

Non-HCAP Management
-Non ICU → initial therapy with anti-pneumococcal β-lactam (ceftriaxone, ertapenem, or ampicillin-sulbactam) + macrolide (cover atypicals), or monotherapy with a FQ
-ICU patients → initial therapy same as non-ICU, add vanco if suspecting MRSA, add anti-pseudomonal drug for COPD or frequent steroid or AB users (β-lactam + FQ)
-Clinical improvement should occur within 72 hours
-Switch from IV to orals with clinical improvement
-F/u CXR for patients over 50 at 7-12 weeks

Empiric HAP/HCAP/VAP Management
-Need to cover MRSA: vanco or linezolid
-Need to cover *Pseudomonas* and other gram negs: Zosyn, cefepime, ceftazidime, aztreonam (only for severe PCN allergy b/c it's not as effective)
-Need additional coverage for gram negs and atypicals: cipro, levo, gentamycin, tobramycin, or carbapenem

Respiratory Syncytial Virus

-Highly contagious, transmitted via aerosols or fomites
-The most common cause of fatal acute respiratory infections in infants and young children
-Causes a spectrum of disease from URTs, LRTs, pneumonia
-Most serious disease is in preemies, chronic lung disease, heart defects, asthma, immunocompromised, and the elderly

Management
-Supportive
-May need hospitalization with fluid and respiratory support
-Albuterol trial
-Steroids only in older kids, not infants
-Ribavirin for select infants

Tuberculosis

Signs & symptoms
-Latent or primary infection: Asymptomatic
-Active infection: cough, fever, weight loss, night seats, hemoptysis, fatigue, decreased appetite, chest pain

Workup
-If high suspicion, most clinics don't workup but put a mask on and send to ER
-CXR: active infection (infiltrates in mid or lower fields, hilar adenopathy, cavitation, emyema) or previous (pulmonary nodules, apical fibrosis, Ghon lesion)
-TB skin test, AFB smear

Active TB drug regimens
-Initial for 2 months: isoniazid, rifampin, pyrazinamide, ethambutol
-Continuation for 4-7 months: isoniazid and rifampin

Latent TB drug regimens
-9 months of isoniazid or 4 months of rifampin

Monitoring
-Sputum smears and cultures throughout treatment
-Vision checks and color vision testing with ethambutol
-CMP, CBC, and bili

NEOPLASTIC DISEASE

Lung Cancer (Bronchogenic Carcinoma)

-85% of cases occur among smokers
-Other contributing causes include radon gas, asbestos, and environmental pollutants
-2 major groups (small cell and non-small cell) account for 95% of lung cancers
-Other lung cancers are rarer and include primary pulmonary lymphoma, carcinoid tumors, bronchoalveolar cancers, and mesotheliomas
-Overall survival rate of 14%

Small Cell Carcinoma
-Metastasize rapidly to regional lymph nodes and distant sites
-Classified as limited or extensive disease
-Very responsive to chemo
-Remission is common but so is recurrence → overall survival of 5%

Signs & symptoms
-Lung cancers are more like to cause paraneoplastic syndromes such as hypercalcemia, SIADH, ectopic ACTH secretion, Lambert-Eaton myasthenic syndrome, and hypercoagulable states
-Nonspecific cough or dyspnea
-Chest pain
-Hemoptysis
-Anorexia, weight loss, fevers, night sweats
-Hoarseness due to compression of the recurrent laryngeal nerve
-Facial or UE swelling from SVC syndrome
-Bone, brain, liver, or adrenal symptoms from mets
-Axillary or supraclavicular adenopathy
-Digital clubbing

Differential
-TB
-Fungal infection
-Mets to the lung
-Sarcoidosis

Workup
-Begin with CXR
-F/u masses with CT
-Sputum cytology
-Bronchoscopy
-Transthoracic needle biopsy
-Node sampling via transbronchial biopsy, mediastinoscopy, or mediastinotomy

Management
-Assess feasibility of surgical resection and overall patient health/quality of life issues
-Radiation for advanced disease or nonsurgical candidates
-Combination chemotherapy for candidates
-Monitoring for recurrence

2004 WHO classification of invasive malignant epithelial lung tumors

Squamous cell carcinoma
Variants: papillary, clear cell, small cell, basaloid
Small cell carcinoma
Variant: combined small cell carcinoma
Adenocarcinoma
Adenocarcinoma, mixed subtype
Acinar adenocarcinoma
Papillary adenocarcinoma
Bronchioloalveolar carcinoma
Variants: nonmucinous, mucinous, mixed nonmucinous and mucinous or indeterminate
Solid adenocarcinoma with mucin production
Variants: fetal adenocarcinoma, mucinous ("colloid") carcinoma, mucinous cystadenocarcinoma, signet ring adenocarcinoma, clear cell adenocarcinoma
Large cell carcinoma
Variants: large cell neuroendocrine carcinoma, combined large cell neuroendocrine carcinoma, basaloid carcinoma, lymphoepithelioma-like carcinoma, clear cell carcinoma, large cell carcinoma with rhaboid phenotype
Adenosquamous carcinoma
Sarcomatoid carcinoma
Variants: pleomorphic carcinoma, spindle cell carcinoma, giant cell carcinoma, carcinosarcoma, pulmonary blastoma
Carcinoid tumor
Variants: typical carcinoid, atypical carcinoid
Salivary gland tumors
Variants: mucoepidermoid carcinoma, adenoid cystic carcinoma, epithelial-myoepithelial carcinoma

Non-Small Cell Carcinoma	
-Arises as discrete masses within the lung parenchyma that can spread to regional lymph nodes and then metastasize to distant sites -Squamous, adeno, and large cell carcinomas	-Limited response to chemo -Surgical resection of limited tumors can be curative -Staged by TNM

Bronchial Carcinoid Tumor	Squamous Cell Carcinoma	Adenocarcinoma
-Previously known as bronchial adenoma -Rare group of pulmonary neoplasms characterized by neuroendocrine differentiation and relatively indolent clinical course -Can also arise in the thymus, GI tract, and ovary -Surgical resection is treatment of choice	-Associated with slow growth and late metastasis	-Peripheral -Rapid growth with mets -Associated with lung scarring, not smoking

Pulmonary Nodules

Classification	Differential	Workup	Further Evaluation
Solitary pulmonary nodule = < 3 cm, round, isolated opacity outlined with normal lungs (no infiltrate, atelectasis, or adenopathy)	-Bronchogenic carcinoma -Infectious granuloma or abscess -Hamartoma -Mets -AVM -Resolving pneumonia -Rheumatoid nodule -Pulmonary infarction -Carcinoid -Pseudotumor	-Review old radiographs -Follow-up suspicious CXR nodules with hi-res chest CT -Infection clues: doubling < 30 days -Benignity clues: doubling > 1 year, well-defined borders, central calcification on CT -Malignancy clues: ill-defined borders, lobular borders, spicules on CT, peripheral halo on CT, stippled or eccentric calcification on CT, cavitary lesions with thick walls	**< 5% probability of malignancy → watchful waiting with serial imaging** -Age under 30 -Lesions stable over 2 years -Benign calcification pattern **Intermediate probability of malignancy →** refer for TTNA, bronchoscopy, PET, or VATS **> 60% probability of malignancy →** refer for staging and excision
Multiple pulmonary nodules	-Mets, bronchogenic cancer, lymphoproliferative cancer, TB, abscess, granuloma, fungus, sarcoidosis, silicosis, coal worker's pneumoconiosis, MAC, AVM, rheumatoid nodules, hamartomas, Wegener's granulomatosis, methotrexate, eosinophilic granuloma, echinococcosis, paragonimiasis		

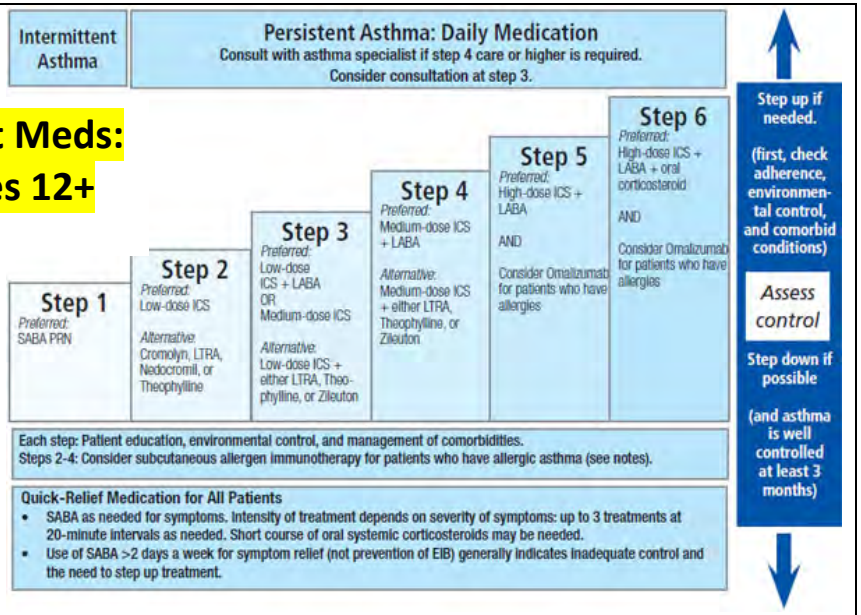
OBSTRUCTIVE PULMONARY DISEASE

Asthma

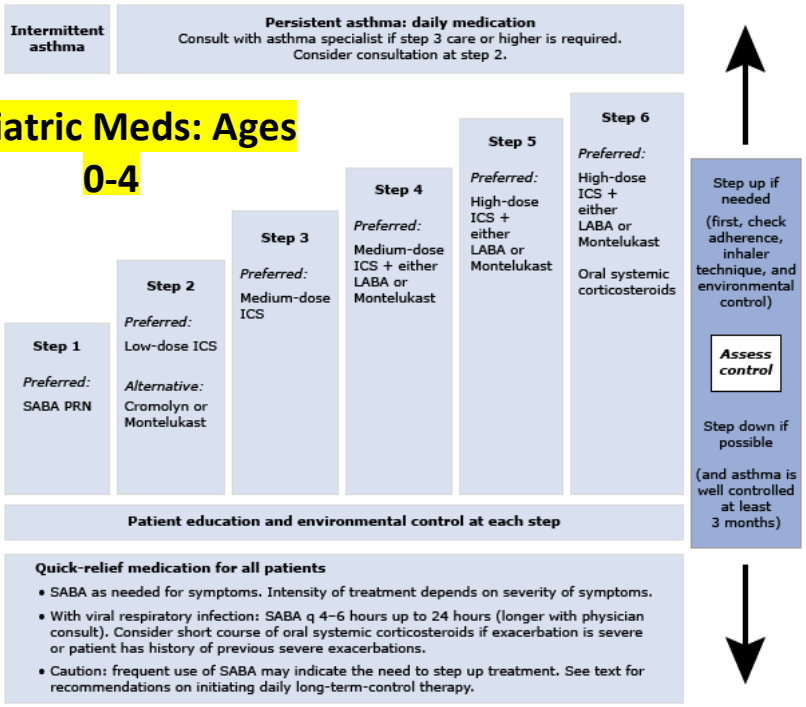
Management and monitoring	PCP treatment for adult acute exacerbation	PCP treatment for pediatric acute exacerbation
-Refer for methacholine challenge if not sure it's asthma -Home peak flow monitoring: have pt establish baseline by measuring am and noon readings over 2-3 weeks, establish green, yellow, and red zones -Assess efficacy of treatments at each visit: nighttime awakenings, use of emergency inhalers, ER or urgent care visits -Step up meds if nighttime awakenings are 2+ times per month -Office spirometry is preferred over peak flow measurement but not all PCPs will have this	-Peak flow > 70% predicted (dyspnea on exertion) → inhaled SaβA up to 3x, can be managed at home -Peak flow 40-69% of predicted (dyspnea limiting usual activity) → inhaled SaβA up to 3x, then oral corticosteroids if no improvement -Peak flow < 40% predicted (dyspnea with inability to speak, diaphoresis) → ER	-Assess severity using Pulmonary Index Score -Mild: SaβA neb up to 3 doses, with oral steroids given after 1st dose if no improvement -Moderate: oxygen if needed, SaβA + ipratropium neb up to 3 doses, with oral steroids after 1st dose

Medication	Notes
SAβAs: albuterol, levalbuterol, pirbuterol	-Use of more than one canister per month indicates need to step up -Use more than twice per week indicates need to step up
Anticholinergics: ipratropium, tiotropium	-Alternative to SaβAs or as adjunct for severe exacerbation
Systemic corticosteroids	-For 3-10 days after exacerbation
Inhaled corticosteroids: budesonide, beclomethasone, flunisolide, fluticasone, mometasone	-Use a spacer
LAβAs: salmeterol, formoterol, indacaterol	-Only use for shortest time needed to control symptoms -Only use long-term for someone whose asthma is not controlled on other meds -Don't use without ICSs, increased risk of death! → make use of combo inhalers to comply
Leukotriene modifiers: montelukast, zafirlukast, zileuton	-Good for exercise-induced asthma
Mast cell stabilizers: cromolyn	-Good for seasonal asthma and exercise-induced bronchospasm -Takes 2 weeks for therapeutic response
Theophylline	-An adjunct to ICSs for management of nighttime symptoms -Requires serum monitoring
Omalizumab	-For severe allergic asthma in patients with frequent exacerbations, already on high steroid dose

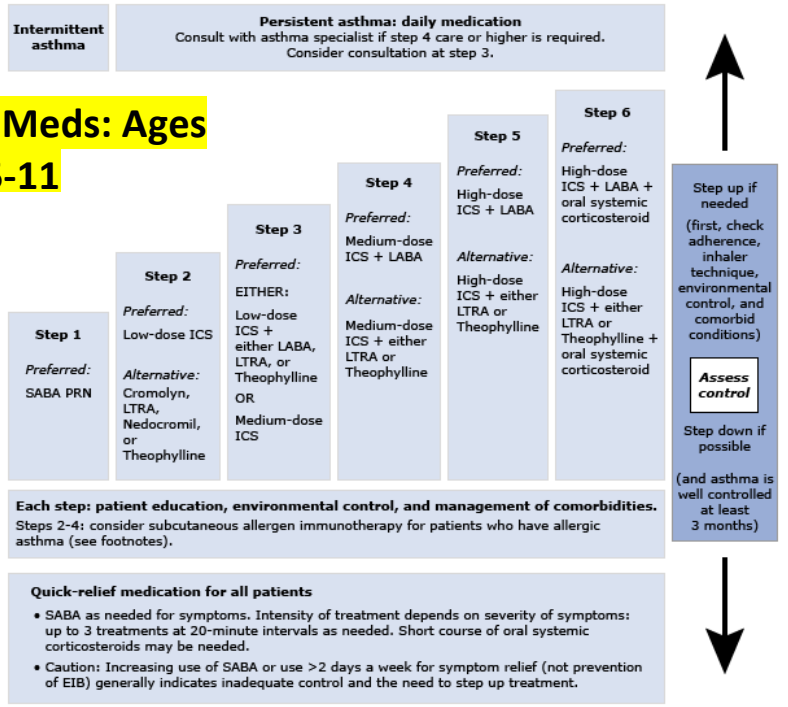
Adult Meds: Ages 12+

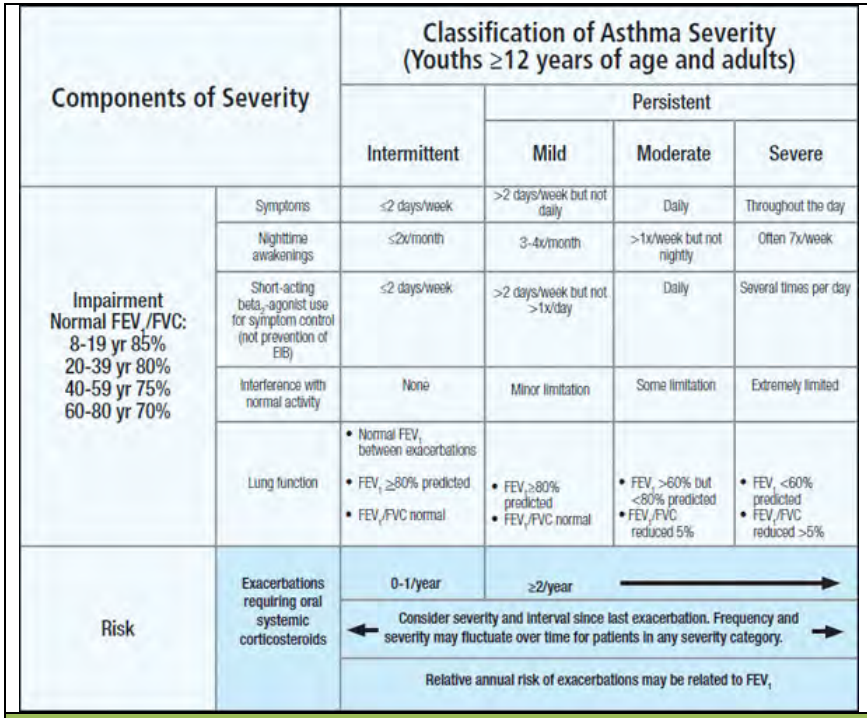


Pediatric Meds: Ages 0-4



Pediatric Meds: Ages 5-11





Components of Severity		Classification of Asthma Severity (Youths ≥12 years of age and adults)			
		Intermittent	Persistent		
			Mild	Moderate	Severe
Impairment Normal FEV_1/FVC: 8-19 yr 85% 20-39 yr 80% 40-59 yr 75% 60-80 yr 70%	Symptoms	≤2 days/week	>2 days/week but not daily	Daily	Throughout the day
	Nighttime awakenings	≤2x/month	3-4x/month	>1x/week but not nightly	Often 7x/week
	Short-acting $beta_2$-agonist use for symptom control (not prevention of EIB)	≤2 days/week	>2 days/week but not >1x/day	Daily	Several times per day
	Interference with normal activity	None	Minor limitation	Some limitation	Extremely limited
	Lung function	• Normal FEV_1 between exacerbations • FEV_1 ≥80% predicted • FEV_1/FVC normal	• FEV_1 ≥80% predicted • FEV_1/FVC normal	• FEV_1 >60% but <80% predicted • FEV_1/FVC reduced 5%	• FEV_1 <60% predicted • FEV_1/FVC reduced >5%
Risk	Exacerbations requiring oral systemic corticosteroids	0-1/year	≥2/year →		
		← Consider severity and interval since last exacerbation. Frequency and severity may fluctuate over time for patients in any severity category. →			
		Relative annual risk of exacerbations may be related to FEV_1			

Asthma Exacerbation

Signs & Symptoms
-Expiratory wheezing
-Cough
-Chest pain/tightness
-SOB with prolonged expiration
-Tachypnea & tachycardia
-Beware the quiet chest with intercostal retractions and accessory muscle use!

Workup
-CXR to eval for other causes of SOB
-Peak flow (exac if < 80% baseline, severe exac if < 50% baseline)
-Check ABG for severe distress

Management
-Duoneb tx (albuterol + ipratropium) q 20 minutes x 3
-MDI albuterol is just as effective as nebulized albuterol
-IV or PO steroids if inadequate response
-Supplemental O2 if needed
-DON'T use ICS
-MgS for refractory cases (has bronchodilator effect)
-Mechanical vent if peak flow persists < 25%
-Admit if not responding in 4-6 hours to treatments
-Nonstandard therapies: montelukast, helium, furosemide, ketamine, macrolides

Cystic Fibrosis

-Autosomal recessive inherited defect of protein regulating chloride channels, bicarb, and other ions (CFTR protein) → defective mucociliary clearance → mucus obstruction, inflammation, infection, and fibrosis
-Also affects the pancreas and vas deferens
-Most commonly affects Caucasians

Signs & symptoms
-Will be on a continuum depending on % of normal CFTR functioning
-No known abnormalities until there is < 10% normally functioning CFTR proteins; < 10% → absence of vas deferens, < 5% → sweat abnormality, < 4.5% → progressive pulmonary infections, < 1% → pancreatic deficiency
-Recurrent pulmonary infections with atypical bacteria (*Staph aureus* in infancy and *Pseudomonas* in adulthood), poorly controlled asthma, failure to thrive, meconium ileus, pancreatitis, vitamin deficiencies, nasal polyps, sinusitis, fatty liver, liver fibrosis, portal HTN, gallstones, jaundice, osteoporosis or frequent fractures from vit D deficiency, rectal prolapse from thick stools, intestinal strictures, appendicitis, GERD, infertility, delayed puberty, smooth muscle growth around bronchioles, respiratory symptoms, diabetes, enlarged or deficient spleen
-Acute exacerbation (will be bronchial rather than pneumonia): increased cough of sputum, sputum color change, dyspnea, fatigue, decreased exercise tolerance, poor appetite, new tachypnea, retractions, wheezing, rhonchi, weight loss, fever, new findings on CXR, ↓ PFTs, hypoxia

Workup
-Newborn screens detect only severe disease
-Genetic screens of 23 most common mutations only identify CF in Caucasians
-Buccal DNA swab for other mutations
-Sweat chloride test is confirmatory

Management
-Dietary support: higher BMI associated with better lung functioning, need high caloric intake to combat malabsorption, salt supplements, pancreatic lipase supplements, fat-soluble vitamin supplements
-Promote mucus clearance: percussion and chest compression vests, upside-down coughing, huff breathing, oral oscillators, exercise, CPAP, saline mist, albuterol
-Infection control: cyclic use of antibiotics against *Pseudomonas*, intermittent IV antibiotics, oral antibiotics for 2-3 weeks after exacerbation
-Frequent office visits with PFTs, sputum culture, diabetes screens, bone densitometry, CBC, PT/PTT, UA, vitamin levels, LFTs, albumin, immunizations

Prognosis
-Lung function declines at about 2% per year, but this will speed up with increasing exacerbations
-Patients are unable to return to previous baseline with each exacerbation
-Median survival age is 38

Chronic Obstructive Pulmonary Disease (COPD)

Chronic bronchitis = proximal predominant
-"Blue bloaters" = depressed respiratory drive with use of accessory muscles → acidosis, productive cough, wheezing, rhonchi, hyperinflation of lungs, cor pulmonale

Emphysema = distal-predominant
-"Pink puffers" = high RR/dyspnea due to damaged vascular beds, distant breath sounds, hyperinflation of lungs, low cardiac output

→ Can have both chronic bronchitis and emphysema, and a subset of these patients also have asthma

-Chronic airway inflammation → systemic release of inflammatory cytokines → CAD, renal insufficiency, neuromyopathy, osteoporosis, cachexia, downward spiral
-Airway obstruction is not fully reversible

Workup
-PFTs demonstrating FEV1/FVC ratio < 0.70

Table 2

Stages and Recommended Treatment of COPD

Stage	Features	Recommended Treatment
All stages		Avoid risk factors such a smoking, irritants, allergens Receive influenza vaccine annually Pneumococcal polysaccharide vaccine Treat complications accordingly
Stage 1: Mild COPD	$FEV_1/FVC<70\%$ $FEV_1 \geq 80\%$ predicted With or without symptoms	Use short-acting bronchodilator as needed
Stage 2: Moderate COPD	$FEV_1/FVC<70\%$ $50\% \leq FEV_1 < 80\%$ predicted With or without symptoms	Maintenance therapy with 1 or more bronchodilators, pulmonary rehabilitation.
Stage 3: Severe COPD	$FEV_1/FVC<70\%$ $30\% \leq FEV_1 < 50\%$ predicted With or without symptoms	Maintenance therapy with one or more bronchodilators Inhaled corticosteroids for patients with recurring exacerbations or with persistent symptoms despite therapy with bronchodilators, pulmonary rehabilitation
Stage 4: Very severe COPD	$FEV_1/FVC<70\%$ $FEV_1 <30\%$ predicted or $<50\%$ predicted plus presence of chronic respiratory failure ($PaO_2 < 60$ mm Hg while breathing room air at sea level)	Regular treatment with 1 or more bronchodilators Use inhaled corticosteroids if symptoms persist despite bronchodilator therapy, pulmonary rehabilitation Long-term oxygen therapy if chronic respiratory failure Consider surgical treatments

FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity; PaO_2 = arterial partial pressure of oxygen.
Adapted from references 17-20.

Management of stable COPD
-Goal is to reduce exacerbations requiring hospitalization
-Get PFTs for diagnosis and classification of disease stage and to follow course of disease (FEV1/FVC < 70% with FEV1 < 80% are diagnostic criteria)
-Add treatments in a stepwise fashion as needed
-New drug roflumilast (PPD-4 inhibitor) indicated for severe COPD with chronic bronchitis and history of acute exacerbations
-Not recommended: expectorants, antitussives (COPD cough not centrally mediated), respiratory stimulants
-Increased survival when oxygen therapy is used > 18 hours per day

Management of acute exacerbations
-SaβA + inhaled anticholinergic
-10-14 day steroid taper
-Antibiotics if there is increased sputum purulence or vol or increased dyspnea
-Supplemental oxygen to 90-94% saturation
-NPPV is the preferred form of ventilation if needed

Bronchiectasis

-Permanent abnormal dilation and destruction of bronchial walls
-Caused by an infectious insult + impaired drainage, airway obstruction, or a defect in host defense (ex. FB aspiration, smoking, CF)
-An obstructive lung disease

Signs & symptoms
-Cough
-Daily production of mucopurulent sputum for months to years
-Dyspnea, hemoptysis, wheezing, pleuritic chest pain

Workup
-Abnormal CXR such as linear atelectasis or dilated and thickened airways
-Labs: CBC, Ig levels
-Sputum smear and culture
-PFTs
-Chest CT is diagnostic

Management
-Outpatient acute exacerbation → FQ
-Inpatient acute exacerbation → begin 2 IV antipseudomonal drugs
-Chest physiotherapy
-Inhaled steroids only for severe wheeze or cough or acute exacerbation

PLEURAL DISEASES

Pleural Effusion

-Excess fluid in the pleural space between the lung and chest wall
-Fluid may be serous, pus, lymph, or blood

Causes
-Usually due to ↑circulatory hydrostatic pressure or permeability: L heart failure, pneumonia
-Decreased circulatory oncotic pressure: ↓albumin
-Decreased pressure in pleural space: pneumothorax
-Impaired lymphatic drainage: malignancy
-Movement of fluid from peritoneal space: ascites

Signs & Symptoms
-Dyspnea
-Cough
-Chest pain

Workup
-Frontal CXR shows blunting of costophrenic margins, can see free fluid in LLD position, should also get lateral view
-May only be able to see very small pleural effusions on CT
-Thoracentesis is diagnostic 75% of the time and is indicated with any new pleural effusion, esp with atypical clinical situation or failure to progress as anticipated and is used to determine transudate vs exudate
-Routine pleural fluid tests: cell count, pH, protein, LDH, glucose
-Additional pleural fluid tests for inconclusive results: amylase, cholesterol, TG, Gram or AFB stains, bacterial or AFB cultures, cytology

Exudates
-Caused by local ↑capillary and membrane permeability, impaired lymph drainage, or fluid shift from peritoneal space
-Defined by 1+ presence of Light's Criteria: pleural protein/serum protein > 0.5, pleural LDH/serum LDH > 0.6, or pleural LDH > 2/3 ULN of serum LDH
-Usually infectious
-Also malignancy, trauma, pulmonary infarction, PE, autoimmunity, pancreatitis, ruptured esophagus, post-CABG, drug reaction

Transudates
-Caused by systemic hydrostatic or oncotic pressure imbalances, or less commonly from fluid shift from another space
-Usually CHF
-Also cirrhosis, ↓albumin, nephrotic syndrome, acute atelectasis, myxedema, peritoneal dialysis, PE, Meig's syndrome, obstructive uropathy

Management
-Thoracentesis ± chest tube
-Treat underlying cause

Pneumothorax

Types
-Primary spontaneous: occurs most commonly in thin 20-40 year old males, but can also occur due to ruptured blebs in smokers
-Secondary spontaneous: occurs in setting of underlying lung disease like COPD and is more life threatening
-Traumatic: a result of penetrating or blunt chest injuries
-Tension: occurs when air continues to fill the pleural space and compresses the heart and great veins

Signs & Symptoms
-Sudden onset dyspnea
-Pleuritic chest pain
-Decreased or absent breath sounds on affected side

Workup
-CXR: will show mediastinal and tracheal push away from tension pneumo, black space from collapsed lung

Management
-Primary spontaneous → aspiration, may need pleural abrasion to prevent recurrences
-Secondary spontaneous or traumatic → chest tube unless very small
-Flutter dressing if open pneumothorax from penetrating trauma
-Tension pneumo requires emergent needle thoracostomy (2nd intercostal space) to convert it to a simple pneumothorax, followed by chest tube insertion (5th intercostal space) for definitive treatment

PULMONARY CIRCULATION DISORDERS

Cor Pulmonale

-A subset of R-sided CHF where diastolic R ventricular failure occurs as a result of pulmonary HTN associated with diseases of the lung, upper airway, or chest wall (most commonly COPD)
-Does NOT include R sided CHF as a result of L sided CHF or congenital heart defect
-Usually slow and progressive but may be acute

Signs & symptoms
-DOE
-Fatigue
-Lethargy
-Exertional syncope or angina
-S4
-↑ JVP
-Peripheral edema

Workup
-Concomitant pulmonary HTN workup

Management
-Treat underlying cause
-Diuretics

Pulmonary Embolism

Etiologies
-Most arise from LE DVT
-Stasis: surgery, heart failure, chronic venous stasis, immobility
-Blood vessel injury: fractures, surgery
-Hypercoagulability: postpartum, malignancy, OCPs, protein C/S/antithrombin III deficiency, lupus anticoagulant, factor V Leiden, prothrombin gene mutations, hyperhomocysteinemia

Differential
-Pneumonia
-Infection
-Obstructive lung disease
-CHF
-Msk disease
-Acute MI
-Anxiety

Management
-Supplemental O2 if hypoxic
-Give empiric heparin while waiting for imaging results (depending on level of suspicion as well as timeframe to get test results back)
-IVC filter for repeat clots or

Classification
-Massive = sustained hypotension, pulselessness, persistent bradycardia, or need for inotropic support
-Submassive = pt is normotensive with myocardial necrosis
-Minor/nonmassive = normotensive with no myocardial necrosis

Signs & symptoms
-Onset does not have to be sudden!
-Dyspnea, pleuritic or anginal chest pain, cough, wheezing
-Leg swelling or pain
-Hemoptysis
-Palpitations, syncope
-Tachycardia and tachypnea, loud P2 from pulmonary HTN
-Diaphoresis
-Fever
-Homan's sign
-Orthopnea
-↓ Breath sounds
-JVD
-"Massive PE" → hypotension

Workup
-D-dimer is only useful if PE is very unlikely! Otherwise risk is too great that there will be a false negative
-PE highly unlikely in ED if pt meets these criteria: age < 50, HR < 100, SpO2 ≥ 95%, no hemoptysis, no estrogens, no prior h/o DVT or PE, no unilateral leg swelling, no surgery or hospitalization in past 4 weeks
-ABG if respiratory distress present: will usually show respiratory alkalosis, overall not very useful in diagnosing PE
-Troponin
-EKG sometimes shows S1Q3T3
-Troponins: May be + in moderate to large PEs from acute R heart overload
-May have concomitant DVT detected by US
-CXR may show edema, cardiomegaly, prominent pulmonary vein, left sided pleural effusion, or atelectasis
-VQ scan, spiral CT pulmonary angiography (test of choice), or pulmonary angiography (gold standard but hi morbidity, requires femoral cath)
-Pregnant? VQ scan vs CTA and radiation dose is debated

poor anticoagulant candidates
-Consider lytics for massive PE
-Consider surgical embolectomy for failed or contraindicated anticoagulation or lytic therapy
-Can be managed outpatient for select stable patients with no comorbidities
-Continue outpatient anticoagulation for at least 3 months

Prognosis
-30% are fatal without treatment
-Less than 10% mortality if treated by anticoagulation

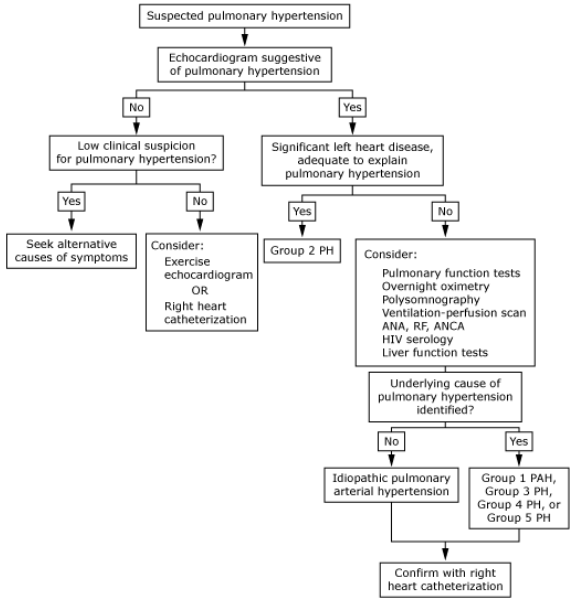
Pulmonary Hypertension

Etiologies
-Arteriolar narrowing in the lungs (idiopathic, familial, scleroderma, portal HTN, HIV, drugs or toxins, chronic hemolytic anemia)
-Left heart disease
-Lung disease or hypoxia: COPD, interstitial lung disease, OSA, chronic high altitude
-Chronic thrombotic or embolic disease
-Splenectomy
→ May be secondary to multiple "hits"

Signs and symptoms
-Dyspnea, fatigue, chest pain, palpitations, LE edema, dizziness, syncope
-Tachypnea and tachycardia
-Evidence of R heart failure: JVD, ascites, edema
-Loud P2 from elevated pulmonic pressures slamming valve shut
-Tricuspid regurgitation murmur because it can't shut all the way due to the dilation of muscle
-Pulmonic regurgitation murmur
-Pulsatile liver

Differential
-Left CHF
-CAD
-Liver disease
-Budd-Chiari syndrome (hepatic or vena cava thrombosis)

Workup



-R heart cath showing mean pulm artery pressure > 25 mm Hg at rest is diagnostic

Management
-Diuretics for fluid retention
-Anticoagulation therapy if cause is familial, drug, or idiopathic
-Supplemental oxygen for resting hypoxia
-CCB
-Last resort is atrial septostomy or lung transplant
-F/u every 3 months

Prognosis
-Progressive and fatal if untreated
-Average time for correct diagnosis is 15 months because symptoms other than SOB may not be present and there is a lot to rule out

RESTRICTIVE PULMONARY DISEASE

Idiopathic Pulmonary Fibrosis

-A chronic, progressive fibrotic disorder of the lower respiratory tract
-Can be genetic or acquired
-Usually affects adults over 40
-Risk factors: smoking, environmental pollution, chronic microaspiration

Signs & symptoms
-DOE
-Persistent nonproductive cough
-Crackles or Velcro rales
-Digital clubbing

Workup
-PFTs
-Chest CT
-Lung biopsy or bronchoalveolar lavage
-Labs: CMP, CBC, CK
-UA
-EKG

Management
-No evidence that any treatment improves survival or quality of life
-Supportive care: oxygen, pulmonary rehab
-N-acetylcysteine pills
-Clinical trial enrollment
-Don't use steroids as monotherapy
-Lung transplant
-Acute exacerbation → broad spectrum antibiotics, high dose steroids, and azathioprine

Pneumoconiosis

-Occupational lung disease and restrictive lung disease caused by the inhalation of dust

Types
-Coal worker's pneumoconiosis (aka miner's lung, black lung, or anthracosis)
-Asbestosis
-Silicosis
-Bauxite fibrosis
-Siderosis
-Byssinosis

Signs & Symptoms
-SOB
-Cough
-Weight loss
-Fatigue
-Pleuritic pain
-May be acute, chronic, or accelerated

Workup
-CXR shows patchy bibasilar interstitial infiltrates and/or honeycombing

Management
-Bronchodilators
-Supplemental O2
-Aggressive management of respiratory tract infections with abx
-Referral for transplant with advanced disease

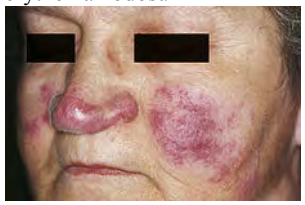
Sarcoidosis

-A rheumatic disease
-Cause is unknown
-More common in black patients
-Course of disease may be acute and severe or mild and chronic

Differential
-Leukemia
-Multiple myeloma
-Amyloidosis
-Diabetes
-Thyroid or parathyroid disease
-IBD
-Hemochromatosis

Signs & symptoms

-Dyspnea
-Cough
-Chest pain
-Fatigue
-Eye manifestations
-Skin manifestations (with predilection for scarred or tattooed areas): lupus pernio or erythema nodosum



-Polyarthritis in the ankles, knees, wrists, or elbows
-Gum hyperplasia
-Myopathies or myositis are uncommon
-Neurologic: CN palsies, meningitis, brain lesions, neuroendocrine dysfunction
-Cardiac: arrhythmias, conduction delays, pulmonary HTN, CHF, pericarditis
-GI: abdominal pain, esophageal involvement
-Liver (90% of pts): jaundice, varices, granulomatous hepatitis
-Endocrine: hypercalcemia, goiter, thyroid nodules
-Renal: calculi, failure, nephritis
-GU: epididymitis, AUB

Workup
-CXR for staging (I-IV)
-Ophtho exam
-PFTs
-UA
-Labs: serum ACE, BMP
-CBC
-EKG
-TB test
-Granuloma biopsy
→ A diagnosis of exclusion

Management
-Aimed at site of disease
-Pulm → steroids, lung transplant
-Arthralgias → steroids, NSAIDs, colchicine, biologics
-Steroid sparing agents: methotrexate, cyclosporine, mycophenolate mofetil, azathioprine, cyclophosphamide

Prognosis
-Acute presentation has a high rate of spontaneous resolution

OTHER PULMONARY DISEASE TOPICS

Acute Respiratory Distress Syndrome (ARDS)

-Noncardiogenic pulmonary edema caused by capillary leaking from infection or inflammation → parenchymal inflammation and edema → impaired gas exchange and systemic release of inflammatory mediators → further inflammation, hypoxemia, and frequently multiple organ failure

Acute lung injury (ALI) = a less severe form of ARDS

Inciting Events
-Pneumonia of any kind
-Chemical inhalation
-Chest trauma
-Sepsis
-Pancreatitis
-Connective tissue disease: lupus
-Vasculitis
-Hypersensitivity rxn to blood transfusion
-Burns

Stages
-Stage I: clear CXR, infiltration of PMNs begins
-Stage II: develops over 1-2 days with patchiness on CXR with edema and type I alveolar cell damage
-Stage III: develops over 2-10 days with diffuse infiltration on CXR, exudates, proliferation of type II alveolar cells functioning as repair cells
-Stage IV: develops > 10 days with diffuse infiltration on CXR, involvement of lymphocytes → pulmonary fibrosis

Workup
Diagnostic criteria: known clinical insult < 1 week ago, bilateral opacities consistent with pulmonary edema on CXR or CT, not explained by cardiac failure or fluid overload, hypoxemia present on minimal ventilator settings ($PaO_2/FiO_2 < 300$)

Management
-Treat underlying cause!
-Usually mechanical ventilation is needed, using low tidal volumes and PEEP
-Use of steroids is controversial
-Supportive care: sedatives, nutritional support, BG control, DVT prophylaxis, GI prophylaxis, neuromuscular blockade for vent dyssynchrony to ↓O_2 demand, keep fluid balance even to negative

Prognosis
-Overall mortality 40-60%

Neonatal Respiratory Distress Syndrome (Hyaline Membrane Disease)

-A result of surfactant deficiency → alveolar collapse and diffuse atelectasis
-Typically occurs in preterm infants

Prevention
-Antenatal glucocorticoid treatment for women at risk for preterm delivery prior to 34 weeks of gestation
-If gestation is greater than 30 weeks, the fetal lung maturity may be tested by sampling the amount of surfactant in the amniotic fluid by amniocentesis

Signs & symptoms
-Respiratory distress and cyanosis soon after birth
-Tachypnea
-Tachycardia
-Chest wall retractions
-Abdominal breathing

Workup
-CXR showing diffuse ground glass appearance with air bronchogram

Management
-Inpatient with fluid balance, CPAP, exogenous surfactant

Foreign Body Aspiration

-Most common site is the right lung, followed by left lung, trachea/carina, and larynx

Signs & symptoms
-Choking episode followed by symptom-free period
-Respiratory distress
-Cyanosis
-AMS
-Generalized wheezing
-Coughing
-Recurrent pneumonia
-Diminished breath sounds

Workup
-Send to ED for bronchoscopy if severe symptoms
-CXR is problematic because most swallowed objects are radiolucent

Cough			
Duration	**Differential**		**Workup/treatment**
Acute = less than 3 weeks **Persistent** = greater than 3 weeks **Subacute** = postinfectious cough lasting 3-8 weeks	-Viral URI or postviral most common cause -Bacterial URI -Postnasal drip -Allergic rhinitis -Pneumonia: VS abnormalities or findings of consolidation -Pulmonary edema -PE		-CXR for smokers, weight loss, persistent cough without prior URI, abnormal VS -Pulse ox -Acute bronchitis → short-acting bronchodilators -Postnasal drip → antihistamine, decongestant, or nasal steroid -Antitussives: not to be used in kids under 2, dextromethorphan has the best evidence
Chronic = greater than 8 weeks	-Postviral -RSV -Parasites -COPD -Bronchiectasis -CF -Bacterial: MAC, TB, pertussis, *Mycoplasma, Chlamydophila* -Asthma -β-blocker -Chronic bronchitis in smokers	-Irritant inhalation -Tumor -ILD disease -Sarcoidosis -Chronic aspiration -GERD -Sinusitis -Laryngitis -TM irritation -ACEI -Psychogenic	1.) D/C ACEI, pertussis testing, r/o post-infectious cough 2.) Clear CXR → 2-4 week treatment for asthma, postnasal drip, or GERD 3.) 2 week prednisone trial 4.) Referral to pulm or ENT for chest or sinus CT, EGD, barium swallow, etc.

GASTROINTESTINAL SYSTEM

ESOPHAGUS

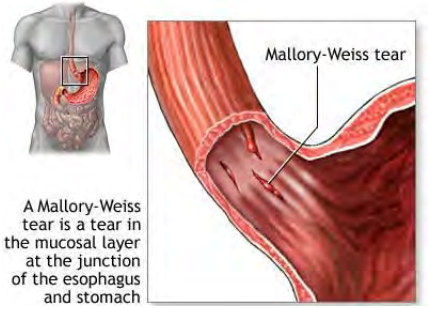
Mallory-Weiss Tear

-A tear of the distal esophagus at the gastroesophageal junction, typically occurring after a bout of vomiting
-Major cause of upper GIB

Risk Factors
-Underlying portal HTN

Signs & Symptoms
-Middle aged male presenting with hematemesis
-May have recent h/o alcohol ingestion

Workup
-Endoscopy is test of choice



Management
-Most will resolve bleeding spontaneously
-May require injection or thermal coagulation

Prognosis
-Risk of rebleeding

Esophageal Neoplasms

Benign	Malignant		
Leiomyoma -Tumor of smooth muscle -Surgical removal if symptomatic **Adenoma** -Tumor arising from glandular tissue -Usually found in areas of Barrett's esophagus **Esophageal Papilloma** -Associated with transformation to SCC	Usually occurs in males 50-70 **Types** -Squamous cell carcinoma: occurs in the upper 2/3 of the esophagus, risk factors are alcohol use, tobacco, achalasia, caustic esophageal injury, head and neck cancers, Plummer-Vinson syndrome, black ethnicity, male -Adenocarcinoma: occurs in the lower 1/3 of the esophagus, risks are Barrett's esophagus, white ethnicity, males -Lymphoma: very rare in esophagus → Recent trend towards adenocarcinoma	**Signs & symptoms** -Progressive solid food dysphagia -Weight loss -Usually is late stage by time patient is symptomatic **Workup** -CXR showing mediastinal widening, lung or bony mets -Barium swallow showing many infiltrative or ulcerative lesions and strictures -Chest CT -Endoscopic US for staging	**Management** -Surgical resection with gastric pull-up or colonic interposition -Palliative radiation -Chemo -Palliative stenting

Esophagitis						
Type	**Etiologies**	**Signs & symptoms**	**Differential**	**Workup**	**Management**	**Prognosis**
Medication-induced	-Major offenders are tetracyclines, anti-inflammatories, KCl, quinidine, alendronate	-Sudden onset odynophagia, retrosternal pain after ingestion of a pill		-Only necessary for severe presentations or atypical symptoms -Endoscopy or barium swallow		-Most cases heal without intervention within a few days
Eosinophilic	-Allergic response	-Dysphagia -Heartburn unresponsive to medications -H/o environmental allergies or atopy -Vomiting -Abdominal pain		-Upper endoscopy with biopsy	-Allergy referral -Elimination diet -Acid suppression -Topical glucocorticoids (swallowed fluticasone) -Esophageal dilation to treat strictures -Repeat endoscopies for change in symptoms	
GERD	-Transient lower esophageal sphincter relaxation -Hypotensive lower esophageal sphincter -Anatomic disruption of the gastroesophageal junction, often with hiatal hernia	-Heartburn -Regurgitation -Dysphagia -Chronic cough -Hoarseness	-Infectious esophagitis -Pill esophagitis -Eosinophilic esophagitis -PUD -Non-ulcer dyspepsia -Biliary tract disease -CAD -Esophageal motility disorder	-Endoscopy with biopsy for alarm symptoms (dysphagia, odynophagia, weight loss, iron deficiency anemia), symptoms refractory to empiric PPI trial, new onset symptoms in a patient over 50, or symptoms > 10 years -Ambulatory pH monitoring for negative endoscopy with persistent symptoms -Esophageal manometry for suspected motility disorder	-Lifestyle modification: ↑ HOB, avoid food before sleep, avoiding trigger foods, weight loss, smoking cessation -Change therapy as needed every 2-4 weeks -Maintain optimal therapy for 8 weeks -Recurrent symptoms within 3 months suggest disease best managed with continuous therapy -Acid suppression will reduce the acid but not the reflux! -Therapeutic regimens in order of increasing potency: OTC antacids or H2 blockers, rx H2 blockers BID (take 30 min to work), PPI for 2 weeks, 20 mg omeprazole daily, 20 mg omeprazole BID or 40 mg daily -Acid suppression with symptoms worse than mild or intermittent → PPI recommended over H2 antagonist -EGD every 3 years for patients with known Barrett esophagus	
Other	-HSV: mostly in immunocompromised -CMV -*Candida*					

Esophageal Motility Disorders

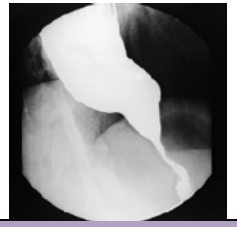
Achalasia

-Absence of normal esophageal peristalsis with increased tone of the LES

Etiologies
-Chagas' disease
-Others

Signs & symptoms
-Months to years of symptoms
-Gradual, progressive dysphagia of both solids and liquids
-Regurgitation, sometimes nocturnally
-Substernal discomfort or fullness after eating
-Poor esophageal emptying

Workup
-Manometry is the gold standard
-CXR showing enlarged, fluid-filled esophagus
-Barium swallow showing "bird's beak" from acute tapering of LES at gastroesophageal junction
-EGD to look for other cause



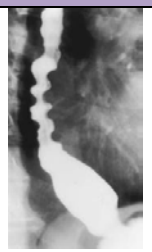
Management
-Smooth muscle relaxers like CCB, nitrates
-Balloon dilation of LES: high perf rate!
-Surgical myotomy
-Botox injection to relax LES

Diffuse Esophageal Spasm

-Simultaneous, nonperistaltic contractions of the esophagus
-Uncommon

Signs & symptoms
-Intermittent dysphagia
-Anterior chest pain unrelated to exertion or eating
-Provocation by stress, large food boluses, hot or cold liquids

Workup
-Barium swallow showing corkscrew contractions or "rosary bead" appearance
-Manometry shows intermittent, simultaneous contractions of high amplitude not related to swallowing along with periods of normal peristalsis



Management
-Disease is usually self-limiting

Scleroderma Esophagus

-Atrophy and fibrosis of esophageal smooth muscle → loss of LES competency, decreased peristalsis, and decreased gastric emptying
-Can also occur with progressive systemic sclerosis, Raynaud's, or CREST

Signs & symptoms
-Severe acid reflux
-Strictures
-Erosions
-Heartburn
-Dysphagia

Workup
-Manometry showing diminished peristalsis with low pressures, relaxed LES
-Barium swallow showing dilated, flaccid esophagus

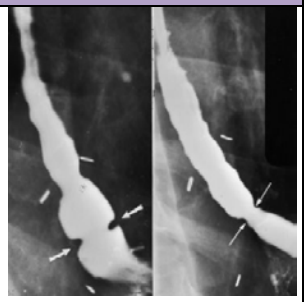
Esophageal Strictures

Causes
-Most benign strictures are from long-standing GERD
-Esophagitis
-Dysfunctional LES or motility
-Hiatal hernia
-Radiation
-Esophageal sclerotherapy
-Caustic ingestions
-Surgical anastomosis
-Rare derm conditions
-TB

Signs & Symptoms
-Pyrosis
-Bitter or acid taste in mouth
-Choking
-Coughing
-SOB
-Frequent belching or hiccups
-Dysphagia
-Hematemesis
-Weight loss

Workup
-Barium swallow or endoscopy

Management
-Treat underlying condition
-Mechanical dilation
-PPI tx to avoid further stricturing
-Intralesional steroid injection for refractory strictures



Esophageal Varices

-Dilated submucosal veins in the lower esophagus as a result of portal HTN

Risk Factors
-Portal vein thrombosis
-Liver disease: alcoholic, viral hepatitis, etc.

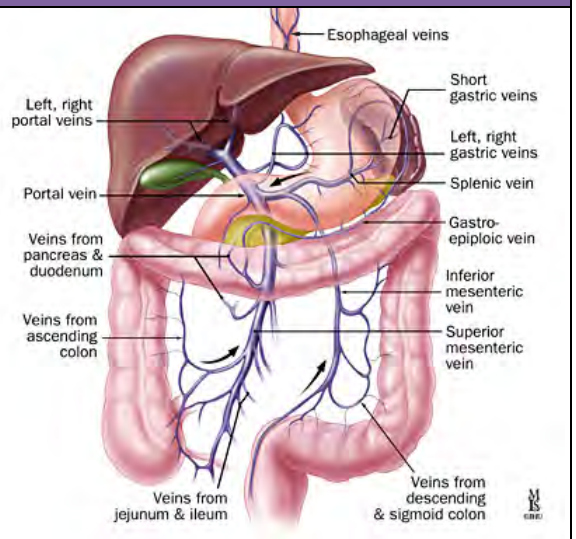
Prevention
-Pts with cirrhosis should have diagnostic endoscopy to screen for varices based on Child score, may need prophylaxis with β-blocker or ligation

Signs & Symptoms
-If ruptured → hematemesis, melena, hematochezia, dizziness
-Sx of cirrhosis and portal HTN

Workup
-Labs will show typical values of liver dz: ↑LFTs and bili, ↓albumin and cholesterol, ↑coags
-Endoscopy is test of choice for diagnosis

Management
-Octreotide (or vasopressin + NG) to ↓ portal vein inflow
-Endoscopic therapy is treatment of choice: sclerotherapy or band ligation
-TIPS procedure for failed endoscopic therapy
-For variceal hemorrhage: pRBCs as needed

Prognosis
-Bleeding varices resolve spontaneously half the time
-Continued bleeding associated with increased mortality



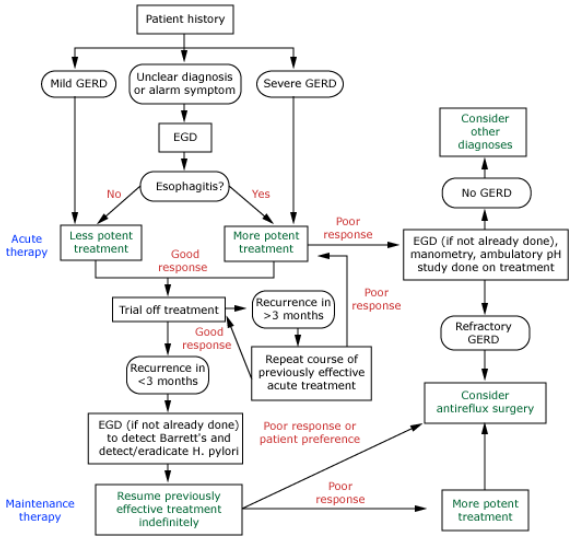
STOMACH

Gastroesophageal Reflux Disease (GERD)

-Common cause is impaired LES function
-Can be associated with hiatal hernia
-Severity of symptoms does not correlate with tissue damage

Signs & Symptoms
-Heartburn 30-60 minutes after meals and/or on reclining
-Regurgitation of gastric contents
-Hoarseness
-Loss of dental enamel
-Relief with antacids
-Alarm symptoms: dysphagia, odynophagia, weight loss, iron deficiency anemia, symptom onset after age 50, symptoms persistent despite PPI therapy → refer for urgent workup and upper endoscopy
-Asthma

Differential
-PUD
-Gastritis
-Non-ulcer dyspepsia
-Cholelithiasis
-Angina pectoris
-Infectious esophagitis: *Candida*, CMV, HSV
-Pill esophagitis
-Esophageal motility disorder
-Radiation esophagitis
-Gastrinoma
-Delayed gastric emptying



Management
-Mild, intermittent symptoms → lifestyle modifications like elimination of triggers, weight loss, avoid lying down after meals, elevate HOB; PRN antacids
-Oral H2 agonists have a 30 minute delay
-Troublesome frequent symptoms → PPI for 4-8 weeks, then consider chronic PPI use if symptoms relapse or persist
-Known GERD complications → long-term PPI therapy
-If unresponsive to PPI → verify drug compliance, refer for endoscopy
-Antireflux surgery such as Nissen fundoplication is last resort

Complications
-Barrett esophagus occurs in 10% of patients with chronic reflux → screening EGD recommended for adults 50 or older with 5-10 year history of GERD, and EGD q 3 years for patients with known Barrett
-Peptic strictures → progressive solid food dysphagia
-Esophagitis
-Asthma, laryngitis, or chronic cough

Gastritis

Differential
-PUD
-Nonulcer/functional dyspepsia
-GERD
-Hiatal hernia
-Biliary disease
-Pancreatitis
-Gastric or pancreatic cancer
-Eosinophilic gastritis
-Hypertrophic gastropathy
-Viral
-Indigestion
-Angina pectoris
-Severe pain → esophageal rupture, gastric volvulus, ruptured AA
-Erosive gastritis s/p NSAIDs or other meds, alcohol, stress due to severe medical or surgical illness, portal HTN
-Pernicious anemia gastritis
-Larval infection from raw fish or sushi

Workup
-Urgent EGD for alarm symptoms: severe pain, weight loss, vomiting, GIB, anemia

Management
-PPIs are superior to H2 blockers for treating NSAID-related dyspepsia and healing related ulcers
-Propranolol for portal HTN
-*H. pylori* eradication therapy

Gastric Neoplasms

Benign	Malignant	
Gastric Polyps -Many can undergo malignant transformation so these should be biopsied **Others** -Lipoma -Fibroma -Glomus tumor -Hemangioma	**Leiomyosarcoma** -Arises from smooth muscle -Rare -Requires local resection, rarely metastasizes **Lymphoma** -Either MALT or diffuse large B cell lymphoma **Sarcoma** -GIST	**Adenocarcinoma** -High incidence in Korea, Japan, and China -Usually occurs after age 60 -Risk factors: pickled foods, salted foods, smoked foods, *H. pylori*, atrophic gastritis, polyps, radiation -Signs & symptoms: early disease is asymptomatic; indigestion, nausea, early satiety, anorexia, weight loss, palpable stomach, hepatomegaly, pallor, Virchow's nodes (L sided supraclavicular) or Sister Mary Joseph nodes (umbilical); advanced: pleural effusions, SBO, bleeding -Workup: EGD preferred, endoscopic US to assess tumor depth, barium swallow, CT of pelvis, chest, and abdomen to look for mets -Management: depends on stage; resection (Billroth or roux-en-Y), chemo, radiation, adjuvants if needed -Prognosis: difficult to cure, most will die of recurrent disease even after resection

Peptic Ulcer Disease

Causes
-*H. pylori*
-Chronic NSAIDs
-Excess acid
-Cigarette smoking
-CMV in transplant pts
-Crohn's

Prevention
-Prevent with cox-2 inhibitors vs NSAIDs, PPIs, high dose H2 blockers, misoprostol if must use NSAIDs

Risk Factors
-Older age
-Anticoagulation
-Steroids
-NSAIDs
-Chronic disease

Signs & symptoms
-Gnawing, dull, or aching pain localized to the epigastrium that is non-radiating
-Gastric ulcers are worse after meals
-Duodenal ulcers are better after meals
-Pain frequently awakens patients from sleep between 2-3 am
-May be asymptomatic

Workup
-Barium upper GI series films are less sensitive
-EGD with gastric biopsy for definitive diagnosis
-*H. pylori* test: fecal or urea breath test preferred over antibody test

Treatment
-*H. pylori* eradication with triple therapy and confirmation via stool or urease breath test (beware, resistance is on the rise!)
-Bland diet NOT shown to be helpful
-Quit smoking as it retards ulcer healing
-H2 blockers block 65% of acid secretion and can heal ulcers in 6-8 weeks
-PPIs block 90% of acid secretion and can heal ulcers in 4-8 weeks, preferred agent

Sequelae
-PUD is the #1 cause of upper GIB

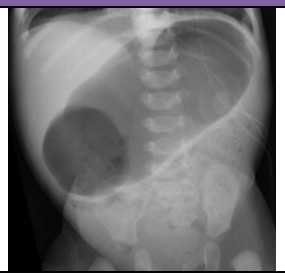
Pyloric Stenosis

-Typically in 3-6 week olds, usually firstborn males
-Rare after 12 weeks

Signs & symptoms
-Projectile nonbilious vomiting
-Ravenous hunger
-Palpable pyloric olive
-Poor weight gain
-Visible peristaltic waves

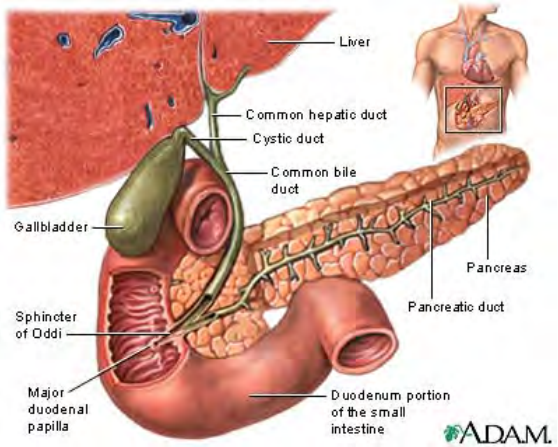
Workup
-KUB showing "caterpillar sign" of distended, hypertrophic stomach
-US showing thickened stomach muscle (preferred imaging)

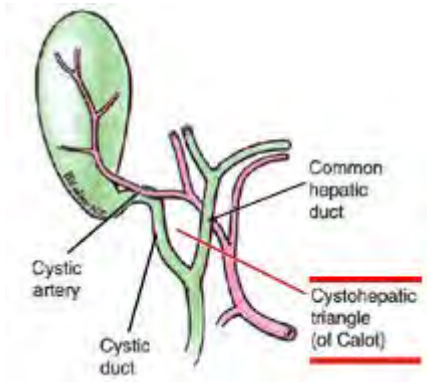
Management
-Refer for surgical pyloromyotomy

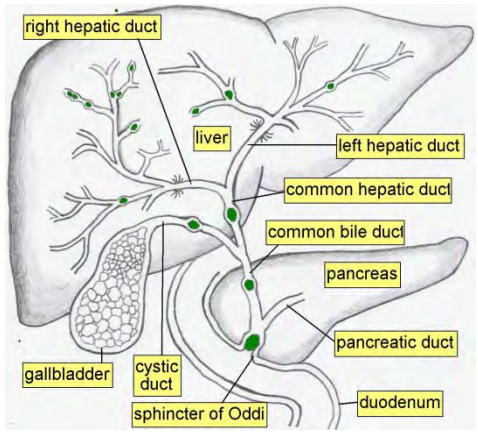


GALLBLADDER

Biliary Disease







Biliary Disease	Info	Risk Factors	Signs & Symptoms	Differential	Workup	Management	Complications
Cholelithiasis	-Most stones are cholesterol, fewer are pigment stones	-Obesity -Rapid weight loss -DM -High carb diet -Crohn's disease -High TG -Pregnancy -Hemolytic anemia -Cirrhosis -Estrogens -TPN -Native American	-Frequently asymptomatic and discovered incidentally -Biliary colic: infrequent episodes of steady severe pain in epigastrium or RUQ with radiation to right scapula (Boas' sign) -May be ppt by large or fatty meal	-Perforated peptic ulcer -Acute pancreatitis -Appendicitis with high-lying appendix -Perforated colonic carcinoma -Liver abscess -Hepatitis -Pneumonia with pleurisy	-RUQ US	-Lap chole is surgical treatment of choice -Nonsurgical candidates: dissolution therapy, shockwave lithotripsy, percutaneous stone removal or drain placement	-Bile peritonitis -Subhepatic abscess -Gallstone pancreatitis -Pancreatitis s/p endoscopic sphincterotomy
Cholecystitis	-Usually due to stone lodged in cystic duct (but can be acalculous) → secondary bacterial infection (usually *Klebsiella* or *E. coli*)		-Severe pain and tenderness in right hypochondrium or epigastrium -N/v -Fever -Murphy's sign, guarding, rebound tenderness		-CBC shows leukocytosis -↑ serum bili, AST/ALT, ALP Amylase may be ↑ -Cholecystitis: US is initial test of choice -HIDA if suspecting acalculous cholecystitis -Choledocholithiasis: endoscopic US or MRCP are tests of choice -Cholangitis: ERCP or abdominal US shows common bile duct dilation		
Choledocholithiasis	-Stone has traveled to common bile duct		-Can be jaundiced -Referred shoulder pain not commonly seen -Dark urine or light stools with choledocholithiasis			-Endoscopic sphincterotomy to allow stone passage through sphincter of Oddi	
Ascending Cholangitis	-When choledocholithiasis progresses to infection -Consider in any pt with h/o biliary disease who presents with recurrent symptoms -Mortality of 50%		-Charcot's triad: fever, RUQ pain, jaundice -Reynold's pentad: Charcot's + confusion and hypotension			-Zosyn or ceftriaxone + metronidazole -Biliary drainage via ERCP	

Biliary Cancer

-90% are fatal
-Pts are usually asymptomatic
-Endoscopic US is imaging of choice for workup

Risk Factors
-Gallbladder polyps
-Gallstone disease
-Congenital biliary cysts
-Anomalous pancreaticobiliary junction
-Chronic infection
-Porcelain gallbladder

Cholangiocarcinoma
-Arises in bile ducts
-Risk factors: primary sclerosing cholangitis, choledochal cysts, *Clonorchis sinensis* infection
-S/s: Courvoisier's sign (palpable nontender gallbladder + jaundice)

Gallbladder Adenocarcinoma
-Accounts for 80% of gallbladder malignancies
-S/s: RUQ pain, weight loss, anorexia, nausea, obstructive jaundice, ascites
-Management: surgical resection

Other Malignancies
-SCC
-Neuroendocrine tumors
-Lymphoma
-Sarcoma

LIVER

Cirrhosis

Etiologies
-Alcoholic: average consumption of 8 12 oz beers, 1 L wine, or ½ pint of spirits per day for 20 years
-Non-alcoholic: a result of chronic inflammation

Signs & symptoms
-Portal HTN
-Ascites: + fluid wave, shifting dullness
-Gastro-esophageal varices
-Splenomegaly → thrombocytopenia
-Encephalopathy from lack of toxin clearance

Workup
-High INR and low albumin from decreased ability to make proteins
-Elevated conj. bili due to inability of liver to process bilirubin, with eventual unconj bili ↑
-US to check for ascites and portal vein thrombosis
-Diagnostic paracentesis to determine true ascites vs. bacterial peritonitis (true ascites is a difference between serum albumin and peritoneal fluid albumin > 1.1)
-Liver biopsy
-Screen for varices with EGD

Management
-For ascites: salt restriction, diuretics, therapeutic paracentesis, TIPS if refractory, AB for secondary infection
-For encephalopathy: lactulose to reduce ammonia
-Treatment of viral hepatitis if present
-Alcoholic abstinence
-Hep A and B immunization
-Transplant

Prognosis
-Variable depending on comorbidities and etiology
-Child-Pugh classification estimates 1-year survival rates
-MELD score used for liver transplant evaluation

Measure	1 point	2 points	3 points
Total bilirubin, µmol/l (mg/dl)	<34 (<2)	34-50 (2-3)	>50 (>3)
Serum albumin, g/l	>35	28-35	<28
PT INR	<1.7	1.71-2.30	> 2.30
Ascites	None	Mild	Moderate to Severe
Hepatic encephalopathy	None	Grade I-II (or suppressed with medication)	Grade III-IV (or refractory)

Points	Class	One year survival	Two year survival
5-6	A	100%	85%
7-9	B	81%	57%
10-15	C	45%	35%

Hepatitis

Etiologies
-Most are due to the hepatitis viruses
-Toxins: alcohol
-Meds
-Industrial organic solvents
-Other infections
-Autoimmune disease
-NASH

Signs & symptoms
-Acute: malaise, myalgias and arthralgias, fever, n/v/d, headache, anorexia, dark urine, scleral icterus, abdominal pain, tender hepatomegaly, lymphadenopathy, splenomegaly
-Chronic: malaise, weakness, cirrhosis symptoms if severe

Type	A	B	C	D	E
Source	Fecal-oral	Blood & assoc fluids, parenteral, sex, , dialysis, tattoing	Blood & assoc fluids, parenteral, sex, IVDU, transfusions	Blood & assoc fluids	Fecal-oral
Incubation	28 days	45-180 days	2-26 weeks		
Transmission	Fecal-oral	Percutaneous/mucosal Transplacental	Percutaneous/mucosal	Percutaneous or mucosal	Fecal-oral
Prevention	Immunization, esp for travelers, MSM, drug users, chronic liver disease	Immunization of all infants, adolescents and adults in high risk groups, perinatal prevention	Blood donor screening, don't share needles, barrier protection during sex	Immunization for HBV	Safe drinking water
Presentation	ACUTE RUQ pain, n/v	CHRONIC in 5% unless you clear it	Usually no acute flare, just becomes chronic Silently progressive	CHRONIC	ACUTE
Investigation	↑ ALT/AST + IgM if acute + IgG if prior/vacc	+ surface Ag with active infection + surface AB with previous infect/vacc + core AB with active or prior infect (NOT vacc) + E Ag with active viral replication + E AB in chronic infect w/o replication, + blood DNA in infection	+ AB in present or previous infection + RNA in active infection	+ AB in present or previous infection + RNA in active infection	+ AB
Treatment		IFN, antivirals Usually clears spontaneously	Type 1 → direct antivirals, pegylated IFN, ribavirin Type 2 or 3 → pegylated IFN, ribavirin		Benign and self-limiting
Chronic infection?	No	Yes, especially in kids under 5	Yes in 70%	Yes	No
Special notes	"Infectious hepatitis" Complications of fulminant hepatitis, cholestatic hepatitis Prevalent in Alaska natives, American Indians	"Serum hepatitis" Chronic increases risk for cirrhosis and HCC	#1 cause for liver transplant 6 genotypes, with 1 most common and hardest to treat Liver biopsy useful for staging chronic	Requires coinfection with hep B	Increased severity in pregnant women Rare in US Endemic in India, Mexico, Iraq, North Africa, etc.

Test Result	Interpretation
HBsAg (—) Total anti-HBc (—) anti-HBs (—)	Susceptible
HBsAg (—) Total anti-HBc (+) anti-HBs (+)	Immune due to natural infection
HBsAg (—) Total anti-HBc (—) anti-HBs (+)	Immune due to hepatitis B vaccination
HBsAg (+) Total anti-HBc (+) IgM anti-HBc (+) anti-HBs (—)	Acutely infected
HBsAg (+) Total anti-HBc (+) IgM anti-HBc (—) anti-HBs (—)	Chronically infected
HBsAg (—) Total anti-HBc (+) anti-HBs (—)	Four interpretations possible 1. Recovering from acute HBV infection 2. Distantly immune and test not sensitive enough to detect very low level of serum anti-HBs 3. Susceptible with a false positive anti-HBc 4. Chronic HBV infection with rare circumstance where HBV does not produce detectable HBsAg

Liver Neoplasms

-Most pts are asymptomatic
-Diagnostic approach depends on risk factors of patient and size of lesion
-Workup: ↑AFP indicates malignancy

Benign

Hemangioma
-The most common benign liver tumor
-Small, asymptomatic
-Finding is incidental

Hepatic adenoma
-Associated with long-term estrogen use
-Can rupture and bleed, so it should be resected

Focal nodular hyperplasia
-May be a response to a congenital malformation
-Should be resected

Others
-Hamartoma
-Cysts: simple, infectious, polycystic liver, biliary cystadenoma, Von Meyenburg complex

Malignant

-Risk factors: EtOH, autoimmune hepatitis, viral hepatitis, alpha-1 antitrypsin deficiency, Wilson's disease

Hepatocellular carcinoma
-Usually occurs with chronic liver disease or cirrhosis
-High risk pts should be screened every 6 months via US
-Heightened suspicion for malignancy in previously compensated cirrhosis pts who develop decompensation
-Lab findings will be nonspecific, but baseline AFP will rise
-Diagnostic imaging shows multiphasic tumor
-Treat by resection or radiofrequency ablation, palliative embolization, or liver transplant

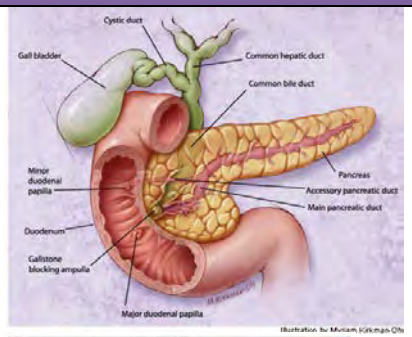
Metastatic Disease
-The most common malignant hepatic neoplasms in the Western world

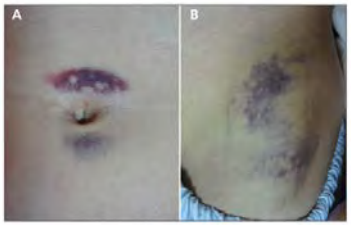
PANCREAS

Acute Pancreatitis

-Occurs with inappropriate activation of trypsin within the pancreas → enzymatic damage to the pancreas

Etiologies
-Gallstones are the most common cause
-Alcohol use
-Other obstructions: pancreatic or ampullary tumors, sphincter of Oddi dysfunction, pancreatic malformation
-Meds: diuretics, azathioprine, 6-mercaptopurine, sulfa drugs, ACEIs, HIV meds
-Infections: mumps, rubella, Coxsackie, echovirus, EBV, HIV
-Metabolic: ↑TG, hyperCa
-Toxins: methanol, ethanol, scorpion sting in Trinidad
-Vascular: vasculitis, ischemia
-Abdominal trauma
-Post-ERCP
-Inherited causes





Signs & symptoms
-Range of severity from mild illness to severe multiorgan failure
-Constant epigastric pain radiation to the back
-Nausea and vomiting
-Tachycardia secondary to hypovolemia from leaky vessels and 3rd spacing
-Fever
-Sepsis
-Icterus or jaundice if there is biliary obstruction
-Abdominal tenderness with rigidity and guarding
-Acute interstitial pancreatitis: mild, with pancreatic edema

-Acute necrotizing pancreatitis: severe, with necrosis of parenchyma and vessels → Gray-Turner's sign and Cullen's sign

Differential
-Acute cholecystitis or cholangitis
-Penetrating duodenal ulcer
-Ischemic colitis
-SBO
-AAA
-Nephrolithiasis
-Pancreatic pseudocyst

Workup
-↑ Amylase: not specific, can be ↑ in appendicitis, cholecystitis, perf, ectopic pregnancy, or renal failure; elevated for 24 hours
-↑ Lipase: more specific for pancreatitis, but can be elevated in renal failure; stays elevated for 3 days
→ Elevated amylase or lipase alone without clinical signs are NOT pancreatitis!
→ Amylase/lipase #s DON'T correlate to severity of disease!
-Bili will be elevated if there is an obstruction blocking it from leaving the liver
-Elevated BUN and hct with vol depletion
-US showing large, hypoechoic pancreas
-CT showing pancreatic enlargement and peripancreatic edema (imaging of choice for pancreatitis)
-MRCP or ERCP

Management
-If mild → NPO with IFV, correction of electrolytes, pain control; resolves in 3-7 days
-Severe → ICU monitoring, early NGT with tube feeds
-Acute necrotizing pancreatitis → imipenem
-Gallstone pancreatitis → sphincterotomy if suspecting risk of cholangitis, otherwise plan for lap chole after recovery

Prognosis
-Complications: inflammatory cascade can cause ARDS, sepsis, or renal failure; pancreatic necrosis or abscess, pancreatic pseudocyst

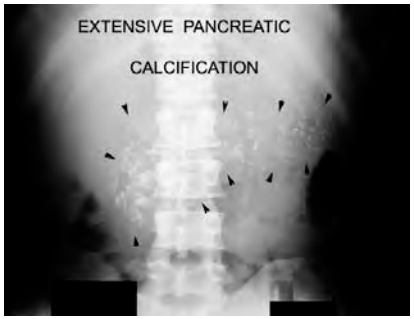
Chronic Pancreatitis

-Chronic inflammation leads to irreversible fibrosis of the pancreas

Etiologies
-Chronic alcohol use
-Chronic pancreatic duct obstruction
-Malnutrition
-Autoimmune
-Hereditary
-Idiopathic

Signs & symptoms
-Recurrent episodes of epigastric and LUQ pain
-Steatorrhea
-Fat soluble vitamin deficiency
-Diabetes

Workup
-Amylase and lipase won't be elevated because the pancreas is burned out by now
-Secretin stimulation test to see if the pancreas still works
-Abdominal x-ray showing pancreatic calcifications
-CT showing calcifications and atrophy
-ERCP showing "chain of lakes" or areas of dilation and stenosis along the pancreatic duct



Management
-Abstinence from alcohol
-Pancreatic enzyme replacement + PPI + low fat diet
-Insulin
-Surgical options for refractory cases: decompression, resection, or denervation procedures

Pancreatic Neoplasms

Benign

-Usually asymptomatic and found incidentally
-Eval further using MRI, endoscopic US with FNA

Serous Cystadenoma
-Most common benign pancreatic lesion
-Low malignancy potential
-Resection not recommended

Mucinous Cystadenoma
-Moderate malignancy potential

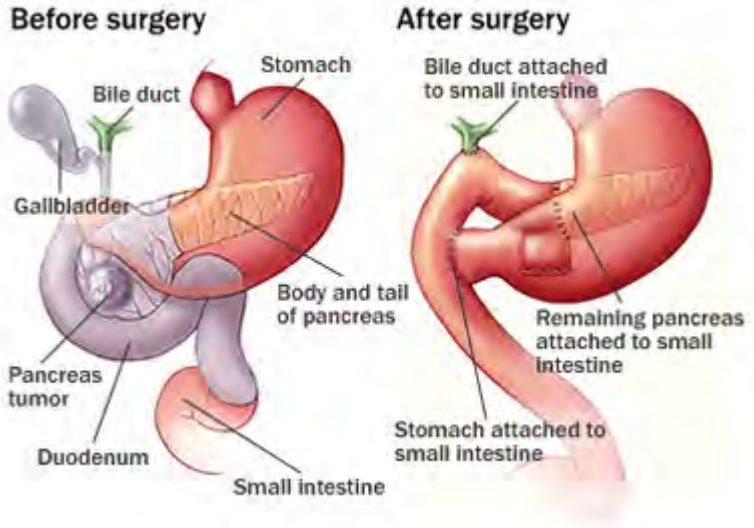
Intraductal Papillary Mucinous Neoplasm
-High malignancy potential if located within main duct

Solid Pseudopapillary Neoplasm
-Low to moderate malignancy potential
-Should be resected

Malignant

-Resection via Whipple procedure

Ductal Adenocarcinoma
-Includes signet ring cell carcinoma, adenosquamous carcinoma, undifferentiated carcinoma, and mucinous non-cystic carcinoma
-Accounts for > 95% of pancreatic malignancies
-Typically occurs in ages 70-80
-Most commonly in the head of the pancreas
-Risk factors: tobacco use, chronic pancreatitis, exposure to dye chemicals, DM2 in nonobese person arising after age 50, history of partial gastrectomy or cholecystectomy, genetic factors including BRCA2
-Signs & symptoms: pain, steatorrhea, weight loss, jaundice, Courvoisier's sign (palpable gallbladder due to compression of bile duct), Trosseau's sign (hypercoagulable state created by the malignancy → migratory thrombophlebitis throughout body)
-Workup: ALP, bili, initial imaging with RUQ US and ERCP, CT for "double duct sign" (dilation of the common bile and main pancreatic ducts), endoscopic US if other imaging is not convincing, confirmation with histologic diagnosis
-Management: serial CA-19-9 to follow trend; surgical resection + radiation (if there is no invasion, lymphatic involvement, or mets), locally advanced disease → radiation only; chemo, pain control, and palliative stents for metastatic disease
-Prognosis: half of all pancreatic cancers are metastatic by the time of diagnosis, with a life expectancy of 3-6 months; resectable disease survival is < 1.5 years; locally advanced disease survival is 6-10 months



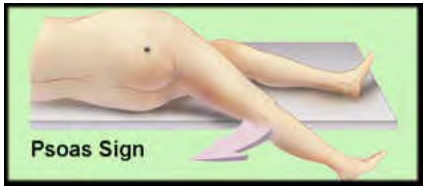
Others
-Acinar cell carcinoma
-Pancreatoblastoma

SMALL INTESTINE, COLON, AND RECTUM

Appendicitis

-Usually caused by a fecalith

Signs & Symptoms
-Dull periumbilical pain that progresses to focal sharp pain with RLQ radiation
-Anorexia, n/v
-Low grade fever
-McBurney's point tenderness
-Obturator sign
-Psoas sign
-Rovsing's sign



Workup
-CBC may show leukocytosis (but can be late finding)
-Abdominal CT preferred in adults and nonpregnant women
-US preferred in peds and pregnancy
-If probability for appendicitis is high, can go straight to surg consult in many cases

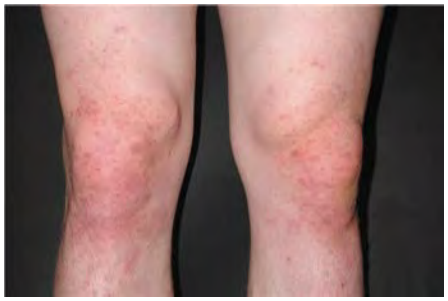
Management
-Surgical consult
-Presence of abscess or rupture will require cipro or Zosyn treatment first followed by appendectomy in 3-4 weeks in kids or colonoscopy in adults (colon ca can manifest as appendiceal abscess)

Celiac Disease

-Immune-mediated inflammation of small intestine due to gluten sensitivity (wheat, barley, rye, ?oats)
-Found primarily in white/European patients
-Genetic factors

Screening
-Serologies recommended for pts with failure to thrive, chronic diarrhea, chronic constipation, recurrent abdominal pain, dental enamel hypoplasia, idiopathic short stature, pubertal delay, iron deficiency anemia not responsive to supp
-Serologies also recommended for 1st degree relatives of pts with celiac, autoimmune thyroiditis, DM1, Down's, Turner's, Williams, IgA deficiency, and autoimmune hepatitis

Signs & symptoms
-Malabsorption: diarrhea, steatorrhea, weight loss, vitamin deficiencies
-Majority have only minor GI complaints or can be asymptomatic
-Secondary hyperparathyroidism from vitamin D deficiency
-Comorbid autoimmune dz: dermatitis herpetiformis, DM1, thyroiditis



Workup
-Can have false neg IgA serology as many celiac pts are IgA deficient
-Definitive dx is endoscopy with small bowel biopsy showing villous atrophy
-Screen for skin dz, DM1, thyroiditis, bone density
-Positive serology with negative biopsy may indicate latent celiac disease

Management
-Gluten-free diet
-Repeat bowel biopsy if dx is uncertain
-Pneumovax as celiac is associated with hyposplenism
-Latent celiac disease should be f/u with repeat monitoring and treated if symptoms develop

Adult Constipation

-May refer to < 3 stools per week, difficult-to-pass stools, sense of incomplete evacuation, abdominal distension, bloating, or pain
-Generally considered to be a functional disorder with 3 subtypes: slowed colonic transit, obstructive defecation, constipation-predominant IBS

Differential
-Most common: low fiber, sedentary activities, inadequate fluid intake
-Systemic: endocrine (hypothyroid, hyperparathyroid, DM), metabolic (hypokalemia, hypercalcemia, uremia, porphyria), neuro (Parkinson, MS, sacral nerve damage, paraplegia, autonomic neuropathy)
-Meds: opioids, diuretics, CCBs, anticholinergics, psychotropics, Ca and Fe, NSAIDs, clonidine, cholestyramine
-Structural: anorectal, perineal descent, colonic mass, colonic stricture, Hirschsprung
-Slowed transit: idiopathic, psychogenic, eating disorder, chronic pseudoobstruction
-Pelvic floor dyssynergia
-IBS

Workup
-Rectal and abdominal exam
-Alarm symptoms → further workup via CBC, TSH, BMP, referral for EGD or flex sigmoidoscopy

Treatment:
-Increase fiber gradually
-Increased # of daily meals
-Laxatives intermittently or chronically for constipation unresponsive to lifestyle change

Pharmacologic Therapies

Fiber laxatives
-First-line therapy for constipation
-Bran powder: may cause gas
-Psyllium (Metamucil, Perdiem)
-Methylcellulose (Citrucel)
-Ca polycarbophil (FiberCon): pill form
-Guargum (Benefiber)

Osmotic laxatives
-Onset within 24 hours
-MgOH (milk of magnesia, epsom salts)
-Sorbitol or lactulose
-Polyethylene glycol (Miralax)
-Sodium phosphate (Visicol, OsmoPrep, Fleet's): not to be used in pts over 55, kidney disease, or pts taking meds affecting kidney function

Stimulant laxatives
-Onset in 6-12 hours if oral or 15-60 min if rectal
-Bisacodyl: cramping, avoid daily use
-Senna (ExLax, Senekot): cramping, avoid daily use
-Cascara (Nature's Remedy)
-Lubiprostone: category C, expensive

Opioid-receptor antagonists
-Effectively blocks peripheral opioid receptors without affecting central analgesia = good option for patients on chronic opioids
-Methylnaltrexone

Stool surfactants/emollients
-Not for constipation but to soften stool for patients who aren't supposed to strain
-Mineral oil
-Docusate sodium (Colace): marginal benefit

Pediatric Constipation

***Encopresis is managed similarly, only without the use of laxatives as long as constipation has been excluded as a cause

-Usually begins with an acute episode of constipation then is self-perpetuating as kids may hold stool to avoid painful BMs or going at school → chronic rectal distension → increased threshold for conscious need to defecate

Signs & symptoms
-Encopresis
-UTIs
-Chronic abdominal pain
-Poor appetite
-Lethargy
-Rectal skin tags

Differential
-Imperforate anus
-Hirschsprung disease repair
-Crohn's perianal disease
-Psychogenic
-Hypothyroidism
-Tethered cord
-Spina bifida
-Anterior displacement of the anus
-Intestinal pseudo-obstruction
-Cystic fibrosis
-Celiac
-Lead intoxication
-Botulism
-Cow's milk constipation

Workup
-Criteria: symptoms must be present for 1 month in toddlers and infants and 2 months in older children
-Labs only for kids not responding to an intervention program

Management
-Initial disimpaction with enema or Golytely (or lactulose or sorbitol-containing juices in infants) followed by maintenance with Miralax (if > 2 years old, but safety has also been demonstrated in infants)
-Adjust maintenance therapy to goal of 1 soft stool per day
-"Rescue plan" to use stimulant laxative, enema, or suppository if there are signs of constipation recurrence
-Behavioral modification with toileting regimen and bowel training → sit on toilet for 5-10 min after each meal, give sticker or game reward for each effort, record BMs and symptoms with log

Rome III criteria for the diagnosis of functional constipation in children

Infants and toddlers	Children with developmental age 4 to 18 years
At least two of the following present for at least one month	At least two of the following present for at least two months
Two or fewer defecations per week	Two or fewer defecations per week
At least one episode of incontinence after the acquisition of toileting skills	At least one episode of fecal incontinence per week
History of excessive stool retention	History of retentive posturing or excessive volitional stool retention
History of painful or hard bowel movements	History of painful or hard bowel movements
Presence of a large fecal mass in the rectum	Presence of a large fecal mass in the rectum
History of large-diameter stools that may obstruct the toilet	History of large-diameter stools that may obstruct the toilet

Diverticular Disease

Diverticulosis

-A 20th century disease associated with Western lifestyle of low fiber, red meat, obesity, and increasing age
-Diverticula are outpouchings of the mucosa and submucosa through the muscular layer of the colonic wall; they can become perforated and infected with constipation or increased intraluminal pressure

Signs & symptoms
-Many cases of colonic diverticula are asymptomatic and discovered incidentally
-Chronic constipation, abdominal pain, fluctuating bowel habits
-May have mild LLQ tenderness

Management
-May reduce complications of diverticulosis with fiber supplements

Diverticulitis

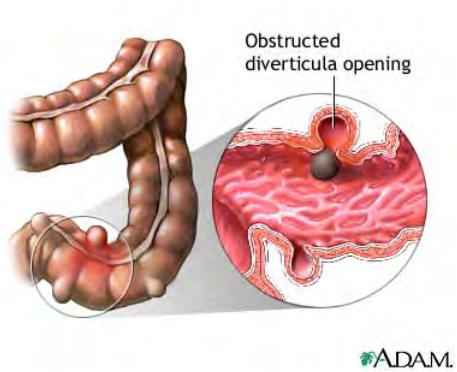
Signs & symptoms
-Mild to severe LLQ or suprapubic pain ± palpable mass
-Acute GIB that is painless and maroon in color
-Fever, malaise, constipation, diarrhea, cramping, bloating, nausea, vomiting, dysuria, and urinary frequency

Differential: perforated colonic carcinoma, Crohn's, appendicitis, ischemic colitis, C. diff, ectopic pregnancy, ovarian cyst, ovarian torsion

Workup
-CBC shows leukocytosis with left shift
-CT with contrast is imaging of choice to assess severity
-Plain films for free air, ileus, or obstruction

Management
-Uncomplicated/simple = empiric treatment with 7-10 days of outpatient antibiotics to cover aerobes and anaerobes (cipro + metronidazole), clear liquids diet until clinical improvement (2-3 days), surgical consult if no improvement or worsening of symptoms in 72 hours
-Refer for colonoscopy, CT colonography, or barium enema with flex sig 2-6 weeks after recovery to evaluate extent of diverticulosis and exclude malignancy (don't want to do this right away due to risk of causing perforation)
-Complicated (peritonitis, obstruction, perf, abscess, or fistula) = hospitalization with IV antibiotics (ampicillin, gentamicin, and metronidazole), IVF, pain management, and antiemetics

Prognosis
-Complications: lower GIB, intra-abdominal abscess or peritonitis secondary to perf, fistulas, obstruction
-30-40% of cases will have episodic abdominal cramps without frank diverticulitis
-30% of cases will go on to have a second attack of diverticulitis



Intussusception

-The most common cause of intestinal obstruction in infants < 1 year
-Most cases are between 6 months and 3 years of age
-Can occur multiple times

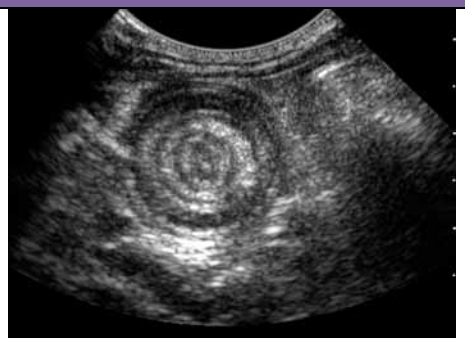
Etiologies
-Idiopathic: most cases
-Viral
-Underlying condition: Meckel diverticulum

Sings & symptoms
-Periodic colicky abdominal pain
-Vomiting
-Bloody "currant jelly" stools
-Palpable mass or "sausage" in RUQ
-Lethargy

Differential
-Malignancy if child is over 3

Workup
-Plain films showing SBO
-US showing "pseudokidney sign" or "lasagna sign" (test of choice)

Management
-Refer for emergent reduction via enema or surgical repair



Lactose Intolerance

-May be primary or secondary due to bacterial overgrowth, enteritis, Celiac disease, IBD, etc.
-High prevalence among Native Americans, patients of African descent, and Hispanics

Signs & symptoms
-Abdominal pain, bloating, farts, diarrhea, and possibly vomiting after ingestion of lactose

Management
-Avoid milk and ice cream as they have the highest amount of lactose
-Lactase supplementation (variable results)
-Add Lactaid to milk and let sit overnight before drinking
-Utilize yogurt or cheese for dietary calcium needs, or supplement

Inflammatory Bowel Disease

-Both are autoimmune
-Incidence highest in 15-40 year olds and > 60 year olds
-Tend to run in families
-Extraintestinal manifestations possible: eye (uveitis, episcleritis), skin (erythema nodosum, pyoderma gangrenosum), liver, joints
-Diagnosis relies on a combination of endoscopy, histology, radiography, labs, and clinical data
-Not everyone needs continued treatment or any treatment at all; treat the affected area
-Response to any given treatment is only 30-70%
-Use steroids sparingly to induce remission
-During flare, check WBCs, H/H, f/u with endoscopy referral if not improving

Crohn's Disease

-Can affect any portion of GI tract from lips to the anus and has transmural involvement, however most common site is ileum
-Disease skips areas → skip lesions
-Bouts of flares and periods of remission

Signs & symptoms
-Aggravated by smoking
-Fistulas and abscesses
-Perianal disease
-Obstructions
-Prolonged diarrhea and abdominal pain
-Fatigue
-Weight loss

Differential
-Ulcerative colitis
-IBS
-Appendicitis
-*Yersinia enterocolitica* enteritis
-Mesenteric adenitis
-Intestinal lymphoma
-Segmental colitis: ischemia, TB, amebiasis, *Chlamydia*
-Diverticulitis with abscess
-NSAID-induced colitis
-Perianal fistula: lymphogranuloma venereum, cancer, rectal TB

Workup
-Labs are not specific or reliable
-Initial imaging is upper GI series with small bowel follow-through
-Colonoscopy shows cobblestoning with varying degrees of mucosal ulceration

Management
-Steroids for flares
-Gentle wiping, sitz baths, perianal pads for perianal disease
-Low-roughage diet only for obstructive symptoms
-Mesalamine trials show that it is not effective for Crohn's
-Antibiotics during flares have shown little to no efficacy
-Steroid courses PRN: budesonide has fewer side effects
-Immunomodulating agents for pts unresponsive to steroids or requiring chronic steroids (refer to rheumatology): azathioprine, mercaptopurine, methotrexate
-Annual colonoscopy recommended with > 8 year disease history

Complications
-Small bowel strictures
-Fistulae to bowel, bladder, vagina, or skin
-High oxalate from malabsorption of ingested fat (binds Ca) → kidney stones, gallstones
-Often require surgical management

Ulcerative Colitis

-Disease begins in the rectum and is limited to the colon with superficial penetration of the mucosal wall
-Bouts of flares and periods of remission

Signs & symptoms
-Proctitis
-Tenesmus
-Lower abdominal or pelvic cramping
-Bloody diarrhea
-Mucus or pus per rectum
-Fever

Differential
-Infectious colitis: *Salmonella*, *Shigella*, *Campylobacter*, amebiasis, *C. diff*, enteroinvasive EC, CMV
-Ischemic colitis
-Crohn's disease
-Diverticular disease
-Colon cancer
-Antibiotic-associated diarrhea or pseudomembranous colitis
-Infectious proctitis: gonorrhea, *Chlamydia*, HSV, syphilis
-Radiation colitis or proctitis

Workup
-Labs: ↓ serum albumin, CBC for anemia, ↑ ESR
-Negative stool cultures
-Sigmoidoscopy with biopsies showing crypt abscesses, chronic colitis for dx
-Barium enema may show "stovepipe" colon due to loss of haustral folds

Management
-Distal colitis → DOC is topical mesalamine, hydrocortisone suppositories PRN, second-line therapy is oral sulfasalazine
-Mild-mod colitis (above sigmoid colon) → oral 5-ASAs, add hydrocortisone foam or enema if needed, refer for immunomodulating agents if no response
-Severe flare → send to ED for hospitalization
-Screening colonoscopies ever 1-2 years for patients with > 8 year history of disease

Complications
-Toxic megacolon
-Extension of colonic disease
-Perforation
-Strictures

Irritable Bowel Syndrome

-Chronic abdominal pain and altered bowel habits in the absence of any organic cause = functional
-Subtypes of constipation-predominant, diarrhea-predominant, and mixed
-Increased risk of developing after acute infectious gastroenteritis

Signs & symptoms
-Pain relieved with defecation
-More frequent stools at onset of pain
-Passage of mucus
-Bloating
-Sense of incomplete evacuation
-Urgency

Differential: dietary, infection, IBD, psychogenic, malabsorption, tumors, endometriosis

Workup
-Need to r/o IBD, infections, cancer
-FOBT
-CBC, CMP, ESR, serum albumin
-Consider TSH
-Celiac panel if diarrhea
-Manning and Rome criteria for diagnosis
-Red flags for something that is NOT IBS that need a colonoscopy: abnormal exam, fever, + FOBT, weight loss, onset in older patient, nocturnal awakening, low Hb, ↑WBCs, ↑ESR

Management
-Patients with elevated IgG can try elimination diets (lactose, gluten)
-Psychiatric eval for anxiety or depression
-Reassurance that there is no change in life expectancy although there is no cure
-Abdominal pain → antispasmodics, antidepressants
-Diarrhea → loperamide cautiously
-Constipation → bulking agents

Ischemic Bowel Disease

-Most cases are acute
-Risk factors: age, atherosclerosis, low cardiac output, arrhythmias, severe valvular disease, recent MI, intra-abdominal malignancy

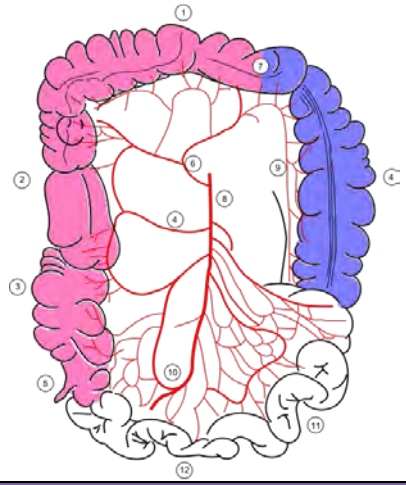
Etiologies
-Low blood pressure
-Clot
-Vasoconstriction
-Idiopathic

Signs & symptoms
-Diarrhea
-Fever
-Hyperactive phase: passage of bloody stool, severe abdominal pain
-Paralytic phase: diffuse abdominal pain, tender abdomen, bloating, no further bloody stools, absent bowel sounds
-Shock phase: fluids leaking through damaged colon lining → metabolic acidosis, dehydration, hypotension, tachycardia, confusion

Workup
-Mesenteric angiography is the gold standard
-Surgical consult

Management
-Restoration of intestinal blood flow
-Hemodynamic support
-Correction of metabolic acidosis
-Initiation of broad spec AB
-NGT for gastric decompression
-Bowel rest

Prognosis
-Most patients make a full recovery without sequelae



Colonic blood supply. Pink - supply from superior mesenteric artery (SMA) and its branches: middle colic, right colic, ileocolic arteries. Blue - supply from inferior mesenteric artery (IMA) and its branches: left colic, sigmoid, superior rectal artery. 7 is for so-called Cannon-Böhm point (the border between the areas of SMA and IMA supplies), which lies at the splenic flexure

Anal Fissure

-A tear or erosion in the epithelium of the anal canal
-Acute or chronic

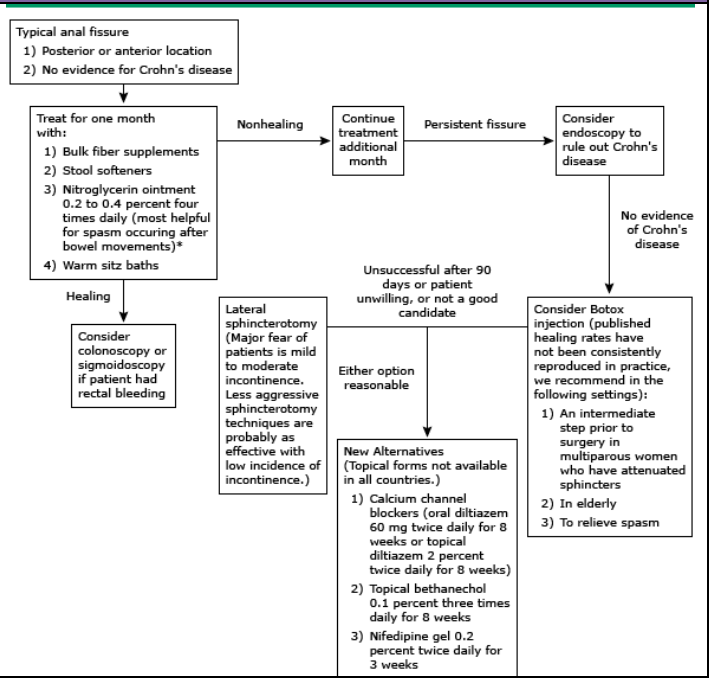
Causes
-Usually due to large or hard-to-pass stool
-Infectious: TB, syphilis, HIV, occult abscess
-Carcinoma
-Granulomatous disease
-IBD
-Prolonged diarrhea
-Anal sex
-Childbirth

Prevention
-Avoid constipation with high fiber and fluid intake
-Wiping with moist cloth

Signs and Symptoms
-Tearing pain with BMs, although less painful if chronic
-Small amount of bright red blood on toilet paper
-Usual location is posterior midline
-Perianal pruritus or skin irritation
-Acute fissures appear like a paper cut
-Chronic fissures usually have raised edges with external skin tags and hypertrophied pillae

Differential
-Perianal ulcer: IBD, TB, STDs
-Anorectal fistula: differentiate from fissure by tract formation

Management
-Stool softeners
-Sitz baths
-1% hydrocortisone cream
-2% nitroglycerine cream: ↑ blood flow and reduces pressure on internal anal sphincter
-Surgical consult if not improving in 6 weeks; possible need for internal sphincterotomy



Small Bowel Neoplasms

-Malignant tumors very rare when compared to incidence in large bowel
-Usually located in the ileum

Signs and Symptoms
-Crampy, intermittent abdominal pain
-Weight loss, nausea, vomiting
-GI bleed
-Intestinal obstruction
-Usually asymptomatic if benign

Risk Factors for Malignancy
-Familial cancer syndromes: HNPCC, Peutz-Jeghers, FAP
-Chronic inflammation: IBS, celiac disease
-Intake of alcohol, refined sugar, red meat, or salt-cured or smoked foods
-Smoking?
-Obesity?

Benign

Adenoma
-Villous adenomas can transform to malignancy
-Duodenal adenomas associated with increased risk for colon cancer
-Tubular adenomas most common in the duodenum

Leiomyoma
-Arise from intestinal submucosa

Lipoma
-Arise from submucosal or subserosal adipose

Other benign small bowel tumors: desmoid tumor, hemangioma, fibroma

Malignant

Adenocarcinoma
-Arises from glandular tissue
-Occurs in duodenum

Carcinoid Tumors
-A neuroendocrine cell tumor arising from the enterochromaffin cells of the gut
-Most common type of small bowel malignancy
-Usually occurs in the ileum
-With mets can have carcinoid syndrome: watery diarrhea, flushing, sweating, wheezing, dyspnea, abdominal pain, hypotension

Lymphoma
-Almost always non-Hodgkin
-Includes MALT lymphoma, diffuse large B-cell lymphoma, mantle cell lymphoma, and Burkitt lymphoma

Sarcoma
-Tumor of the mesenchymal cells
-Most common type is GI stromal tumor (GIST): may be considered benign but all have the potential for malignant transformation; should be resected if > 2 cm; consider imatinib (Gleevec) as neoadjuvant prior to resection if large; rarely metastasizes

Colorectal Neoplasms

Benign

Non-Neoplastic Polyps
-Hyperplastic polyps = not pre-malignant → more frequent screening not needed
-Hamartomatous polyps
-Inflammatory polyps
-Lymphoid polyps

Neoplastic Epithelial Polyps
-These are pre-malignant → need screens more frequently for monitoring
-Tubular adenoma
-Tubulovillous adenoma
-Villous adenoma

Leiomyoma
-Tumor of smooth muscle
-Can occur in colon or rectum

Others
-Lipoma
-Neuroma
-Hemangioma
-Lymphangioma

Malignant: Adenocarcinoma

-Accounts for 95% of primary colon cancers
-30% will be in the rectum, 25% on the right colon
-Risk factors: age, FH (up to 30% have a genetic component), DM2, metabolic syndrome, ethnicity, IBD, high red meat or processed meat consumption, inactivity, obesity, smoking, heavy alcohol use
-Prevention: diet with plant foods, healthy BMI, limited red meats, physical activity

Associated Familial Syndromes
-FAP: also incurs risk of thyroid, pancreas, duodenal, and gastric cancers
-HNPCC: associated with endometrial, ovarian, gastric, urinary tract, renal cell, biliary, and gallbladder cancers
-Most occur after age 50

Screening
-Begin assessing risk at age 20
-Begin screening at 40-45 for AA patients, at 50 for all other patients of average risk; continue until life expectancy is estimated to be less than 10 years or 85 years at the latest
-Begin screening those with FH at least 10 years before the age at which the youngest affected family member was diagnosed
-Colonoscopy every 10 years
-CT colonography or flexible sigmoidoscopy every 5 years
-FOBT annually for patients in whom imaging or visualization is not possible

Signs and symptoms
-Rectal bleeding
-Iron deficiency anemia
-Fatigue and weight loss
-Obstruction
-Change in stool quantity or caliber
-Abdominal mass or pain
-Weakness
-Mets to the liver and lung

Workup
-Colonoscopy for biopsy
-Abdominal/pelvis CT for staging ("apple core" lesions)
-CXR for mets
-Labs: CBC, CMP, baseline CEA for f/u
-PET

Management
-Early stage tumors may be removed endoscopically
-Hemicolectomy with lymph node dissection
-Local treatment of mets
-Chemo to eradicate micromets
-Radiation not typically used due to its high toxicity in the gut

Bowel Obstruction

-Obstruction can be mechanical (intrinsic: post-op) or functional (paralytic: electrolyte abnormality, DM)

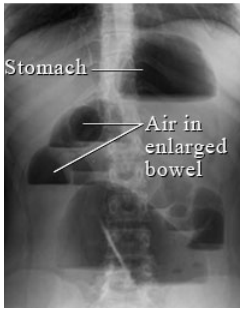
Types
-Simple obstruction = blood supply intact
-Strangulated obstruction = compromised blood supply
-Closed loop
-Obstruction can be complete, partial, or intermittent

Causes of Large Bowel Obstruction
-#1 is neoplasms
-Diverticular disease
-Volvulus: usually sigmoid or cecal
-Adhesions

Causes of Small Bowel Obstruction
-#1 cause is adhesions from previous surgeries
-Hernias
-Neoplasm
-Strictures
-Intussusception
-Meckel's diverticulum
-Volvulus
-Intramural hematoma

Signs & Symptoms
-Crampy, generalized abdominal pain
-No signs of peritonitis
-Abdominal distension with diffuse midabdominal tenderness to palpation
-Suspect ischemia with localized TTP
-Nausea
-Vomiting, may have coffee-ground emesis or feculent material
-Reduced urine output
-Inability to pass gas
-However, pts may still be passing gas and having flatus up to 12-24 hours after onset of obstruction, since the colon requires this much time to empty distal to the obstruction

Differential
-Paralytic ileus: occurs post-op or after peritonitis (will see dilated small bowel in presence of dilated colon on KUB)
-Intestinal pseudo-obstruction: recurrent abdominal distension in the setting of no mechanical obstruction
-Gastric outlet obstruction
-Intestinal malrotation



Workup
-BMP shows electrolyte derangements from fluid shifts
-Check lactate if concerned for bowel strangulation or ischemia
-Initial imaging with KUB shows distended loops of small bowel, air-fluid levels, free air under diaphragm if perforated, "swirl sign" where bowel has twisted on its mesentery, and "bird's beak" or "corkscrew" if volvulus is also present
-Can f/u KUB with CT for further localization
-SBO in absence of prior abdominal surgery should trigger malignancy workup

Management
-IVF
-Antibiotics
-NPO with NGT decompression
-Volvulus: rectal tube for decompression followed by surgical repair to prevent recurrence
-Ischemia or perforation: immediate surgical intervention

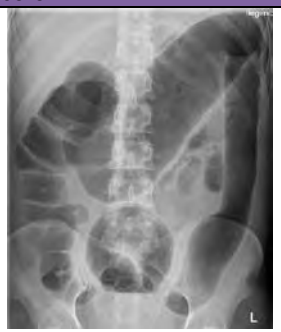
Toxic Megacolon

-A potentially lethal complication of colitis that is characterized by total or segmental nonobstructive colonic dilation + systemic toxicity

Etiologies
-IBD
-Infectious colitis
-Ischemic colitis
-Volvulus
-Diverticulitis
-Obstructive colon cancer

Signs & symptoms
-Severe bloody diarrhea

Workup
-Abdominal plain film showing R colon dilation > 6 cm, dilation of transverse colon, absence of normal colonic haustral markings, and air-filled crevices between large pseudopolypoid projections extending into the gut lumen



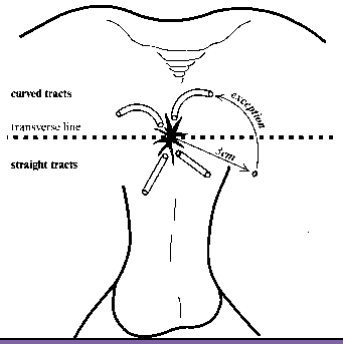
Management
-Fluid resuscitation
-Correction of abnormal labs
-IV vanco and metronidazole
-Complete bowel rest
-Bowel decompression with NGT
-Surgical consult for subtotal colectomy with end-ileostomy for pts not improving on medical management

Rectal Abscess

-Most arise from infected anal glands

Signs & symptoms
-Severe anal or rectal pain
-Fever or malaise
-Area of fluctuance or patch of erythema may be visible externally or many only be palpable on DRE
-Fistula may form with non-healing anorectal abscess

Workup
-Pelvic MRI for nonvisible nonpalpable abscess
-Use Goodsall's rule to assess tract location if fistula is suspected

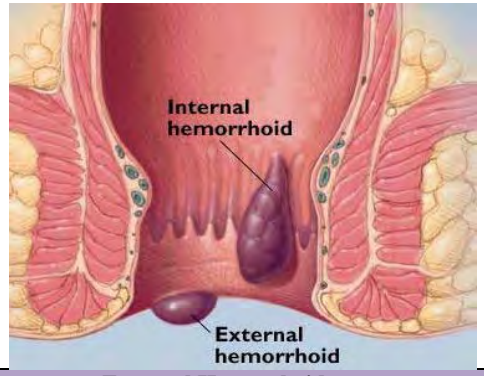


Management
-I&D with culture for pts needing abx or with pain out of proportion to clinical findings
-Complicated abscess location may need OR
-Antibiotics only for pts with valvular heart disease, immunosuppression, extensive cellulitis, or DM
-Surgical management if fistula present

Hemorrhoids

-Engorgement of the venous plexuses of the rectum, anus, or with; with protrusion of the mucosa, anal margin, or both
-Classified as internal or external based on position in relation to dentate line

Causes
-Constipation or straining
-Portal HTN
-Pregnancy



Internal Hemorrhoids

Classification

- Grade I hemorrhoids are visualized on anoscopy and may bulge into the lumen but do not extend below the dentate line.
- Grade II hemorrhoids prolapse out of the anal canal with defecation or with straining but reduce spontaneously.
- Grade III hemorrhoids prolapse out of the anal canal with defecation or straining, and require the patient to reduce them into their normal position.
- Grade IV hemorrhoids are irreducible and may strangulate.

Signs & symptoms
-Painless bleeding after defecation
-Visible during anoscopy
-Not palpable or painful on DRE

Management
-1% hydrocortisone
-Refer to GI for rubber band ligation if prolapsed (bulging out of anus)

External Hemorrhoids

Signs & symptoms
-Rarely bleed but are extremely painful, especially if thrombosed (exquisitely tender blueish perianal nodule)
-Itching
-Visible externally on perianal exam

Management
-Sitz bath
-1% hydrocortisone
-Stool softeners
-May need to remove thrombosed clot
-Surgical referral if refractory to medical management

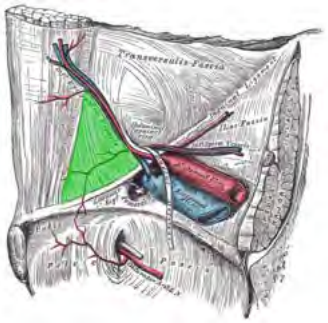
HERNIAS

Inguinal Hernias

-Risk factors: h/o or FH of hernia, older age, chronic cough, chronic constipation, strenuous exercise, abdominal wall injury, h/o AAA, smoking, ascites
-Differential: hydrocele, inguinal adenitis, varicocele, ectopic testis, lipoma, hematoma, sebaceous cyst, hidradenitis, psoas abscess, lymphoma, metastatic neoplasm, epididymitis, testicular torsion, femoral hernia, femoral adenitis, femoral aneurysm
-Workup: groin US if uncertain of mass etiology

Direct Inguinal Hernia

-When intestine plows through weak abdominal tissue in area of Hesselbach's triangle (bordered by inguinal ligament, inferior epigastric vessels, and rectus abdominis)



Causes
-Increased intra-abdominal pressure
-Weakening of tissue due to age or smoking

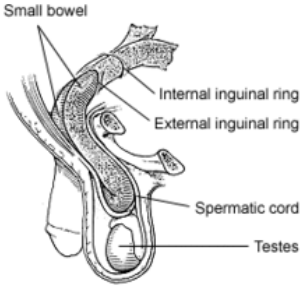
Signs and Symptoms
-Bulge in area of Hesselbach's triangle
-Only mild, intermittent pain or discomfort unless incarcerated or strangulated
-Signs of sepsis in an incarcerated hernia

Management
-If only mild symptoms or asymptomatic → consider watchful waiting
-Attempt manual reduction of incarcerated hernias
-If symptomatic → surgical hernia repair, usually laparoscopic if bilateral or recurrent

Prognosis
-High post-op recurrence

Indirect Inguinal Hernia

-Most common type of hernia
-Occurs when intestine slips through an abnormally open inguinal canal (patent processus vaginalis)



-Variation is a *pantaloon hernia* which is a combined direct and indirect inguinal hernia where both hernias straddle each side of the inferior epigastric vessels

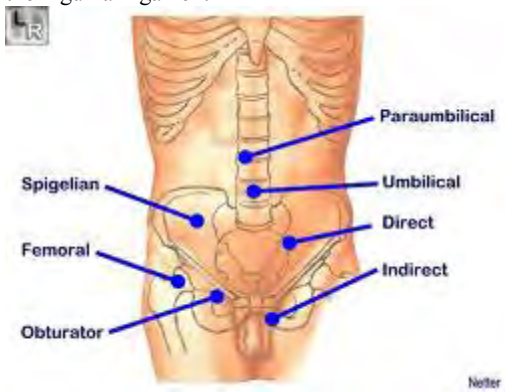
Signs and Symptoms
-Bulge in scrotum due to herniation through inguinal canal

Management
-Higher risk of strangulation so surgical repair is indicated

Prognosis
-Risk of postoperative pain syndrome from damage to ilioinguinal nerve

Femoral Hernia

-Occurs through the femoral canal, which is just below the inguinal ligament



-Bordered by femoral vein laterally, lacunar ligament medially, and Cooper's ligament below
-More common in females

Signs and Symptoms
-Commonly presents emergently as an incarceration or strangulation

Management
-Surgical repair

Umbilical Hernia

-Technically a type of ventral hernia since it is an abdominal wall defect
-Caused by open umbilical ring, which usually closes in all kids by 5 years but may be slower to close in black children
-May interfere with feeding if it contains bowel
-Rarely become incarcerated or strangulated in kids

Management
-Referral for surgical repair indicated when hernia is incarcerated, extremely large, or symptomatic

Ventral Hernias

-Caused by defects in the abdominal wall
-Diastasis recti is an abdominal wall defect but is not a true hernia and does not require repair

Types
-Incisional: occurs through site of previous surgical incision
-Epigastric: occur between umbilicus and xiphoid process
-Spigelian: hernia through Spigelian fascia

Signs and Symptoms
-Spigelian hernias may not be detected on physical exam but pts present with mid or lower abdominal pain and swelling lateral to rectus muscle

Workup
-CT to visualize Spigelian hernia

Management
-Most incisional hernias should be repaired (mesh is preferred) due to risk of incarceration unless very small or large, or upper abdominal and asymptomatic
-Epigastric hernias have low risk for incarceration and only need repair if symptomatic
-Surgical repair of Spigelian hernias due to high risk of strangulation

Other Hernias

Internal Hernia	Obturator Hernia	Littre Hernia	Richter Hernia	Richter Hernia
-Occurs after abdominal surgeries when the bowel gets trapped as a result of new anatomic relationships	-Occurs when small bowel herniates into the obturator canal	-Any groin hernia that contains Meckel's diverticulum	-Occurs when a knuckle of bowel protrudes into a hernia defect, but only a portion of the circumference is involved and the bowel lumen remains patent	-Any hernia that contains intra-abdominal organs

Diarrhea

Acute Diarrhea

-Less than 2 weeks' duration

Etiologies
-Most common causes are infectious agents, bacterial toxins, or drugs
-Anal sex*: Neisseria gonorrhoeae*, syphilis, lymphogranuloma venereum, HSV
-Noninfectious: drug reaction, UC, Crohn's, ischemic colitis, fecal impaction, laxative abuse, radiation colitis, emotional stress

Inflammatory
-Invasive or toxin-producing bacteria
-Causes: shigellosis, salmonellosis, *Campylobacter, Yersinia, C. diff*, EHEC, *Entamoeba histolytica, Neisseria gonorrhoeae, Listeria*
-Blood, pus, fever = fecal leukocytes usually present
-Diarrhea is typically smaller in quantity
-Associated LLQ cramps, urgency, tenesmus
-Workup with stool cultures, *C. diff*, ova, parasites

Non-inflammatory
-Viral or noninvasive bacteria
-Infectious causes: virus, preformed toxin, toxin producing bacteria, protozoa (ETEC, *Staph, Bacillus cereus, Clostridium*, viruses, *Giardia*)
-Watery, nonbloody = no fecal leukocytes
-Diarrhea may be voluminous
-May have periumbilical cramps, bloating, n/v
-Prominence of vomiting suggests food poisoning or viral enteritis
-Typically only eval if persists beyond 7 days or worsens

When to evaluate further
-Signs of inflammatory diarrhea: fever > 101.3, bloody diarrhea, abdominal pain
-Passage of > 6 loose stools in 24 hours
-Profuse watery diarrhea and dehydration
-Frail older patients
-Immunocompromised patients
-Hospital-acquired diarrhea
→ Tests indicated: fecal leukocytes, routine stool culture, *C. diff* if recent hospitalization or antibiotics, 3x ova and parasites if > 10 d, travel, community water outbreak, HIV, MSM

Management
-Rehydration: ½ tsp salt, 1 tsp baking soda, 8 tsp sugar, 8 oz OJ diluted to 1 L with water
-Antidiarrheals for mild to moderate illness
-Loperamide (non-systemic opioid) as long as there is no blood, high fever, or systemic toxicity
-Bismuth subsalicylates (Pepto Bismol) good for traveler's diarrhea as it is antibacterial and anti-inflammatory
-Empiric antibiotic treatment only for immunocompromised, significant dehydration, mod-severe fever, tenesmus, bloody stools, or presence of fecal lactoferrin → cipro, Septra, or doxycycline
-Antibiotics are not recommended in nontyphoid *Salmonella, Campylobacter*, EHEC, *Aeromonas*, or *Yersinia*
-Antibiotics are recommended in shigellosis, cholera, extraintestinal salmonellosis, traveler's diarrhea, *C. diff*, giardiasis, and amebiasis
-Hospitalization for severe dehydration, severe or worsening bloody diarrhea, severe abdominal pain, signs of sepsis, or worsening diarrhea in patients > 70

Chronic Diarrhea

-Greater than 4 weeks' duration
-Not attributed to viruses or bacteria other than *C. diff*

Differential and signs/symptoms
-Osmotic (lactose intolerance or other osmotic agents, factitious Mg overuse or laxative use): stool volume changes with fasting, ↑ stool osmotic gap
-Secretory (hormonally mediated, factitious, villous adenoma, bile salt malabsorption, meds): > 1L stool per day, little change with fasting, normal stool osmotic gap, nonanion gap metabolic acidosis, hyponatremia
-Inflammatory (UC, Crohn's, microscopic colitis, malignancy, radiation): fever, hematochezia, abdominal pain, anemia, hypoalbuminia, ↑ ESR or CRP
-Meds: SSRIs, cholinesterase inhibitors, NSAIDs, PPIs, ARBs, metformin, allopurinol
-Malabsorption: weight loss, elevated fecal fat, anemia, hypoalbuminia
-Motility disorders (IBS): systemic disease or prior abdominal surgery
-Chronic infections (parasites, AIDS-related)

Workup
-Ask if diarrhea occurs at nighttime or while fasting
-Exclude causes of acute diarrhea, lactose intolerance, IBS, previous gastric surgery, parasitic infections, meds, systemic disease
-Initial tests: CBC, CMP, Ca, P, albumin, TSH, vitamin A, vitamin D, INR, ESR, CRP, IgA for Celiac
-Stool studies: ova, parasites, electrolytes, fat stain, occult blood, leukocytes or lactoferrin
-Consider antigen detection for *Giardia* and *Entamoeba*
-Consider acid stain for *Crypto* and *Cyclospora*
-Refer for colonoscopy with biopsy
-Further testing: 24 hour fecal fat, neuroendocrine tumors

Management
-Loperamide
-Diphenoxylate with atropine
-Codeine and deodorized tincture of opium: only for intractable chronic diarrhea
-Clonidine
-Octreotide: for neuroendocrine tumors and AIDS diarrhea
-Cholestyramine

VITAMIN AND NUTRITIONAL DEFICIENCIES

Childhood Nutritional Deficiencies

-Supplements indicated for children from neglected or deprived environments, anorexia, inadequate appetite, lead poisoning, failure to thrive, limited sunlight exposure, with chronic disease affecting absorption and utilization of nutrients, who are trying to lose weight, or are on restrictive diets

Vitamin	Iron	Vitamin D	Calcium
Screening	-Hb routinely checked at 12 months, 3 years, annually in teen females, once in teen males -Screen at 15-18 months for high risk infants	-Screen kids with risk factors (premature, exclusively breast fed, vegetarian diet, high altitude, malabsorption)	-Ask about milk consumption at well child visits
Signs of deficiency	-Anemia -Impaired psychomotor or mental development -Susceptibility to infection -Decreased exercise capacity -Thrombosis	-Rickets -Osteomalacia	-Rickets -Susceptibility to fracture
Workup	-Hb or CBC -Ferritin, Hb electrophoresis, B12, folate -FOBT -Celiac workup -IBD workup	-25-OH vitamin D level	-DEXA scan
Recommendations	-Iron supplements for preterm infants until 12 months -Iron-fortified infant formulas -No cow's milk until 12 months -Supplement as needed with oral iron -Recheck CBC every 4 weeks during therapy	-At least 400-600 IU daily -Follow supplementation with laboratory testing	-Whole milk from 1-2 years of age -Kids 1-3 need 700 mg of Ca (~2 cups of milk) -Kids 4-8 need 1000 mg of Ca (~2-3 cups of milk) -Kids 9-18 need 1300 mg of Ca (~3+ cups of milk) -Decrease soda intake (P in it associated with bone fx) -Other sources: white beans, broccoli, fortified OJ, salmon, sweet potatoes -Calcium in spinach is not bioavailable!

Adult Nutritional Deficiencies

Nutrient	Copper	Iodine	Selenium	Zinc
At risk	-X-linked transport mutation -Malabsorption after GI surgery or gastric bypass -Ingestion of high doses of Zn	-Consumption of uniodized salt	-Chinese diet devoid of selenium -TPN without supplementation	-Low protein intake -From developing country
Signs & symptoms	-Anemia -Ataxia -Myeloneuropathy -Fragile hair -Skin depigmentation -Edema -Osteoporosis -Hepatosplenomegaly	-Goiter -Hypothyroidism → ↓ growth, development, and cognitive function	-Cardiomyopathy -Skeletal muscle dysfunction	-Growth retardation -Hypogonadism -Oligospermia -Alopecia -Impaired taste -Immune dysfunction -Night blindness -Impaired wound healing -Skin lesions

Nutrient	Chromium	Iron	Magnesium	Vitamin B12	Vitamin D
At risk	-Hospitalized patients with increased metabolic demands, especially diabetics -Short bowel syndrome -Burns or trauma -TPN without supplementation	-Menstruating women	-ICU	-Vegetarians	-Elderly -Homebound -Limited sun exposure -Obesity -Celiac or IBD -CKD -Gastric bypass -Cystic fibrosis -Anticonvulsants
Signs & symptoms	-Impaired glucose tolerance	-Microcytic anemia	-Insulin resistance -Constipation -Migraines -RLS -Cramping -HTN -Fibromyalgia -Weakness -Trosseau and Chvostek signs	-Lethargy -Unwanted weight loss -Mental status change -Anxiety -Depression	-Osteoporosis -Nonspecific msk pain -Rickets -Alopecia

METABOLIC DISORDERS

Phenylketonuria

-Autosomal recessive disorder
-Screened for in newborn metabolic screening
-From defective conversion of phenylalanine to tyrosine
-Phenylalanine is found in breast milk and standard formulas

Signs & symptoms
-Intellectual disability
-Epilepsy
-Abnormal gait, posture, or stance
-"Mousy" urine or body odor
-Eczematous rash

Differential
-BH4 deficiency

Workup
-Elevated serum phenylalanine

Management
-Dietary restriction
-Frequent phenylalanine and tyrosine monitoring

OTHER GASTROINTESTINAL SYSTEM TOPICS

Hyperbilirubinemia

-Jaundice is common in newborns since it is formed at high levels during this time and not cleared as well as in adults
-Hyperbilirubinemia puts infant at increased risk for encephalopathy and kernicterus
-Jaundice within first 24 hours of life is worrisome
-Jaundice developing in 72-96 hours is physiologic and resolves in 1-2 weeks
-"Breast milk jaundice" begins in the first week after birth, peaks at 2 weeks, and then declines; it is not dangerous and is probably due to the infant's immature liver and intestines

Risk and protective factors
-Major risk factors for infants ≥ 35 weeks' gestation: predischarge total bili in the high risk zone, jaundice in first 24 hours, positive DAT or known hemolysis, gestational age 35-36 weeks, previous sibling received phototherapy, cephalohematoma or significant bruising, exclusive breastfeeding, East Asian race
-Minor risk factors: predischarge total bili in the high intermediate risk zone, gestational age 37-38 weeks, jaundice observed before discharge, previous sibling with jaundice, macrosomic infant of diabetic mother, maternal age > 25 years, male gender
-Decreased risk factors: total bili in low risk zone, gestational age > 41 weeks, exclusive bottle feeding, black race, hospital d/c after 72 hours

Signs & symptoms
-Appearance of jaundice begins in the face and progresses to the chest, abdomen, arms, and then legs

Screening
-Usually done routinely at time of metabolic screening prior to discharge (USPSTF grade I); infants with total bili > 95th percentile are at increased risk
-Routine follow-up appointments after discharge are timed to assess developing jaundice, with f/u in 3 days for infants d/c before 24 hours (or sooner if high-risk), and later for infants d/c after 48 hours or beyond

Management
-Total bili values are compared in percentiles (Bhutani nomogram)
-Calculate risk zone of infant based on risk factors and total bili values
-Admit for phototherapy if needed
-Admit for exchange transfusion if needed: initiated when phototherapy has failed or infant has signs of neuro dysfunction
-Home measures for low-risk infants: increasing frequency and efficacy of breastfeeding, supplementing inadequate breastfeeding with formula

Pediatric Abdominal Pain

Chronic Abdominal Pain = greater than 1-2 months duration

-Most digestive tract pain is perceived in the midline, so any lateralizing is usually the gallbladder, kidney, ureter, ascending/descending colon, or ovary

Organic Etiologies

Right upper quadrant
Hepatitis
Cholecystitis
Cholangitis
Biliary colic
Pancreatitis
Budd-Chiari syndrome
Pneumonia/empyema pleurisy
Subdiaphragmatic abscess
Right lower quadrant
Appendicitis
Salpingitis
Ectopic pregnancy
Inguinal hernia
Nephrolithiasis
Inflammatory bowel disease
Mesenteric adenitis (yersina)
Epigastric
Peptic ulcer disease
Gastroesophageal reflux disease
Gastritis
Pancreatitis
Myocardial infarction
Pericarditis
Ruptured aortic aneurysm
Periumbilical
Early appendicitis
Gastroenteritis
Bowel obstruction
Ruptured aortic aneurysm

Left upper quadrant
Splenic abscess
Splenic infarct
Gastritis
Gastric ulcer
Pancreatitis
Left lower quadrant
Diverticulitis
Salpingitis
Ectopic pregnancy
Inguinal hernia
Nephrolithiasis
Irritable bowel syndrome
Inflammatory bowel disease
Diffuse
Gastroenteritis
Mesenteric ischemia
Metabolic (eg, DKA, porphyria)
Malaria
Familial Mediterranean fever
Bowel obstruction
Peritonitis
Irritable bowel syndrome

Functional Etiologies

Rome III diagnostic criteria for functional gastrointestinal disorders of childhood childhood (ages 4 to 18 years)

Functional dyspepsia (must include all of the following):

Within the preceding two months, at least weekly occurrence of:

- Persistent or recurring pain or discomfort in the upper abdomen, **and**
- No evidence of inflammatory, anatomic, metabolic, or neoplastic process to explain the symptoms, **and**
- Pain or discomfort not relieved by defecation or associated with the onset of a change in stool frequency or form

Irritable bowel syndrome (must include all of the following):

Within the preceding two months, at least weekly occurrence of:

- Abdominal discomfort or pain associated with ≥2 of the following:
  - Relieved with defecation, **and/or**
  - Onset associated with a change in frequency of stool, **and/or**
  - Onset associated with a change in form (appearance) of stool, **and**
  - No evidence of inflammatory, anatomic, metabolic, or neoplastic process to explain the symptoms

Functional abdominal pain (must include all of the following):

Within the preceding two months, at least weekly occurrence of:

- Episodic or continuous abdominal pain, **and**
- Insufficient criteria for other functional gastrointestinal disorders, **and**
- No evidence of inflammatory, anatomic, metabolic, or neoplastic process to explain the symptoms

Childhood functional abdominal pain syndrome

Within the preceding two months, at least weekly occurrence of:

- Childhood functional abdominal pain at least 25 percent of the time and ≥1 of the following:
  - Some loss of daily functioning, **and/or**
  - Additional somatic symptoms such as headache, limb pain, or difficulty sleeping

Abdominal migraine pain (must include all of the following):

Within the preceding 12 months, ≥2 episodes of:

- Paroxysmal episodes of intense, acute, periumbilical pain that lasts for ≥1 hour, **and**
- Intervening periods of usual health lasting weeks to months, **and**
- The pain interferes with normal activities, **and**
- The pain is associated with ≥2 of the following:
  - Anorexia, **and/or**
  - Nausea, **and/or**
  - Headache, **and/or**
  - Photophobia, **and/or**
  - Pallor, **and**
  - No evidence of inflammatory, anatomic, metabolic, or neoplastic process to explain the symptoms

Management

-Goal is to return to normal function vs. complete elimination of pain
-Pain diaries
-Biopsychosocial model of care receives higher satisfaction in this setting
-Relaxation techniques
-Dietary changes: removing lactose or increasing fiber
-Set plan for return to school (may begin part-time but homeschooling is discouraged)
-Medications for pain triggers: acid, constipation, altered motility
-Refer to GI for alarm symptoms of active or persistent bleeding, weight loss, early satiety with peptic symptoms, loss of appetite, persistent chest pain, persistent vomiting, or failure to improve with medical therapy

Pediatric Acute Abdominal Pain

Differential

Neonate	2 months-2 years	2-5 years	>5 years	
Colic Dietary protein allergy Volvulus Necrotizing enterocolitis Testicular torsion Adhesions	Gastroenteritis Viral illness Trauma (including inflicted injury) Incarcerated hernia Intussusception Urinary tract infection Foreign body ingestion Sickle cell syndrome vasoocclusive crisis Dietary protein allergy Tumor Hirschsprung disease Adhesions Hemolytic uremic syndrome Toxin Meckel's diverticulum Hepatitis	Gastroenteritis Viral illness Trauma (including inflicted injury) Appendicitis Pharyngitis Constipation Urinary tract infection Pneumonia Intussusception Foreign body ingestion Sickle cell syndrome vasoocclusive crisis Henoch Schönlein purpura Ovarian torsion Intraabdominal abscess Tumor Adhesions Hemolytic uremic syndrome Hepatitis Meckel's diverticulum Toxin Primary bacterial peritonitis	Gastroenteritis Viral illness Appendicitis Trauma Constipation Pharyngitis Pneumonia (lower lobe → diaphragm irritation) Urinary tract infection: may also cause diarrhea Diabetic ketoacidosis Sickle cell syndrome vasoocclusive crisis Henoch Schönlein purpura Ovarian torsion Testicular torsion Inflammatory bowel disease	Intraabdominal abscess Ruptured ovarian cyst Cholecystitis Pancreatitis Urolithiasis Hepatitis Meckel's diverticulum Perforated ulcer Adhesions Hemolytic uremic syndrome Myocarditis, pericarditis Primary bacterial peritonitis Familial Mediterranean fever Abdominal migraine

Workup

-Unnecessary for kids that are otherwise healthy, well-appearing, and have normal Pes
-CBC with smear for infection and red cell morphology
-Hct for bleeding
-Liver enzymes and amylase for suspected hepatitis, cholecystitis, or pancreatitis
-BMP for DKA
-Urinalysis
-Urine HCG for all menstruating females
-Rapid *Strep* test
-Imaging for kids with hx of trauma, peritoneal irritation signs, obstructive signs, masses, distension, or focal tenderness or pain (for pediatric appendicitis, consult with pediatric surgeon before ordering imaging) → abdominal film for obstruction, upper GI series with contrast for volvulus, US or contrast enema for intussusception, CT with contrast when a wide variety of dx are being considered, US or non-contrast helical CT for urolithiasis

Management

-Pain control with morphine is shown to not affect exam results
-Rule out life-threatening etiologies

MUSCULOSKELETAL SYSTEM

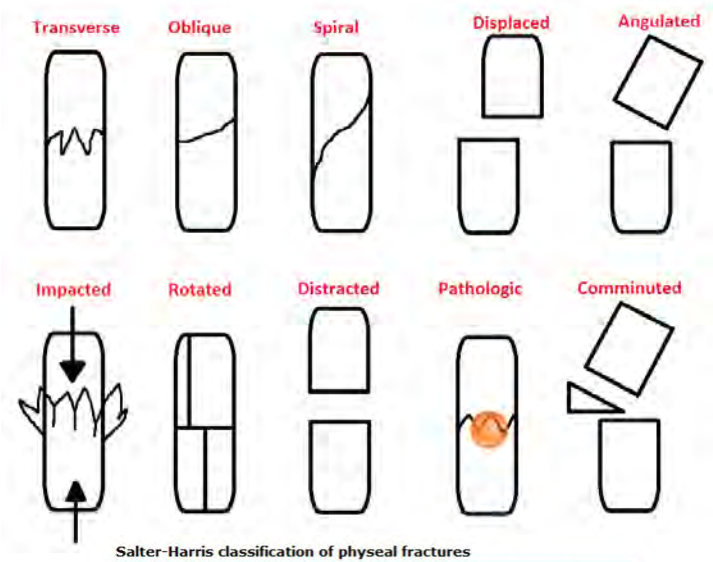
FRACTURE BACKGROUND

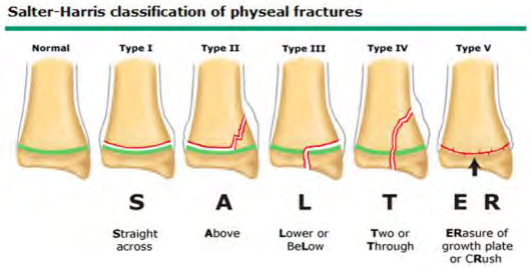
Pediatric Fractures

-Bowing and greenstick fx are unique to kids due to their skeletal immaturity
-Growth plate fx are classified by Salter-Harris
-Most fx only require closed reduction
-Kids heal faster due to more active periosteum and higher % cartilage

Fractures Associated with Child Abuse
-Metaphyseal corner fx: child abuse until proven otherwise
-Posterior rib fx: child abuse until proven otherwise
-Any fracture in a child under 1
/-LE fracture in a non-ambulatory child
-Multiple fractures in various stages of healing
-Sternal or scapular fx: high impact mechanism such as MVC required or else it may be child abuse
-Spinous process fracture
-Lower specificity: clavicular fx, long bone fx, linear skull fx

Types of Fractures





General Information

-May involve part or all of bone cortex
-Open = skin or overlying mucous membrane is breached; closed = no damage to skin or mucous membrane

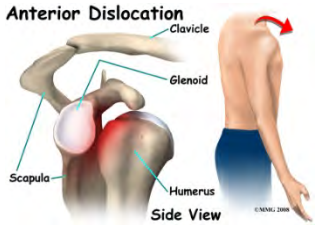
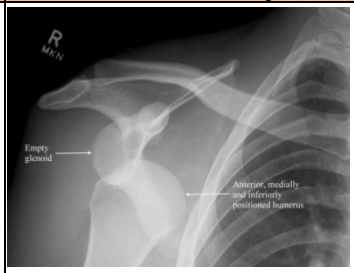
Presentation
-Will always cause pain
-Tender, swollen, and with mobility at the fracture site
-Loss of limb function

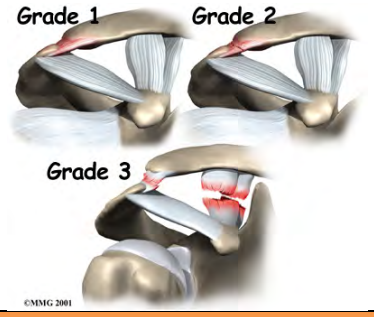
Workup
-All suspected fractures need at least 2 views for radiographs: AP, lateral
-CT for subtle stress fractures or for inability to detect on x-ray but with high suspicion
-MRI: T1 for new fractures, T2 for older fractures

Complications
-Most commonly DVT or PE
-Compartment syndrome
-Avascular necrosis
-Nerve injury
-Malunion, nonunion, or delayed union
-Complex regional pain syndrome form injury to sympathetics (burning pain, skin changes, swelling, excessive sweating at site of injury)

DISORDERS OF THE SHOULDER

Dislocations, Separations, and Fractures

Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Shoulder Dislocation	-Usually anterior 	-Pt will support affected arm with the other arm -Shoulder may appear flattened -Displaced greater tuberosity and unusual subclavicular bulge -May have loss of sensation over shoulder due to axillary nerve entrapment		-Posterior dislocation can be life-threatening → immediate ortho consult -Manual relocation -Can resolve spontaneously after a few weeks

Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Clavicle Fracture	-Usually a pediatric fracture -MOI: direct force to lateral shoulder from a fall or sporting injury	-Affected extremity held close to body -Shoulder is slumped downward, forward, and inward		-Immobilization in figure 8 dressing
Scapula Fracture	-MOI: direct violent trauma	-May also have injury to ribs, chest wall, or shoulder girdle -Shoulder is adducted and arm is held close to the body		-Immobilization with sling and swathe dressing
AC joint separation	-MOI: direct blow to shoulder	-Step deformity -Tenderness over joint	-Crossover test -X-rays 	-Depends on classification -May need surgery

Common Shoulder Disorders

Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Subacromial bursitis		-Shoulder pain with motion as well as rest -Tenderness of bursa	-Bursal fluid aspiration for if signs of inflammation present	-Indomethacin or other NSAID at anti-inflammatory doses -Steroid injection for severe or persistent pain
Biceps tendon injury	-Includes tendonitis, tendinosis, subluxation, partial tears, and complete tears	-Pain over anterior shoulder that radiates to biceps -Pain aggravated by lifting, pulling, or overhead motion -Click -Night pain -Ecchymosis and swelling with rupture -May have other associated injuries such as SLAP tear or rotator cuff injury	-Speed's and Yergason's tests -US or MRI	-Rest -NSAIDs -PT -Subacromial or biceps tendon sheath injections -Refer to ortho for rupture
Rotator cuff injury		-Rotator cuff tendinopathy: night pain, pain with overhead motion -Rotator cuff tear: weakness, difficulty reaching, may be asymptomatic -Tears can be chronic or acute	-US is gold standard -MRI	-Cryotherapy, rest, ice, NSAIDs -6 weeks of PT -1-2 steroid injections for pain refractory to orals -Refer to ortho if suspecting tear or with no improvement in 6-9 months
Shoulder impingement syndrome	-Refers to compression of the GHJ with shoulder elevation	-Increased translation of humeral head -Abnormal acromion morphology -Osteophytic changes of the AC joint -Pain with overhead activity	-R/o rotator cuff tear and adhesive capsulitis -Neer and Hawkins impingement tests -Radiographs -US	-PT -Refer to ortho after 3 months of failed conservative treatment
Shoulder instability	-A result of excessive laxity of the GHJ → failure to keep head of humerus centered in glenoid fossa	-Recurrent episodes of shoulder instability and pain: looseness, crepitus, anterolateral shoulder pain	-R/o unidirection cause of instability such as a tear -Positive sulcus sign -Crepitation or popping -Positive apprehension test	-PT -Referral to orthopedic surgery for persistent pain or recurrent dislocation episodes

Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Adhesive capsulitis	-Contraction of GHF capsule -Usually secondary to immobilization after an injury -Can also be from thyroid issues, chemo, radiation, DM	-Painful stage for up to 3 months -Followed by adhesive stage for 3-6 months (less pain but increased stiffness and reduced movement) -Recovery stage: ↓pain, slow ↑ ROM, spontaneous but incomplete recovery	-R/o rotator cuff tear	-PT -NSAIDS -Injections -Surgical lysis of lesions

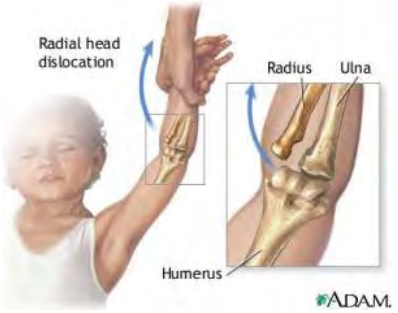
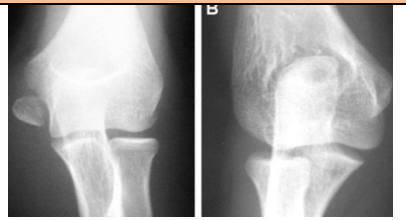
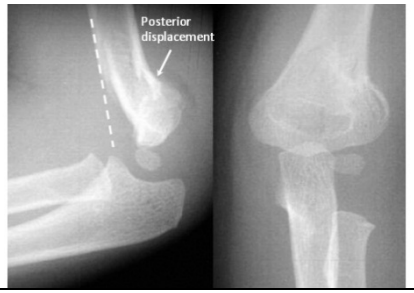
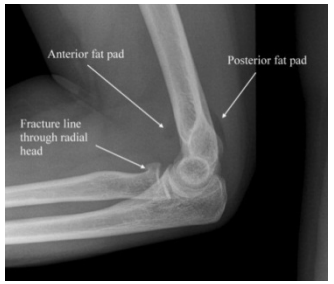
DISORDERS OF THE ARM

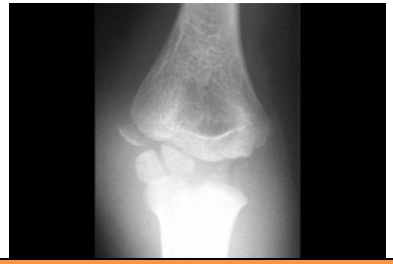
Arm Fractures

Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Humeral shaft fracture	-Typically from trauma in the elderly	-Extensive bruising of upper arm -Wrist drop from radial nerve damage		-Wrist splinting and casting over site of break

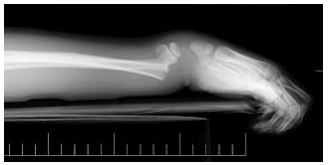
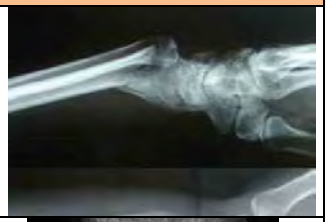
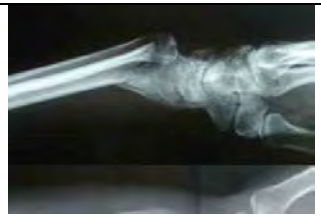
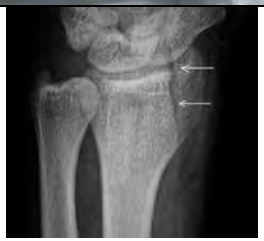
DISORDERS OF THE ELBOW

Elbow Dislocations and Fractures

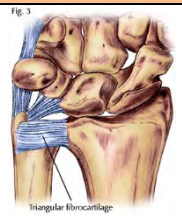
Injury Type	Information	Injury Type	Information
Nursemaid's Elbow (Radial Head Subluxation)	-MOI: being pulled up too hard by hand or wrist → radial head slipping out of annular ligament -S/s: crying, screaming, holding arm flexed against belly, refusal to use arm -X-ray & assess neurovascular involvement -Reduce with flexion and supination of the arm (usually occurs during x-ray positioning) 	**Medial epicondyle fracture**	-MOI: acute valgus sress during FOOSH, posterior stress, chronic muscular traction (throwing) -Associated with elbow dislocation or subluxation 
Supracondylar fracture	-Pediatric fracture -Usually involves distal humerus -S/s: Limb ischemia if branchial artery is damaged -X-ray showing posterior sail sign, anterior humeral line drawn will not bisect the capitate 	**Radial head fracture**	-MOI: FOOSH -Decreased ROM in elbow -Difficult to see on x-ray, may displacement of fat pad, elbow effusion  see

Lateral condylar fracture	-MOI: FOOSH with extended elbow, traction forces, or acute varus stress 			

Common Elbow Disorders				
Injury Type	**Information**	**Signs & Symptoms**	**Workup**	**Management & Prognosis**
Lateral or medial epicondylitis	-A chronic tendinosis from repetitive motion and overuse rather than acute inflammatory process	-Pain with flexion (tennis or racquet) -Pain with extension (golfing)		-Ice -Iontophoresis -Stretch -Surgical release -Strap brace -Injections
Olecranon bursitis	-Onset may be from trauma or may be idiopathic -Often seen in truck drivers from repetitive leaning	-Red, swollen joint -May be painful	-R/o infection, gout, and triceps rupture	-Compression sleeve -Anterior splint -Rarely bursectomy if chronic

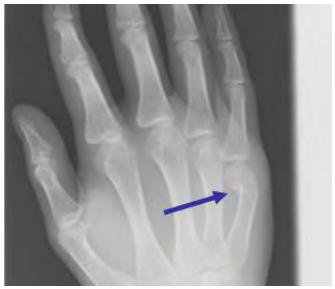
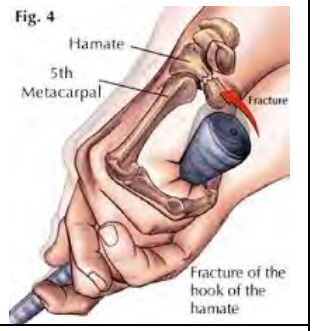
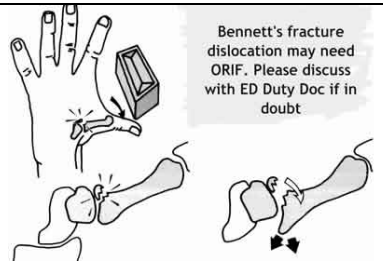
DISORDERS OF THE WRIST			
Wrist Fractures			
Injury Type	**Information**	**Injury Type**	**Information**
Colles Fracture of Distal Radius	-MOI: FOOSH → posterior displacement of wrist ("dinner fork deformity") -Casting alone if nondisplaced -Closed reduction followed by casting if slightly displaced -ORIF & short arm cast if displaced 	**Chauffeur's Fracture of Distal Radius**	-MOI: FOOSH → fracture of radial styloid, usually due to compression against the scaphoid 
Smith Fracture of Distal Radius	-MOI: opposite Colles = fall on back of hand -ORIF & short arm cast 	**Scaphoid Fracture**	-MOI: FOOSH -S/s: fullness or pain in the anatomical snuffbox -Difficult to see on x-ray → 4 view x-ray & repeat imaging in 10-14 days if negative -Immobilize in thumb spica -Risk of scaphoid necrosis due to poor blood supply 

Common Wrist Disorders				
Injury Type	**Information**	**Signs & Symptoms**	**Workup**	**Management & Prognosis**
DeQuervain's tenosynovitis	-Tendinosis of sheath surrounding abductor pollicis longus and extensor pollicis brevis tendons -Common in new moms picking up babies	-Radial wrist pain		-Ice -Iontophoresis -Rest -Injection -Thumb spica -Last resort is surgical release
Scapholunate dissociation	-Traumatic scapholunate ligament tear	-Wrist pain and instability	-Radiograph showing Letterman sign of increased SL joint space	-Surgical -Can lead to degenerative arthritis and scapholunate collapse without repair

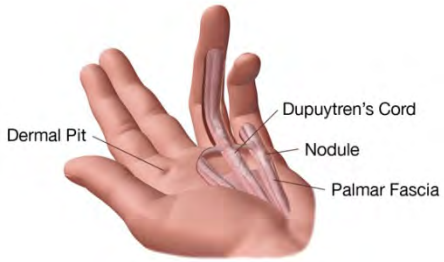
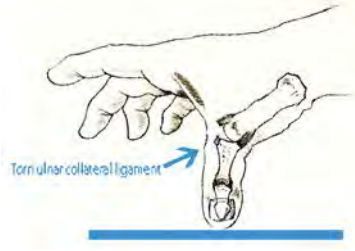
Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Triangular fibrocartilage complex (TFCC) tear	-Acute or from repetitive use 	-Tenderness over TFCC		-Splint -NSAIDs -PT -Injections -Surgical repair

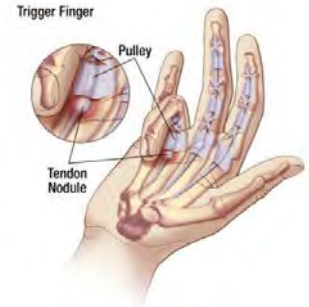
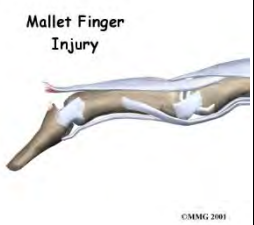
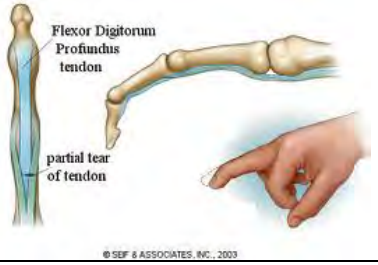
DISORDERS OF THE HAND

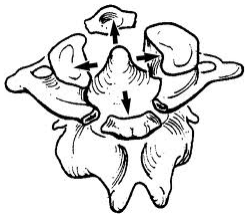
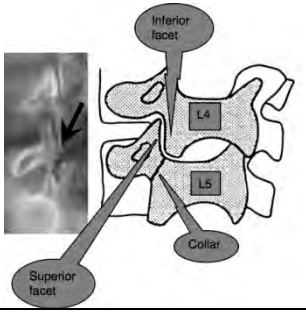
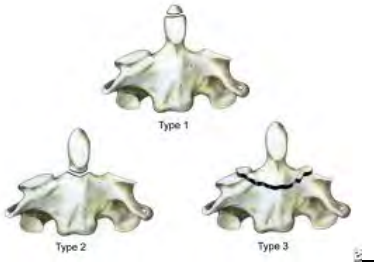
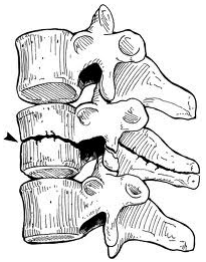
Hand Fractures

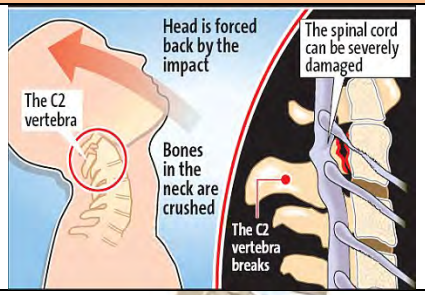
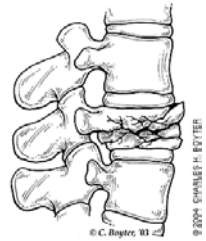
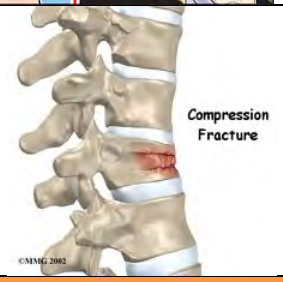
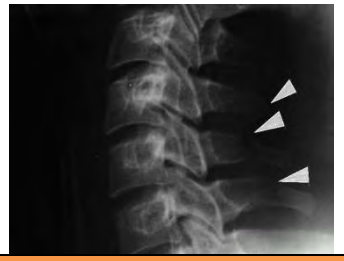
Injury Type	Information	Injury Type	Information
Boxer's Fracture of Distal 5th Metacarpal	-MOI: blow of closed fist against another object -Splinting vs percutaneous pinning 	**Hook of the hamate fracture**	-Bust in hamate due to forceful impact as in racquetball -Check ulnar nerve -May need CT to see -Need to excise fragment or cast 
Bennet's fracture	-Fracture of the thumb at the metacarpal base → abductor pollicis pulls fragment away -ORIF if unstable 	**Phalangeal fracture**	-Distal: splint or CRPP if displacedd -Middle or proximal: buddy tape or ORIF if displaced

Common Hand Disorders

Injury Type	Information	Injury Type	Information
Depuytren's contracture	-Occurs when thickened palmar fascia forms nodules over the flexor tendons → flexion contracture -Surgery indicated for contractures > 30 ° 	**Skier's thumb (gamekeeper's thumb)**	-Torn ulnar collateral ligament due to abduction stress (breaking rabbit necks) -Thumb spica if nondisplaced -Surgical repair if displaced or complete tear 

Injury Type	Information	Injury Type	Information
Trigger finger	-AKA stenosing tenosynovitis -Congenital thickened flexor tendon or nodule at pulley → locking, stiffness, pain in finger -More common in RA, OA, DM -Injection at the finger flexor crease into sheath -Surgical release 	**Mallet finger**	-Traumatic rupture of extensor tendon distal to DIP -Splint 
Jersey finger	-Traumatic rupture of flexor digitorum profundus at DIP (grabbing football jersey as player pulls away) -Surgical repair within 2 weeks or it will be permanent 		

DISORDERS OF THE BACK & SPINE			
Fractures			
Injury Type	**Information**	**Injury Type**	**Information**
Jefferson fracture (atlas burst)	-Fracture of anterior and posterior arches of atlas -A result of axial load on back of the head or hyperextension of neck -May be accompanied by other cervical spine fractures -Treatment depends on stability of injury = whether or not transverse ligament is intact -Stable = rigid C collar for 3 months -Unstable = halo for 3 months -Spinal fusion for large displacements 	**Spondylolysis**	-Stress fx of pars interarticularis, usually L5 -Seen in gymnasts, football players, weight lifters -Pain adjacent to midline and aggravated with extension and rotation -May be asymptomatic -X-ray showing scotty dog with collar -Modification of activities -Core strengthening 
Odontoid fracture	-Break in the dens from hyperflexion or hyperextension -Can also have Jefferson fracture -Treatment is reduction and halo immobilization for 3 months -Can have permanent neurologic injury 	**Chance fracture**	-Compression injury to the anterior portion of the vertebral body as well as a transverse fracture through the posterior vertebra/vertebral body -MOI: MVA where seatbelt immobilizes pelvis while thorax is thrust forward -Unstable -Lateral view radiograph 

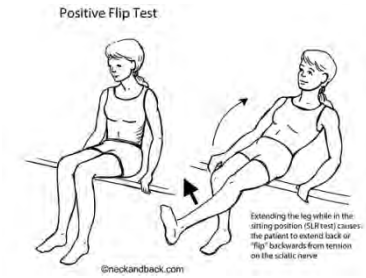
Injury Type	Information	Injury Type	Information
Hangman's fracture (C2 spondylolisthesis)	-Fracture of both pedicles of the axis -Unstable -Immobilize in halo -ORIF if severe 	**Burst Fracture**	-Vertebral body collapse -From high height fall with land on feet or butt -Unstable -May have neuro sx from fragments extending into the spinal canal 
Wedge fracture	-Collapse of anterior vertebral body with intact posterior wall -From hyperflexion +/- osteoporosis -Stable 	**Spinous process fracture**	-AKA clay shoveler's fracture if at C7 -From sudden forceful ligamentous traction on or below the spinous process -Stable 

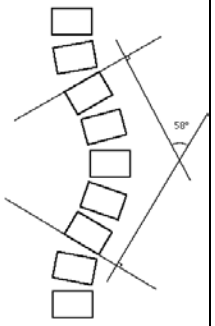
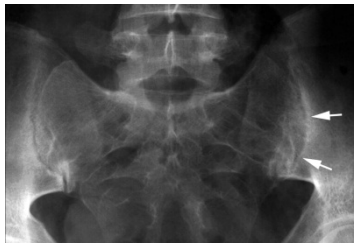
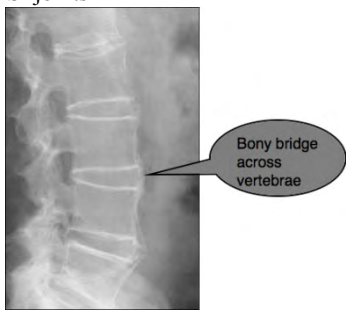
Common Cervical Spine Disorders

Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Cervical strain or sprain	-Trapezial strain will have localized pain that is reproducible on palpable, may have torticollis, treatment is supportive -Strain from whiplash may have associated ligamentous injury → get x-ray to eval stability -Acute cervical sprain is ligamentous injury and is graded depending on severity of disruption → investigate with x-rays, will need hard color or immobilization for subluxation			
Cervical spondylosis	-Degenerative OA of the vertebral discs from repetitive strain or trauma -Usually in C5-C6 -Risk factors: frequent lifting, smoking, excessive driving	-Neck pain -Radiculopathy -Myelopathy (location will point to area of problem!)	-MRI or CT myelogram	-Supportive -Face injections -Surgical decompression with diskectomy or laminectomy
Cervical stenosis	-Narrowing of the cervical spinal canal -May be congenital or acquired -Usually in C5-C6	-Often asymptomatic until neuro sx	-Severity assessed with radiograph and Torg's ratio (canal:vertebral body width)	-Conservative treatment if no neuro deficits -Decompressive laminectomy for progressive neuro deficit
Brachial plexus neurapraxia (stinger or burner)	-Stretch injury of the brachial plexus	-Sudden burning or numbness in lateral arm, thumb, and index finger -Lasts 1-2 minutes	-MRI fi symptoms persist > 15 minutes or are repeated	-ROM -Strengthening

Common Thoracic Spine Disorders

Injury Type	Information	Signs & Symptoms	Management & Prognosis
Hyperkyphosis	-Occurs in 20-40% of older adults	-Thoracic pain -Decreased pulmonary functioning -Increased fractures	-Exercise-based treatment focusing on postural alignment, flexibility, and strengthening -Incurs increased risk of mortality

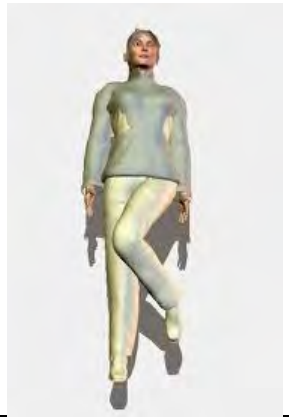
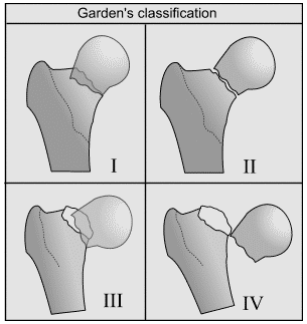
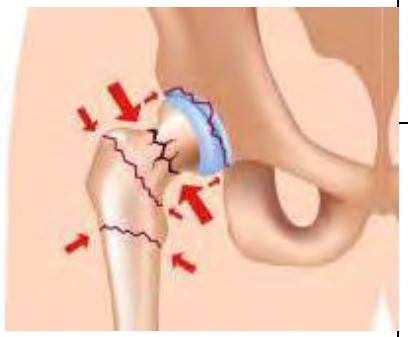
Common Lumbar Spine Disorders				
Injury Type	**Information**	**Signs & Symptoms**	**Workup**	**Management & Prognosis**
Spondylolisthesis	-Displacement of vertebrae anteriorly or posteriorly -Often seen in dancers and gymnasts -Causes: congenital, traumatic, degenerative, mets	-May go unnoticed until adulthood	-Radiograph -Graded depending on degree of displacement	-If asymptomatic → core exercises, no restrictions -If symptomatic → bracing and core exercises -If progressive or deficits → surgery
Lumbar strain	-Accounts for 70% of low back pain	-Tender paravertebral or erector spinae muscles with minimal radiation		-Modified activity with exercise -Short-term pain relief
Herniated nucleus pulposus	-When torn annulus → impingement of nucleus on spinal cord -Usually at L5	-Sciatica -Positive SLR and FLIP -Pain worse on back extension -Nerve deficits according to root involved	-R/o infection or tumor -MRI or CT 	-NSAIDs -Muscle relaxers -Exercise -Some may need surgery
Sacroiliac dysfunction	-Acute or chronic injury to the SI joint	-SI pain with + FABER -No discogenic pain (no radiculopathy or pain with flexion)		
Cauda equina syndrome	-Compression of L2-L4 nerve roots → paralysis without spasticity	-Loss of bowel and bladder control -Bilateral LE weakness and sensory deficits	-Differential: central disc herniation, abscess, hematoma	-Emergency surgical decompression
Lumbar spinal stenosis	-Progressive degeneration of the disc and facet joints → narrowing of the canal and compression of nerve roots	-Leg cramping -Radiculopathy -Increased pain with sitting or spinal extension with relief of pain with spinal flexion = pt walks stooped over -Sensory disturbances -Decreased DTRs -Mild weakness	-MRI	-PT with core strengthening -NSAIDs -Decrease impact and bending -Surgical decompression for progressive disease
Piriformis syndrome	-Irritation of the sciatic nerve beneath the piriformis muscle -Caused by trauma, spasm, or anatomic defect	-Sciatic notch tenderness	-R/o herniated nucleus pulposus	-Rest, ice, stretching -Injection

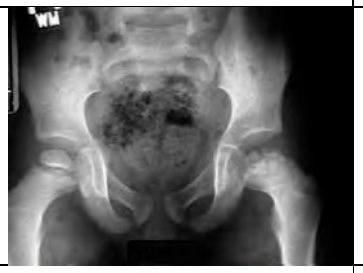
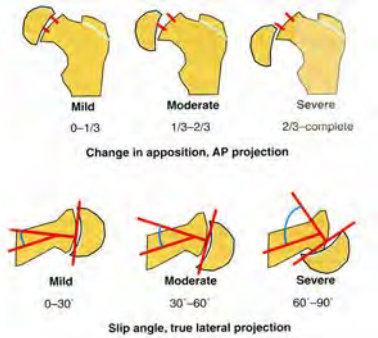
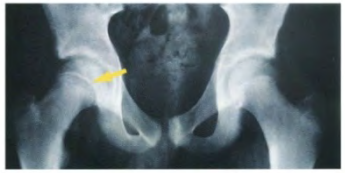
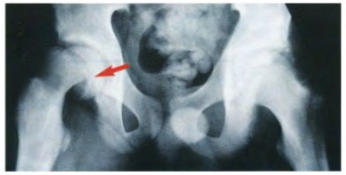
Injury Type	Information	Injury Type	Information	Injury Type
Scoliosis	-Defined as Cobb angle > 10° -Causes: congenital, neuromuscular, idiopathic (the most common kind) -Many schools provide screening but efficacy is not proven (USPSTF grade D) -UpToDate recommends routine screening at well-child visits, especially before growth spurts -Bright Futures: begin after age 8	-May be detected incidentally -Severe curves may result in restrictive pulmonary disease -Pain or rapid progression of curve suggests non-idiopathic etiology	-Arm span measurement to detect Marfan's -Skin examination for neurofibromatosis, spinal dysraphism, tumor, or Marfan's or Ehlers-Danlos -Leg length examination to detect compensatory scoliosis -Foot examination and full neuro exam (esp abdominal reflex) to detect neuromuscular disease -Adams forward bend test  -MRI for associated neuro signs, associated pain, early onset with rapid progression, or abnormalities on x-ray -Calculate Cobb angle -Assess skeletal maturity to determine risk for progression of curvature	-Adolescents with curves at low risk for progression may be followed by primary care -Refer to orthopedic surgery for increased rotation or Cobb angle, or progression of Cobb angle by more than 5° -Refer to specialist for severe pain or neuro symptoms -Efficacy of bracing is disputed -Most patients with untreated idiopathic scoliosis have little functional limitation or pain in adulthood
Ankylosing spondylitis	-Inflammatory back pain > 3 months' duration that is improved by exercise and worsened with rest -Strong association with HLA-B27	-Bilateral sacroiliitis -May also have involvement of hips, shoulders, and joints of the LEs, as well as extra-articular manifestations in the eye and heart -Schober's test shows loss of lumbar flexion 	-Spine film showing syndesmophytes (bony growth within spinal ligament) → bridging and fusion of vertebral bodies → "bamboo spine" -Pelvis film showing erosions and sclerosis at the SI joints 	-Initial therapy with NSAID like indomethacin with trial for at least 4 weeks -Augment with other non-opioid and low potency opioid analgesics as needed -Exercise program or PT -Joint injections for persistent peripheral joint involvement, enthesitis, or sacroiliitis pain -Nonbiologic DMARDs are ineffective for axial disease, need to use anti-TNF agent if nonresponse to NSAIDs -For peripheral disease, can use sulfasalazine or methotrexate -Surgical intervention for severe cases: joint replacement, wedge osteotomy -Poor prognostic indicators are severe hip disease, early age of onset, persistent elevation of ESR

DISORDERS OF THE HIP

Hip Dislocations and Fractures

-Typically occurs in elderly females
-Mortality 20-35% in the first year
-Majority will require corrective surgery

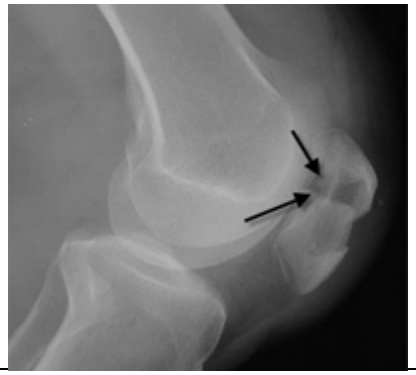
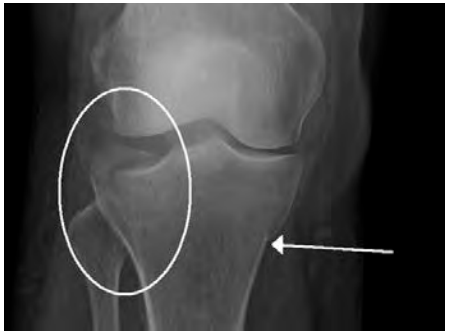
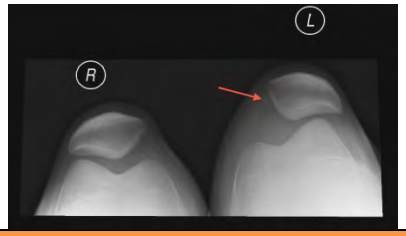
Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Hip dislocation	-Usual posterior (MVA, knee slamming into dashboard)	-Hip flexed, adducted, internally rotated 	-Radiograph -Assess neurovascular involvement	-Pain relief -Reduce with Allis maneuver
Extracapsular fracture	-Does not affect blood supply to femoral head = complications of nonunion are rare -Stable vs unstable (detached fragment of lesser trochanter)	-H/o fall or trauma -Leg may be shortened and externally rotated if displacement is present -May also have fx at another site, usually proximal humerus or distal radius -Rarely neurovascular injury, but can have sciatic nerve injury 		-Internal fixation
Intracapsular fracture	-Can affect blood supply to femoral head, especially if displaced = commonly complications with nonunion and avascular necrosis			-Internal fixation if no displacement -Hemiarthroplasty often the treatment of choice due to high risk of avascular necrosis
Femoral stress fracture	-Usually occurs in the femoral neck -Seen in thin, young endurance athletes	-Groin pain with running that progresses to ADL pain -Pain limits extremes of hip internal and external rotation	-Hip may be radiographically negative → need bone scan in 2-8 days	-If nondisplaced → no weight bearing for 6-8 weeks -If displaced → ORIF
Avulsion fracture of the ASIS	-Occurs where sartorius originates -MOI: knee flexion with hip hyperextension	-Pain over ASIS and with resisted hip flexion	-X-ray	-Nondisplaced → RICE, splint in knee flexion with progressive weight bearing -If displaced → ORIF
Avulsion fracture of the ischial tuberosity	-Occurs where hamstrings originate -Vigorous hip flexion with knee hyperextension	-Pain in buttock -Tenderness at ischial tuberosity		-Nondisplaced → RICE with progressive weight bearing -If displaced → ORIF

Common Hip Disorders				
Injury Type	**Information**	**Signs & Symptoms**	**Workup**	**Management & Prognosis**
Avascular necrosis	-Compromise of bone vasculature → death of bone and marrow cells -Causes: femoral neck fx, dislocation, minor trauma, steroid administration, alcohol use, sickle cell, SLE, radiation	-Groin, thigh, or buttock pain -Weight-bearing pain -Rest and night pain	-Radiograph: can remain normal for months after symptom onset, early findings are mild density changes followed by sclerosis and cysts, pathognomonic crescent sign from subchondral collapse -MRI if radiograph is nondiagnostic	-Goal is to preserve native joint for as long as possible
Developmental dysplasia	-Pediatric disease -Abnormal development of acetabulum and proximal femur with mechanical instability of the hip joint -Risk factors: breech, FH, female	-Abnormal newborn hip exam (Barlow-Ortolani) -Abnormal leg creases -Excessive lordosis -Trendelenburg gait	-US is initial imaging of choice	-Referral to orthopedic surgeon
Groin pull	-Strain of hip adductors from forced abduction during fall or collision		-R/o hernia or torsion	-Rest -Ice -Stretching & strengthening
Hip pointer	-Contusion of the iliac crest from direct blow	-Swelling, tenderness, ecchymosis at iliac crest	-X-ray to r/o fracture	-Pain meds -Ice -Compression -Progressive stretching
Legg-Calve-Perthes disease	-Pediatric disease -Avascular necrosis of the femoral head	-Pt is usually age 2-11 -Limp -Insidious groin and thigh pain -Loss of internal and external hip rotation	-X-ray shows mottled femoral head 	
Slipped capital femoral epiphysis	-Occurs when femoral head is displaced from the femoral neck -Obese, hypogonadic adolescent males are at increased risk 	-Unilateral or bilateral, with many uni cases progressing to bi -Limp -Affected leg turns out and appears shorter -Loss of hip flexion, internal rotation, and abduction	-X-ray  	-An orthopedic emergency, requires surgical repair

Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Snapping hip syndrome	-When ITB or iliopsoas tendon snaps over ASIS	-Hip pain that is worse with activity -Snapping with flexion -Increased pain with resisted hip flexion		-Ice -Activity modification -Strengthening & stability -Injections
Trochanteric bursitis	-Inflammation of burse of greater trochanter	-Extreme point tenderness over bursa -Pain at night when lying on affected side -Pain with hip flexion and extension -Crepitus over greater trochanter		-Hip stretching -NSAIDs -Injection

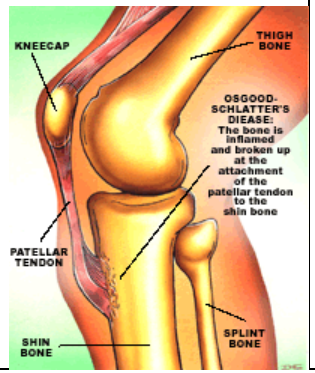
DISORDERS OF THE KNEE

Knee Dislocations & Fractures

Injury Type	Information	Injury Type	Information
Patellar Fracture	-MOI: direct blow -S/s: knee pain, difficulty walking, swelling and bruising, point tenderness -Aspiration will show hemarthrosis with fat globules -Lower extremity immobilization and no weight bearing 		-MOI: impact, direct axial load, or shearing force -Lower extremity immobilization and no weight bearing 
Patellar dislocation	-AP, lateral, merchant, sunrise, and Laurin x-ray views -Tx: extension brace, quad strengthening, consider surgical repair with multiple recurrences 		

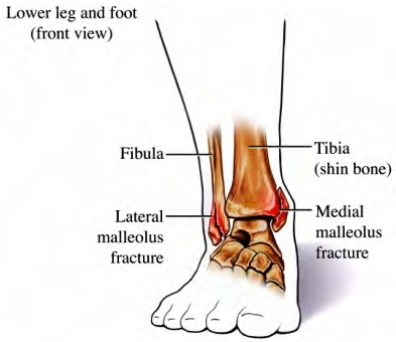
Common Knee Disorders

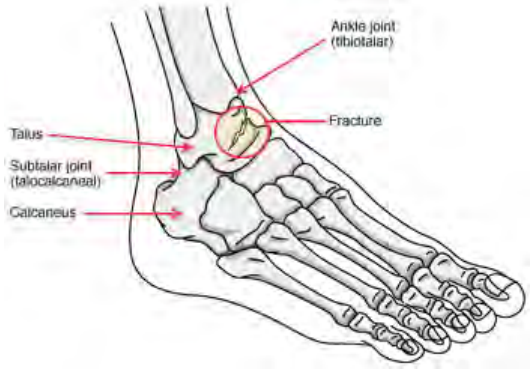
Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Torn meniscus		-Knee swelling, locking, and catching -May have Baker's cyst	-Positive Apley's grind test	
MCL tear	-MOI: valgus stress	-Medial pain over joint line	-Positive valgus stress test -Graded depending on degree of tear	-NSAIDs -Rest -Bracing -PT -Surgery is rarely required

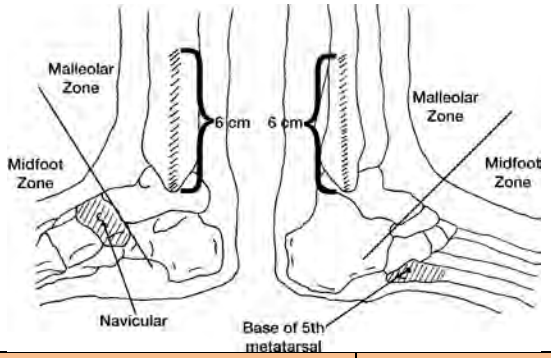
LCL tear	-MOI: varus stress		-Positive varus stress test -Graded depending on degree of tear	-Conservative vs surgical depending on degree of tear
ACL tear		-May have associated Segond fracture (avulsion fx of tibial plateau)	-MRI to r/o other causes of injury	-PT prehab -NSAIDs -Surgical repair with postop brace
PCL tear			-Positive posterior drawer, recurvatum, and quad active tests	
Plica syndrome	-When folds of synovial membrane get stuck in joint spaces → catching	-Painful snapping -Local swelling -Palpable plica	-MRI to r/o other causes of injury	-PT -Injection -Icing after exercise -Surgical excision -NSAIDs
Osteochondritis dissecans	-Avascular necrosis → death of bone → loss of support for articular cartilage → particles of bone and cartilage rub around in joint space	-Swelling after exercise -Small effusion -Locking and catching -Tender femoral condyles -Vague pain		-No weight bearing for 6+ weeks
IT band syndrome	-Irritation of ITB due to rubbing at the femoral head or lateral femoral epicondyle	-Snapping hip or knee -Instability		-PT -Injections -Orthotics -Changing running surface or shoes
Patellofemoral syndrome	-Pain involving the patella and retinaculum -Usually an overuse injury	-Anterior knee pain that may be acute or gradual -May be precipitated by trauma -Exacerbated by squatting, running, prolonged sitting, or when climbing or descending stairs -Tight hamstrings	-Positive patellofemoral compression test -Positive patellar glide test	-PT -Short-term NSAIDS
Osgood-Schlatter disease	-MOI: anterior tibial tuberosity avulsion due to overuse -Most common in males age 10-14	-Anterior knee pain that increases gradually over time -Worse with kneeling, running, jumping, squatting, or stairs -Relieved by rest -Recent growth spurt -Recent increased activity -Localized pain and swelling -Step-offs	-X-ray to rule out fracture 	-Self-limiting, pain typically subsides after closure of the tibial growth plate at 14-18 years of age -Activity as tolerated -Stretching, strengthening, and icing -Patellar brace

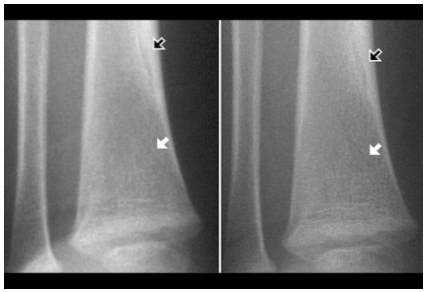
DISORDERS OF THE LEG, ANKLE, AND FOOT

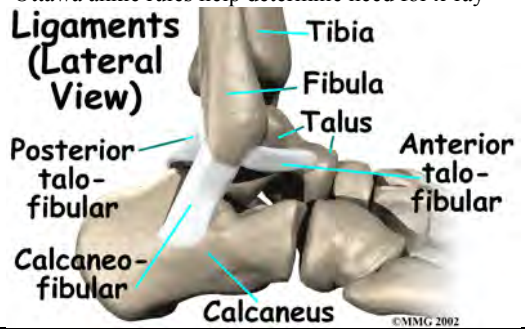
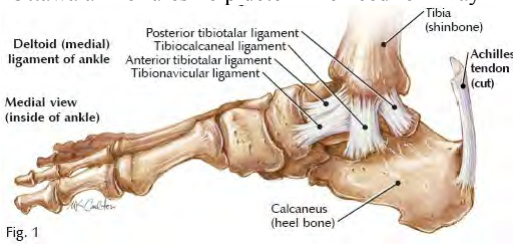
Fractures

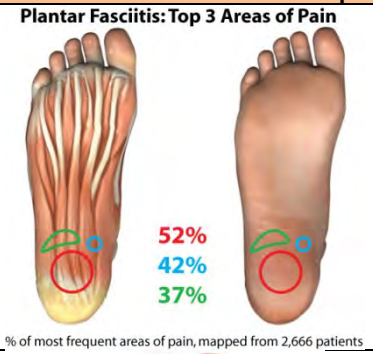
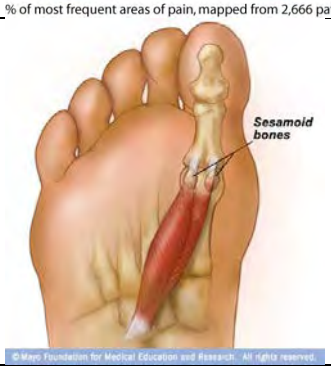
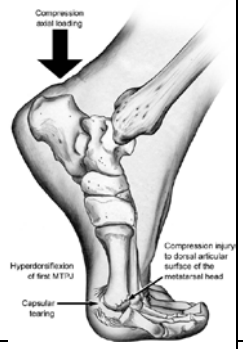
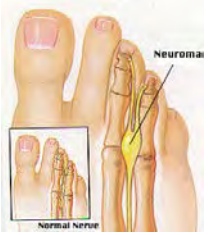


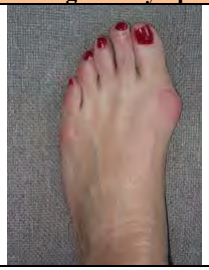




Fracture Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Ankle Fracture	-Involves lateral, medial, or posterior malleolus -MOI: eversion or lateral rotation on the talus	-Tenderness in these areas suggests fracture vs strain or sprain (Ottawa ankle rules)	-Ottawa ankle rules help determine need for x-ray -Standard AP and lateral views on x-ray (plus AP view with 15° internal rotation if suspecting ankle fracture) -Beware commonly missed "FLOAT" fractures	-Elevation and ice -Short leg cast
Foot Fracture	-Involves talus, calcaneus, metatarsals, or phalanges			
Toddler Fracture of Distal Tibia	-Spiral fx of distal tibia -Typically in 1-3 year olds -Salter-Harris classification	-Limp -Refusal to bear weight -May not be painful	-X-ray: may show subtle fracture only on 1 view 	-Long-leg casting

Common Ankle and Foot Disorders				
Injury Type	**Information**	**Signs & Symptoms**	**Workup**	**Management & Prognosis**
Lateral ankle sprain	-Usually an ATFL injury -The most common ankle injury -MOI: ankle inversion → stretch or tear of lateral ligaments		-Ottawa ankle rules help determine need for x-ray 	-PRICE -NSAIDs -Crutches until weight bearing without limp -MRI for symptoms persisting beyond 6-8 weeks -Prevent recurrence with lace-up supports and PT
Medial ankle sprain	-From injury to deltoid ligament -Rare!	-May have medial fracture such as Maissoneuve fx	-Ottawa ankle rules help determine need for x-ray  Fig. 1	-Stretching and band exercises
Syndesmosis sprain	-AKA "high ankle sprain"	-Minimal swelling -Pain over ATFL	-Positive squeeze test -Positive external rotation test	-Refer to ortho -Non-weight-bearing followed by walking boot
Achilles rupture		-H/o shot-like sound followed by pain	-Positive Thompson test	-Serial casting with PT -Surgical repair
Achilles tendonitis		-Gradual onset of posterior pain with activity		-Short-term heel lift -NSAIDs -Icing after activity -PT
Medial tibial stress syndrome (shin splints)	-Posterior tibial muscle tendonitis	-Pain worse with activity -Point tenderness	-Differential: stress fx if presenting with point tenderness and night pain, compartment syndrome if presenting with numbness, pain, and swelling	-Ice -NSAIDs -Decrease mileage -Orthotics -Stretch & strengthen
Sever's disease (calcaneal apophysitis)	-Repetitive microtrauma to the calcaneal growth plates -Common in 7-15 year olds	-Pain at calcaneus and Achilles tendon insertion -Heel pain worse with activity		-Ice -Massage -Stretch -Heel cups or orthotics
Haglund's deformity (pump bump)	-Overgrowth of bone on lateral and posterior calcaneus due to recurrent friction			-Change shoes -Padding -Ice -Excision of overgrowth

Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Plantar fasciitis	-Excessive pull at origin of plantar fascia on calcaneus → inflammation	-May have heel spur		-Orthotics -Higher heeled shoes -Ice -Heel cord stretch -NSAIDs
Sesamoiditis	-Repeated or direct trauma to the sesamoids at the 1st metatarsal			-Metatarsal lift pad
Turf toe	-1st MTP joint sprain from hyperextension of big toe	-Swelling and tenderness -Decreased ROM at 1st MTP joint	-Graded based on degree of tear 	-PRICEMM
Morton's neuroma	-Fibrosis of plantar nerve from injury such as repeated walking in tight high-heeled shoes	-Feels like walking on a marble		-Injection -Excision

Injury Type	Information	Signs & Symptoms	Workup	Management & Prognosis
Bunion (hallux valgus)	-Lateral deviation of the big toe at the MTP joint -Caused by tight shoes			-Shoe modification -Osteotomy

INFECTIOUS DISEASES

Osteomyelitis

Agents
-GAS or GBS
-*Staph aureus*
-Polymicrobial in IVDU
-H/o direct trauma → *Staph* or *Pseudomonas*
-*Salmonella* in sickle cell patients

Etiologies
-Hematogenous seeding (usually monomicrobial)
-Contiguous spread from adjacent soft tissue or joint (polymicrobial)
-Direct inoculation of bone from trauma or surgery (polymicrobial)

Signs & symptoms
-Onset can be insidious or chronic
-Pain, swelling, tenderness, warmth, overlying cellulitis, fever, chills, n/v
-Progression of diabetic foot ulcer

Workup
-Gold standard is bone biopsy with culture
-Can also treat based on + blood cultures
-CBC showing leukocytosis
-X-rays only good for chronic osteomyelitis
-MRI best modality for obtaining details of bone marrow and soft tissue inflammation

Management
-IV AB for 4-6 weeks
-MSSA → nafcillin, oxacillin, or cefazolin
-MRSA or *Staph epidermidis* → vanco
-Surgical draining and debridement
-Gram negs → cipro, levo, ceftazidime, cefepime
-Empiric → vanco + Zosyn

Septic Arthritis

Agents
-*Staph aureus*
-Streptococci
-*Kingella kingae* in kids
-*Neisseria gonorrheae*
-Syphilis

Etiologies
-Hematogenous spread (most cases)
-Bite or other trauma
-Joint surgery
-Spread of infection from adjacent bone

Signs & symptoms
-More likely to manifest in joint with previous arthritis
-Usually monoarticular in nongonococcal arthritis (usually knee)
-Fever, chills, n/v
-Overlying cellulitis

Differential diagnosis of acute monoarthritis

Infection	**Tumor**
Bacterial	Pigmented villonodular synovitis
Fungal	Chondrosarcoma
Mycobacterial	Osteoid osteoma
Viral	Metastatic disease
Spirochete	**Systemic rheumatic disease**
Crystal-induced	Rheumatoid arthritis
Monosodium urate	Spondyloarthropathy
Calcium pyrophosphate dihydrate	Systemic lupus erythematosus
Hydroxyapatite	Sarcoidosis
Calcium oxalate	**Osteoarthritis**
Lipid	Erosive variant
Hemarthrosis	**Intraarticular derangement**
Trauma	Meniscal tear
Anticoagulation	Osteonecrosis
Clotting disorders	Fracture
Fracture	
Pigmented villonodular synovitis	

Workup
-Synovial fluid aspiration
-Plain radiographs in kids to ID joint

-GC test

Management
-IV antibiotics for 4-6 weeks based on Gram stain

NEOPLASTIC DISEASE

Bone Tumors

-Lesions are classified according to matrix produced

Benign lesions
-Osteoid osteoma
-Osteoblastoma
-Osteochondroma
-Solitary enchondroma
-Enchondromatosis
-Periosteal chondroma
-Chondroblastoma
-Chondromyxoid fibroma
-Fibrous dysplasia
-Osteofibrous dysplasia
-Nonossifying fibroma
-Unicameral bone cyst (simple bone cyst)
-Aneurysmal bone cyst
-Langerhans cell histiocytosis
-Giant cell tumor

Malignant lesions
-Osteosarcoma: peak age 13-16
-Chondrosarcoma
-Ewing's sarcoma
-Angiosarcoma
-Fibrosarcoma/malignant fibrous histiocystoma
-Chordoma
-Adamantinoma

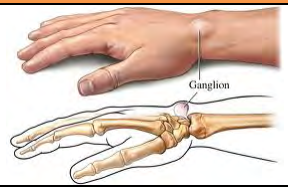
Signs & symptoms
-Usually asymptomatic and discovered incidentally
-Localized pain, swelling, or deformity
-Pathologic fracture
-Aggressive or malignant lesions may have lung mets

Workup
-X-ray
-Lesion biopsy if in doubt of nature

Management
-Surgical excision for giant cell tumor due to aggressive natu
-Surgical management of malignant lesions +/- radio or chemotherapy

Ganglion Cysts

-Swelling in wrist from leakage of joint fluid



Signs & symptoms
-Commonly occur on the wrist, finger joints, or top of foot
-Swelling
-Pain
-Difficulty moving joint

Workup
-Light penetration test

Management
-Drainage
-Surgical excision

OSTEOARTHRITIS (DEGENERATIVE JOINT DISEASE)

Causes
-Primary
-Secondary: due to trauma, congenital disorder, crystal disease, or other bone/joint disorder

Risk factors
-Age > 40
-Female
-Obesity
-Inadequate exercise
-Repetitive low-impact exercise only on neuroanatomically abnormal joints
-Repetitive high-impact exercise on normal joints
-FH of OA

Signs & symptoms
-May be localized or generalized
-Commonly affected joints: cervical or lumbar spine, 1st CMC, PIP (Bouchard's nodes, also associated with RA), DIP (Heberden's nodes), hip, knee, subtalar joint, 1st MTP
-Rarely affected: shoulder, wrist, elbow, MCP joints
-Pain that is typically exacerbated by activity and relieved with rest
-Morning stiffness that resolves < 30 min after awakening
-Gelling
-Crepitus
-Bony enlargement
-Decreased ROM
-Malalignment
-Tenderness to palpation
-May have comorbid CPPD

Differential
-CPPD
-Inflammatory osteoarthritis
-Connective tissue disease: RA, SLE, scleroderma, Sjogren's
-Trauma
-Avascular necrosis
-Sickle cell
-Tendonitis
-Bursitis
-Polymyalgia rheumatica
-Reflex sympathetic dystrophy
-Seronegative spondyloarthropathy: reactive arthritis, psoriatic arthritis, ankylosing spondylitis, IBD
-Infectious arthritis: septic arthritis, Lyme, hep B or C, parvo B19, rubella

Clinical distinction between rheumatoid arthritis and osteoarthritis

Feature	Rheumatoid arthritis	Osteoarthritis
Primary joints affected	Metacarpophalangeal	Distal interphalangeal
	Proximal interphalangeal	Carpometacarpal
Heberden's nodes	Absent	Frequently present
Joint characteristics	Soft, warm, and tender	Hard and bony
Stiffness	Worse after resting (eg, morning stiffness)	If present, worse after effort, may be described as evening stiffness
Laboratory findings	Positive rheumatoid factor	Rheumatoid factor negative
	Positive anti-CCP antibody	Anti-CCP antibody negative
	Elevated ESR and C reactive protein	Normal ESR and C reactive protein

Workup
-Arthrocentesis if pain is severe or acute: synovial fluid aspirate will show clear fluid, few WBCs, normal viscosity, no crystals
-X-ray will show joint space narrowing, osteophytosis, subchondral sclerosis, subchondral cysts
-Chondrocalcinosis is a sign of a metabolic, endocrine, or heritable disorder predisposing to OA
-Normal ESR/CRP, RF, anti-CCP

Nonpharmacologic management
-Beneficial nonpharmacologic therapy: exercise programs, weight loss, wedged sole inserts, canes to offload weight, moist heat
-Benefit of acupuncture or TENS therapy is controversial

Topical drugs
-Topical NSAID benefit appears to wane after several weeks of use
-Topical capsaicin appears to have symptomatic benefit

Oral drugs
-DOC is acetaminophen 650 mg q 6 hours or 1000 mg TID
-NSAIDs shown to be better at relieving overall pain but have greater GI risks = consider only for failed acetaminophen or mod-severe pain
-Avoid indomethacin for long-term treatment of hip OA (↑ joint damage)
-Only use COX-2 for severe GI risks factors and no CV risk factors
-Tramadol is useful as add-on therapy to acetaminophen, NSAID, or COX-2
-Opioid analgesics should be avoided long-term
-Colchicine for frequent acute inflammatory episodes that don't respond to NSAIDs, injections, or joint irrigation
-Investigational meds: doxycycline (anti-inflammatory)

Supplements
-Glucosamine and chondroitin appear to have little clinically relevant benefit

Injections
-Intraarticular glucocorticoids can be useful in painful joints despite NSAID use or when NSAIDs are contraindicated
-Intraarticular hyaluronans may have some benefit

Prognosis
-Course is generally slowly progressive

OSTEOPOROSIS

Risk factors
-Meds associated with bone loss: glucocorticoids, anticoagulants, anticonvulsants, aromatase inhibitors, cyclosporine, tacrolimus, GnRH agonists, barbiturates, Li, Depo-Provera, chemo, TPN
-Previous fracture
-Parental history of hip fracture
-Low body weight
-Current cigarette smoking
-Excessive alcohol consumption
-Rheumatoid arthritis
-Hypogonadism, premature menopause, malabsorption, chronic liver disease, or IBD
-Advancing age

Prevention
-Ca and vit D supplementation recommended for all patients with inadequate intake
-Ca carbonate: Tums, Caltrate, Os-Cal, Viactiv → should be 500-600 mg BID
-Ca citrate: Citracal → should 215 mg QID
-Vit D supplements: should be 800-1000 IU daily
-Exercise, smoking cessation, counseling on fall prevention, avoidance of heavy alcohol use
→ Also recommended for all postmenopausal women with osteoporosis

Screening
-DEXA preferred over peripheral measurements
-Screen women of average risk > 65 with DEXA
-Screen women younger than 65 who have risk factors
-Screening for men generally not recommended unless there is evidence of radiographic osteopenia, h/o trauma fx, loss of > 1.5 in in height, taking risky meds, androgen deprivation therapy for prostate cancer, hypogonadism, hyperthyroidism, etc.
-Women with initially good DEXA results need not be screened again for 10-15 years
-Women with osteopenia on their initial DEXA should be re-screened anywhere from 1-5 years later, depending on how low their T-score was

Signs & Symptoms
-Low trauma fracture
-Decreasing height

Workup
-DEXA: diagnostic if BMD is < 2.5 SD below the young normal mean at the hip or spine
-Osteopenia is diagnosed if the BMD is 1.0-2.5 SD below the young normal mean
-If premenopausal, also need to check CMP, CBC, Ca, P, vit D, TSH, 24 hour urine for Ca and Cr

Management of low T score
-Treat all postmenopausal women with established osteoporosis, fragility fracture, and select postmenopausal women with osteopenia with pharmacologic therapy
-First-line therapy is oral bisphosphonates, 2nd line is raloxifene
-Re-check DEXA after 2 years

Bisphosphonates
-MOA: inhibit bone resorption
-Alendronate, resideronate, ibandronate, zoledronic acid
-Must take on empty stomach and wait 30-60 minutes before eating and drinking while sitting upright (causes acid reflux)
-AEs: hypocalcemia, dysphagia, esophageal inflammation, gastric ulcer, visual d isturbance, arthralgia, HA, myalgia, fever after first dose, possible atypical femoral fx = take a break every 5 years, a-fib?, possible osteonecrosis of the jaw in cancer pts receiving IV treatment
-Contraindications: inability to sit upright for 30 min, esophageal strictures, hypocalcemia

PTH Antagonists
-Calcitonin: comes in a nasal spray
-Teriparatide: costs $950 per month, AEs

Hormonal
-Raloxifene: black box warning for risk of DVT and fatal stroke with women with CHD
-AEs: DVTs, hot flashes, edema, arthralgia, flu syndrome, MI, breast cancer, stroke

Monoclonal AB
-Prevent osteoclast formation
-Costs $825/injection
-Lots of AEs

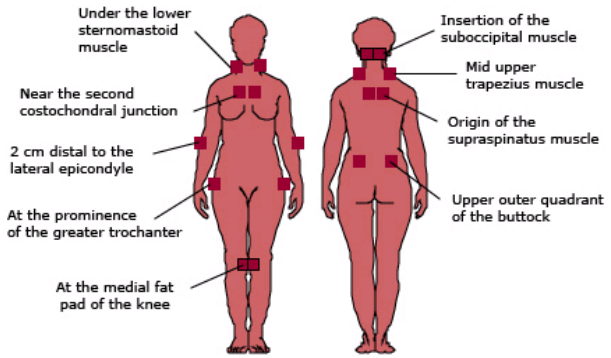
RHEUMATOLOGIC CONDITIONS

Fibromyalgia

-Chronic myalgias and arthralgias in the absence of joint or muscle inflammation on physical exam or laboratory findings
-Thought to be caused by alterations in CNS pain processing as well as genetic and environmental factors

Signs & symptoms
-Multiple tender points at specific soft tissue locations
-Comorbid sleep disorder, depression, anxiety, inflammatory rheumatic disease, or noninflammatory msk pain

Tender points in fibromyalgia



The 18 "tender points" important for the diagnosis of fibromyalgia. Note the bilateral symmetry of the labeled regions. Tenderness on palpation of at least 11 of these sites in a patient with at least a three month history of diffuse musculoskeletal pain is recommended as a diagnostic standard for fibromyalgia.

Differential
-RA
-SLE
-Sjogren's
-Ankylosing spondylitis
-Polymyalgia rheumatica
-Inflammatory myopathy
-Hypothyroidism
-Peripheral neuropathy
-Multiple sclerosis
-Myuasthenia gravis
-Myofascial pain syndrome
-Functional somatic syndromes
-IBS
-Migraine
-Chronic fatigue syndrome
-Myofascial pain: will be localized to one anatomic region

Workup
-CBC
-ESR or CRP
-TSH
-BMP

Management
-Treatment is individualized
-Education about uncertain natural history of fibromyalgia
-Exercise
-Initial DOCs are amitriptyline, duloxetine (good for fatigue), milnacipran, or pregabalin (good for sleep)
-Cyclobenzaprine is an alternative
-No evidence for opioids
-Combination drug therapy for refractory pain, such as SNRI + pregabalin, or fluoxetine + amitriptyline
-Continued refractory pain → refer for CBT or to specialist such as rheum for further w/u

Symptoms that distinguish between chronic fatigue syndrome, fibromyalgia, and temporomandibular join disorder

Symptoms common to all three conditions
Muscle pain, aching, or discomfort
Problems falling or staying asleep, or sleeping too much
Wake up feeling tired, unrefreshed after a full night's rest
Difficulty concentrating or thinking, forgetfulness
Abdominal pain relieved by a bowel movement
Hard, loose, or watery stools
Symptoms that distinguish CFS and FM from TMD
Fatigue greater than six months
Fatigue resulting in a 50 percent reduction of normal activity
Unexplained muscle weakness
Migratory arthralgias without redness or swelling
Burning, shooting, or throbbing muscle pain
Symptoms that distinguish CFS from FM and TMD
Mild fever (37.5°C to 38.6°C) or chills
Sore throat
Symptoms that distinguish FM from CFS and TMD
Pain made better by heat or massage
Pain made worse by sitting or standing
Symptoms that distinguish TMD from CFS and FM
Pain in jaw muscles, temporomandibular joint, or inside the ear

CFS: chronic fatigue syndrome; FM: fibromyalgia; TMD: temporomandibular joint disorder.

Gout

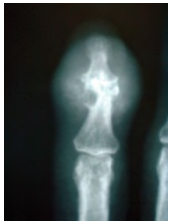
-Monosodium urate deposition → arthritis and inflammation
-Peak incidence in males ages 40-50
-Risk factors: infection, trauma, weight loss, hospitalization, dyslipidemia

Etiologies
-Usually a result of decreased renal clearance of uric acid (alcohol, CKD, low urine vol, HTN, diuretics, aspirin, vasopressin, lactic acidosis, myxedema, respiratory acidosis, preeclampsia, MI, renal insufficiency)
-Sometimes due to increased renal production of uric acid (purine consumption, alcohol use, myeloproliferative disorders, polycythemia, leukemia, EBV, psoriasis, drugs)
-Also influenced by body type, diet, insulin resistance, CHF, and organ transplantation

Stages of gout
1.) Hyperuricemia: typically asymptomatic
2.) Acute gout: development may take decades after onset of hyperuricemia
3.) Intercritical gout: period between attacks
4.) Chronic tophaceous gout: caused by repeated attacks → resembles RA with chronic symmetric polyarthritis and tophaceous deposits, less sudden attacks that take longer to resolve

Signs & symptoms
-First attack is usually podagra (sudden inflammation of the 1st MTP)
-Warmth, swelling, dusky red appearance just like a septic joint
-Usually manifests at night due to fluid shifts
-Systemic signs may be present
-Maximum severity in 12-24 hours
-If chronic: tophi in articular structures, tendons, bursae, or bone

Differential
-Septic arthritis
-Trauma
-CPPD/pseudogout
-Other inflammatory arthritis



Workup
-Joint aspiration with Gram stain, culture, and microscopy is mandatory if possible for all new cases of monoarticular inflammation, will see urate crystals in joint fluid
-Otherwise can diagnose clinically using Rome, New York, or ACR diagnostic criteria
-Plain films will show well-defined erosions in random distribution with overhanging edges a well as tophi
-MRI only used to detect early changes

Management
-Do not treat asymptomatic hyperuricemia!
-Acute attack → continue taking any gout prophylaxis meds, treat arthritis first with NSAIDs, consider giving colchicine for continued symptoms, then treat the hyperuricemia 24 hours after resolution of attack (allopurinol ± probenecid, pegloticase, febuxostat)
-Oral or injectable steroids only for suboptimal response to NSAIDs & colchicine
-Consider pharmacologic prophylaxis with uric acid lowering agents (and colchicine bridge if needed to avoid ppt attack) if > 2 attacks per year, x-ray erosions, nephrolithiasis or uric acid nephropathy, or chronic polyarticular gout (wait at least 4-6 weeks after most recent attack and treat for 3-12 months until uric acid goal is reached then d/c)

Prognosis
-Without urate-lowering treatment there is greater risk of tophus formation, development of chronic gouty arthritis, nephrolithiasis, and chronic urate nephropathy

Juvenile Rheumatoid Arthritis

-Classification of subtypes is still a work in progress
-Most commonly used classification is ILAR, with further classification of each group based on age at onset, duration and pattern, and presence of ANA or rheumatoid factor

ILAR Classification of Idiopathic Arthritides of Childhood

Group	Information	Signs & symptoms	Differential	Workup	Management	Prognosis
Systemic arthritis	-An autoimmune condition probably unrelated to other forms of childhood arthritis, requiring different therapy -Accounts for 10-20% of cases -Can present in kids as young as 1	-High fever -Macular, salmon pink rash related to fever spikes -Hepatomegaly -Lymphadenopathy -Arthralgias typically in the wrists, knees, and ankles	-Postinfectious arthritis -Reactive arthritis -SLE and other connective tissue diseases -Malignancy -Malaria	-Diagnosis is clinical, based on presence of intermittent fever for at least 2 weeks and arthritis -Labs will show increased WBCs, thrombocytosis, anemia, high ESR	-NSAIDs for 6-12 weeks for mild and nondisabling symptoms -Add steroid taper or biologics for severe cases or for those unresponsive to NSAID trial, followed by DMARD -Anticytokine therapy for refractory disease	-Follows one of three patterns: systemic symptoms and no progressive arthritis, persistent systemic symptoms and progressive arthritis, or resolution of systemic symptoms with progressive destructive arthritis
Polyarthritis	-Involves at least 4 joints during first 6 months of illness	-Younger kids: begins with 1-2 affected joints then spreads -Older kids: rapid onset in multiple joints -Usually symmetric -Sausage fingers -Uveitis	-Reactive arthritis -Psoriatic arthritis -Spondyloarthropathy -SLE -Systemic vasculitis -Sarcoidosis -IBD -Epiphyseal dysplasia -Minocycline-induced autoimm	-No characteristic labs, may have elevated ESR, anemia	-NSAID trial for 3 weeks, followed by a different NSAID if no response -Methotrexate or biologic	-Will be chronic and progressive without treatment

Group	Information	Signs & symptoms	Differential	Workup	Management	Prognosis
Pauciarthritis (oligoarthritis)	-Involvement of < 5 joints during the first 6 months of disease onset -May involve more joints over time (= extended pauciarthritis)	-Limping without complaint -Usually large joints are affected but not the hips -Swollen, tender joints	-Psoriatic arthritis -Enthesitis-related arthritis -Infection -Malignancy	-Diagnosis is clinical based elimination of other causes and on presence of arthritis in a single joint for at least 3 months or 2+ joints for at least 6 weeks -ANA is usually + -No rheumatoid factor	-NSAIDs -Intraarticular steroids -Methotrexate or biologics rarely required	-Many cases resolve within 6 months -May recur -Uveitis is the most serious complication and occurs in 20% of cases
Enthesitis-related arthritis	-Includes childhood spondyloarthropathies	-Arthritis + enthesitis -Arthritis + 2 or more of the following: SI joint tenderness, inflammatory spinal pain, FH, uveitis, + HLA-B27 -Gradual onset that may first be recognized following fever or msk trauma			-NSAIDs for 3-6 months: frequently diclofenac or piroxicam are used -Sulfasalazine, biologics, or DMARDs if no improvement	-May progress to psoriatic arthritis
Psoriatic arthritis	-Psoriasis + arthritis	-May need to search for hidden psoriasis lesions -Joints tend to be less tender than other inflammatory arthritides -Nail pitting or onycholysis -Pitting edema -Uveitis -Dactylitis	-Reactive arthritis -Ankylosing spondylitis	-Usually seronegative -No specific tests	-NSAIDs: typically don't induce remission -Steroid injections into joints -DMARDs -Add second DMARD or biologic if needed -Monitor for uveitis	-Clinical remission achieved in most patients after 5 years of treatment

Polymyositis		
-A persistent inflammatory muscle disease that causes proximal weakness of the skeletal muscles -Caused by killer T-cells attacking muscle cells expressing MHC class I (slow fibers) → muscle fiber necrosis, degeneration, and inflammatory cell infiltration -Related to dermatomyositis and inclusion body myositis -May be triggered by certain cancers	**Signs & symptoms** -Insidious onset with nonspecific symptoms -Proximal muscle weakness (can't get up from chair, can't raise arms above head) -Muscle atrophy if long-standing -Dysphagia, nasal regurgitation, aspiration -Sclerodactyly -Low-grade fever -Peripheral lymphadenopathy -Interstitial lung disease -Frequent co-occurrence with other systemic autoimmune diseases, another connective tissue disease, or bacterial or viral infection	**Workup** -Elevated CK, LDH, aldolase, and LFTs -Autoantibodies (anti-Jo) and + ANA -EMG alteration -Positive muscle biopsy **Management** -High dose steroid taper -DMARDS for patients unresponsive to steroids

Vasculitis Syndromes

-The vasculitides are characterized by inflammatory leukocytes in vessel walls with reactive damage to mural structures
-Can be a primary or secondary process
-May case bleeding and compromise of lumen → downstream tissue ischemia and necrosis
-Affected vessels vary in size, type, and location

Types
-Large vessel: Takayasu arteritis, giant cell arteritis
-Medium vessel: polyarteritis nodosa, Kawasaki disease, primary CNS vasculitis
-Small vessel: Churg-Strauss, granulomatosis with polyangiitis (Wegener's), microscopic polyarteritis, Henoch-Schonlein purpura, essential cryoglobulinemic vasculitis, hypersensitivity vasculitis, vasculitis secondary to connective tissue disease, vasculitis secondary to viral infection
→ Criteria for classification of the major forms of vasculitis have been established but only include characteristics that help distinguish one disorder from other vasculitides = good for research but not very helpful clinically

Signs & symptoms
-Systemic symptoms in combination with evidence of single or multiorgan dysfunction
-Nonspecific: fatigue, weakness, fever, arthralgias, abdominal pain, HTN, renal failure, neurologic dysfunction
-Specific: mononeuritis multiplex, palpable purpura, combined pulmonary and renal involvement

Differential
-Fibromuscular dysplasia
-Cholesterol emboli
-Infective endocarditis
-Malignancy
-Mycotic aneurysm with embolization
-Bacteremia
-Rickettsial infection
-Thrombocytopenia
-Radiation fibrosis
-Neurofibromatosis
-Congenital coarctation of the aorta
-Amyloidosis
-Livedo reticularis
-Cocaine abuse
-Hereditary disorders: Marfan, Ehlers-Danlos, etc.
-Atherosclerosis
-Vasospasm

Workup
-Ascertain type of vasculitis: CMP, CK, ESR, hepatitis serologies, UA, CXR, echo; may need CSF, CNS imaging, PFTs, blood or tissue culture
-More specific tests: ANA, complement (deficiency in lupus and mixed cryoglobulinemia), ANCA (Wegener's)
-EMG if neuromuscular symptoms are present
-Tissue biopsy of affected organ is essential for diagnosis
-Arteriogram if suspecting vasculitis of large and medium arteries to look for aneurysms, occlusions, and vascular wall irregularities

Management
-Depends on severity and type of vasculitis
-Stop offending drugs
-Antihistamines, NSAIDs, or steroid course for inflammation
-Monitoring with US

Prognosis
-60-80% 5 year survival with polyarteritis nodosa
-60% survival with Churg-Strauss
-75% survival with Wegener's
-85% + survival with hypersensitivity vasculitis, Henoch-Schonlein purpura, giant cell arteritis, Takayasu arteritis

Polyarteritis Nodosa

-Rheumatic vasculitis of medium-sized arteries with occasional involvement of small muscular arteries

Etiology
-Most cases are idiopathic
-Hep B or hep C or hairy cell leukemia are linked to some cases

Signs & symptoms
-Systemic presentation: fever, fatigue, weakness, loss of appetite, weight loss
-Infarctions manifest as renal failure, HTN, edema, oliguria, uremia, skin lesions, arthralgias, myalgias, peripheral neuropathies, MI, CHF, pericarditis, and GI tract issues, but typically spares the lungs
-Skin manifestations include tender erythematous nodules, purpura, livedo reticularis, ulcers, bullous or vesicular eruptions (may be focal or diffuse and can progress to infarction and gangrene)
-Limb edema

Workup
-CBC showing leukocytosis
-↑ESR or CRP
-CMP
-Rheum workup tailored to differential
-Confirmatory diagnosis should be made by biopsying clinically affected organ
-Alternative for dx is arteriography or cross-sectional imaging

Polymyalgia Rheumatica

-Seen mostly in white female patients over age 50

Signs & symptoms
-Pain and stiffness of neck or torso, shoulders or proximal regions of the arms, and hips or proximal aspects of the thighs for at least 1 month
-Most severe in the morning and lasts at least 30 min
-Decreased ROM of the shoulders, neck, and hips
-No weakness or decrease in muscle strength
-Often co-exists with giant cell arteritis

Differential
-Seronegative RA
-Bursitis or tendonitis
-Spondyloarthropathy
-CPPD
-Hypothyroid
-Fibromyalgia
-Malignancy
-Infective endocarditis
-Dermatomyositis or polymyositis
-Vasculitides

Workup
-Elevated ESR/CRP

Management
-Prednisone

Reactive Arthritis (Reiter Syndrome)

-Acute inflammatory arthritis following 1-4 weeks after a GI or GU infection

Agents
-Usually *Chlamydia trachomatis*
-*Yersinia*
-*Salmonella*
-*Shigella*
-*Campylobacter*
-*Clostridium difficile*
-*Chlamydia pneumoniae*

Differential

-Postinfectious arthritis (basically any other organism causing arthritis that isn't on the proven list of reactive arthritis agents)
-Septic arthritis
-Crystal arthritis
-Spondyloarthropathy

Signs & symptoms
-Asymmetric oligoarthritis often affected LEs
-Enthesitis
-Dactylitis ("sausage fingers")
-Inflammatory back pain
-Conjunctivitis or uveitis
-GU symptoms
-Oral mucosal ulcers
-Constitutional symptoms
-Cutaneous manifestations: keratoderma blennorrhagica, circinate balanitis, psoriasis-like nail changes

Workup
-Elevated ESR
-Negative stool and serologies
-Nondiagnostic x-rays
-*Chlamydia* test

Management
-Treat active infection found, such as *Chlamydia*
-NSAIDs at anti-inflammatory doses
-Intraarticular glucocorticoids are 2nd line
-Low dose systemic steroids are 3rd lien
-Nonbiologic DMARD is last resort

Prognosis
-Usually spontaneous remission after 6-12 months
-May evolve to chronic spondyloarthritis

Rheumatoid Arthritis

-Genetic role with strong association with HLA-DR1 and HLA-DR4
-Risk factors: nulliparity, older age, FH, female, cigs, certain infections
-Protective factors: estrogen, tea, high vit D, breastfeeding

Pathophysiology
-Triggering incident → proliferation of macrophages and fibroblasts
-Lymphocytic invasion of the perivascular space
-Local blood vessels become occluded → outpouching of synovial membrane ("pannus") that eventually invades cartilage and bone → release of cytokines, proteases, and IL → further joint destruction by T-cells and macrophages

Signs & symptoms
-Slow, insidious onset with duration of symptoms over weeks to months
-Waxing and waning of symptoms
-Usually > 5 joints involved, with small bones of hands and feet usually the first to be involved, with later progression to larger joints
-Morning stiffness for at least 1 hour
-Aggravated by prolonged periods of rest in the same position
-Fatigue, malaise, low-grade fever, weight loss
-Chronic swelling and joint warmth (but usually no erythema), decreased ROM
-Hands: boutonniere or swan neck deformities of the fingers, ulnar deviation of the fingers
-Feet: subluxation of MTPs → calluses on bottom of feet
-Wrists: synovial proliferation → median nerve compression, extensor tendon rupture
-TMJ syndrome
-Manifestations in the atlantoaxial joint → UE paresis with head movements, pain radiation to occiput
-Less common manifestations: palindromic rheumatism, monoarthritis of large joint, extra-articular manifestations (skin, CV, pulm, eye, neuro, heme, renal, bone)

Differential
-Post-infectious sequelae
-Other systemic rheumatic disease: SLE
-Lyme arthritis
-Fibromyalgia -Psoriatic arthritis
-IBD-associated arthritis
-CPPD
-Polyarticular gout

Workup
-Bilateral radiographs of hands, wrists, and feet → imaging shows bony erosion with preservation of joint space
-Rheum labs: ESR and CRP, RF, ANA, anti-CCP
-Arthrocentesis if diagnosis is uncertain
-Diagnostic criteria: inflammatory arthritis of 3+ joints, +RF or +anti-CCP, ↑CRP or ESR, symptoms > 6 weeks, exclusion of other diseases on differential (however, it is possible to have seronegative RA)

Management
-Goals are to prevent further joint damage, prevent loss of function, decrease pain, control systemic complications, and maximize quantity of life
-NSAIDs should not be used alone as they don't alter disease course
-Glucocorticoids are usually used long-term-DMARDS should be started within 3 months of diagnosis
-PT/OT
-Survey for infections, malignancy, osteoporosis, and depression

Prognosis
-Disease will be lifelong with 3-5 year reduction in life expectancy
-Spontaneous remission can occur
-Complications: infection with unusual pathogens from tx, Felty's syndrome, Bakers cysts, risk of malignancy

DMARDs

Agent & MOA	**Methotrexate:** inhibits difolate reductase in WBCs	**Hydroxychloroquine:** interferes with Ag presentation	**Sulfasalazine:** impairs lymphocyte transformation and suppresses NK cells	**Leflunomide:** inhibits synthesis in WBCs	**Biologics**
Info	-First-line DMARD -Best for moderate-severe disease -Slows radiographic damage and may reduce mortality	-The best-tolerated DMARD, best for mild disease -Takes 1-6 weeks to work -Does not slow radiographic damage so should not be used alone	-Good for mild disease -Takes 1-3 months to work -Slows radiographic progression	-Can be used alone or with methotrexate	-Anti-TNFs -Costly -Used in combination with other DMARDs
Risks & AEs	-AEs: n/v/d, anorexia, alopecia, rash, myelosuppression, liver or renal failure, hyperuricemia, oral ulcers, cough, SOB → need to monitor LFTs, CBC, Cr, CXR, liver biopsy every 1.5 g -Pregnancy X -Interactions with NSAIDs, penicillins	-AEs: n/v/d, myopathy, HA, retinopathy, agranulocytosis, skin pigmentation → monitor with eye exams, CBC, neuro exam	-AEs: HA, photosensitivity, rash, n/v/d, anorexia, myelosuppression, liver and kidney failure, oligospermia → monitor CBC, LFTs, BMP -Interactions with thiazides and warfarin	-AEs: diarrhea, weight loss, HTN, alopecia, rash, elevated LFTs → monitor LFTs, Cr, CBC, signs of infection, pregnancy tests -Pregnancy X and elimination can take up to 2 years so couples wishing to conceive must undergo a cholestyramine washout -Contraindicated in hepatitis or h/o alcohol abuse b/c it's metabolized in the liver	-AEs: HA, infusion reaction, abd pain, vomiting

Systemic Lupus Erythematosus

-A result of abnormalities in apoptotic cell clearance → generation of autoantibodies to nuclear antigens, phospholipids, and other cell surface proteins
-A type III hypersensitivity
-Hereditary predisposition based on MCH II polymorphisms (HLA-DR2 or HLA-DRR3) or complement deficiencies → can be triggered by an exposure (infection, UV light, drugs, stress) in a genetically susceptible individual
-Most common in nonwhite women 15-40

Signs & symptoms
-Relapsing and remitting symptoms
-Fatigue, low-grade fevers
-Malar rash, discoid rash (differentiate from tinea corporis by its non-fluorescence), psoriasis-like rash
-Skin photosensitivity
-Painless oral or nasal ulcers
-Inflammatory arthritis, arthralgias, tenosynovitis, tendon rupture, osteonecrosis, myositis, myalgias
-Vasculitis
-Serositis around the heart or lung
-Glomerulonephritis
-CNS lupus: seizures, psychosis, transverse myelitis, depression, peripheral neuropathy, optic neuritis
-Autoimmune hemolytic anemia
-Pneumonitis, pulmonary hemorrhage, pulmonary HTN, shrinking lung syndrome
-Myocarditis, CAD
-IBD, pancreatitis, liver disease, lupoid hepatitis
-Lymphadenopathy

Workup
-Findings accumulate over time, dx may take years
-ACR has SLE classification criteria for dx
-CBC, Cr, ESR, CRP, ANA, anti-dsDNA, anti-Sm, anti-RNP, antiphospholipid, anti-Ro, anti-La
-X-rays demonstration Jaccoud's arthropathy (ulnar deviation, MCP subluxation, swan-neck deformities, and diffuse soft-tissue swelling as a result of tendon laxity rather than RA-type bony destruction)
-Head CT may show unidentified bright objects of unknown clinical significance
-UA may show proteinuria and RBC casts if there is lupus nephritis

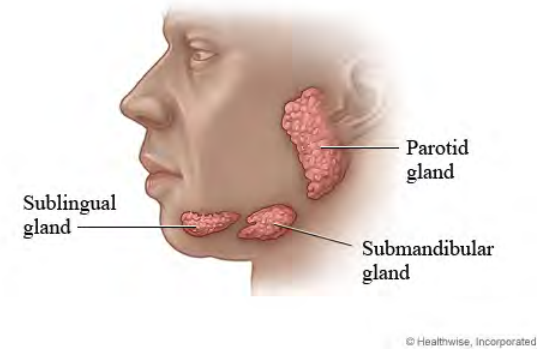
Management
-Always hydroxychloroquine, which is "lupus life insurance"
-Sunscreen
-NSAIDs for msk complaints, fever, HA, and mild serositis
-Systemic steroids for patients with renal, CNS, or other significant organ involvement
-Immunosuppressives (methotrexate, cyclophosphamide, azathioprine, mycophenolate, rituximab, etc) for patients with significant organ involvement and inadequate response to steroids
-CV risk reduction
-For resistant disease, consider high dose chemo followed by autologous stem cell transplant

Sjogren Syndrome

-Inflammatory disorder of the exocrine glands characterized by infiltration of glandular tissue by CD4 T-cells

Types
-Primary Sjogren's: when there is no other autoimmune disease present
-Secondary Sjogren's: when there is another autoimmune disease such as RA or SLE

Signs & symptoms
-Sicca syndrome: dry eyes and mouth
-Gritty or sandy feeling in eyes
-Oral candidiasis
-Parotid or submandibular gland enlargement
-Fatigue
-Myalgias
-Vaginal dryness and dyspareunia
-Recurrent nonallergic rhinitis and sinusitis, dry cough
-Extraglandular organ involvement: cutaneous vasculitis, lupus-like lesions, ILD, CNS and PNS abnormalities, interstitial nephritis



Workup
-Salivary gland biopsy
-Anti-Ro and anti-La, other autoantibodies
-Tests for keratoconjunctivitis sicca: Schirmer test (wetting paper), Rose Bengal stain
-CBC
-CMP
-ESR or CRP
-UA

Management
-Artificial tears or punctal plugs for dry eyes
-Biotene mouthwash
-Frequent water sipping
-Cholinergic agonists
-Hydroxychloroquine for systemic symptoms
-May need systemic therapy if severe

Prognosis
-Increased risk of developing Hodgkin lymphoma if severe

Scleroderma

-A chronic systemic autoimmune disease due to defective fibroblast metabolism → skin changes, fibrosis, vascular alterations, and autoantibodies
-May also involve the lungs, kidneys, heart, GI tract, tendon sheaths, and some endocrine organs
-Increased incidence in women and black patients
-Usually occurs after age 30

Etiologies
-Largely known
-Exposure to environmental triggers in genetically susceptible hosts?

Signs & symptoms
-Usually begins in fingers, hands, and face
-Skin thickening and hardening
-Sclerodactyly
-Edematous swelling and erythema
-Malaise, fatigue
-Arthralgias and myalgias
-Carpal tunnel, trigeminal neuralgia, or tendon rubs
-Raynaud phenomenon
-Pulmonary involvement: ILD, pulm HTN
-Renal disease: albuminuria, HTN, ↑ Cr, scleroderma renal crisis in 10-20% pts
-Cardiac disease
-Myopathies and neuropathies
-HA, seizure, stroke
-Radiculopathy or myelopathy
-GU symptoms
-Gastric antral vascular ectasia
-GI involvement: rigidity and thinning of oral mucosa, GERD, esophagitis, strictures, abnormal motility, malabsorption, bacterial overgrowth, diarrhea

Workup
-Autoantibody serologies
-Serum and urine protein electrophoresis
-High res CT and PFTs for lung concerns

Management
-Antihistamines for pruritus
-Lasering of facial telangiectasias
-Monitor BP, BMP, UA, urine protein
-ACEI if HTN present
-GI involvement may require PPIs, abx, prokinetics, TPN, or surgery
-Acetaminophen or NSAIDs for arthritis
-PPD-5 inhibitors for ED
-ILD: IV monthly cyclophosphamide or azathioprine + low dose steroids, PCP prophylaxis
-Screen for ILD with initial high res CT for all systemic sclerosis pts as well as regular PFTs
-Screen for esophageal dysmotility with manometry
-Screen for pulmonary HTN with echo for all systemic sclerosis pts
-Surgical repair of flexion contractures

Prognosis
-Decreased life span
-Increased risk of cancer

Classification of localized scleroderma

Circumscribed (plaque) morphea
One or more circumscribed patches of skin with sclerotic changes in one anatomic site
Involvement is confined to the superificial panniculus (subcutaneous tissue)
Generalized morphea
Four or more plaques that affect at least two anatomic sites
Often located on the trunk or limbs
Similar clinical and histologic features as circumscribed morphea
Bullous morphea
Bullous involvement
May occur in other forms of morphea
Linear morphea
Most common form of morphea in children
One or more elongated sclerotic areas of skin
Lesions are typically asymmetric and oriented along the affected limb
Associated with growth impairment of the involved extremity
Lesions on the face or scalp are called en coup de sabre
Deep morphea
Least common form of morphea in children
Most disabling form of morphea
Primary site of involvement is the panniculus (subcutaneous tissue)

Classification of systemic sclerosis

Limited cutaneous scleroderma
Raynaud phenomenon for years, occasionally decades
Skin involvement limited to hands, face, feet, and forearms (acral distribution)
Nailfold capillary pattern typical of scleroderma predominantly nailfold capillary loops with capillary drop-out
A significant (10 to 15 percent) late incidence of pulmonary hypertension, with or without skin calcification, gastrointestinal disease, telangiectasias (CREST syndrome), or interstitial lung disease
Renal disease rarely occurs
Anticentromere antibody (ACA) in 50 to 60 percent but other patterns also occurring in 5 to 10 percent (especially anti-PM-Scl and anti-Scl-70)
Diffuse cutaneous scleroderma
Raynaud phenomenon followed, within one year, by puffy or hidebound skin changes
Truncal and acral skin involvement; tendon friction rubs
Nailfold capillary pattern typical of scleroderma with dilatation (early), dilatation and drop-out (active), and tortuosity with drop-out (late)
Early and significant incidence of renal, interstitial lung, diffuse gastrointestinal, and myocardial disease
Anti-Scl-70 (30 percent) and anti-RNA polymerase-I, II, or III (12 to 15 percent) antibodies
Scleroderma sine scleroderma
Presentation with pulmonary fibrosis or renal, cardiac, or gastrointestinal disease
No skin involvement
Raynaud phenomenon may be present
Antinuclear antibodies may be present (anti-Scl-70, ACA, or anti-RNA polymerase-I, II, or III)
Environmentally induced scleroderma
Generally diffuse distribution of skin sclerosis and a history of exposure to an environmental agent suspected of causing scleroderma
Overlap syndromes
Features of systemic sclerosis that coexist with those of another autoimmune rheumatic disease such as systemic lupus erythematosus, rheumatoid arthritis, dermatomyositis, vasculitis, or Sjögren's syndrome.
Pre-scleroderma
Raynaud phenomenon
Nailfold capillary changes (early or active pattern typical) and evidence of digital ischemia
Specific circulating autoantibodies - anti-topoisomerase-I (Scl-70), anti-centromere (ACA), or anti-RNA polymerase-I, II, or III or other hallmark scleroderma reactivity

OTHER MUSCULOSKELETAL TOPICS

Compartment Syndrome

-Occurs when increased pressure within a compartment bounded by fascial membranes compromises the circulation and function of tissues within the membrane

Causes
-Acute: long bone fracture or other trauma, ischemia-reperfusion injury, coagulopathy, venom reaction or other bite, extravasation of IFV, recreational drug injection, prolonged limb compression
-Chronic: overuse injury in endurance athletes

Signs & Symptoms
-Acute: pain out of proportion to the injury, pain with passive muscle stretch, rapid progression of symptoms, motor deficits are a late finding
-Chronic: pain in involved compartment (usually bilaterally) shortly after start of exercise with resolution once exercise has stopped

Workup
-Compartment pressure measures: should be < 30 mmHg difference between compartment pressure and systemic diastolic pressure; one normal reading does not rule out!

Management of acute compartment syndrome
-Remove any dressing, splint, cast, or other restriction
-Do not elevate limb
-Pain management
-Supplemental O2
-IVF for hypotension
-Fasciotomy is definitive treatment

Management of chronic exertional compartment syndrome
-Reduce training volume
-Address strength or flexibility deficiencies
-Orthotics
-Surgical fasciotomy usually successful

Crush Injuries

-Really a result of acute traumatic ischemia
-Rhabdomyolysis from sarcolemma failure → permeability of muscle membranes → leak of myoglobin and K+ out of cell with leak in of water, Ca, Na
-Also have local vasoconstriction and platelet aggregation → ischemia
-Compartment syndrome from increased pressure within muscle compartments → muscle, tissue, and nerve death

Causes
-Building collapse
-Trapped in machinery
-Natural disasters
-MVCs
-Prolonged duration of wearing antishock garment
-Inability to move away from hard surface (CVA, CO, hypoglycemia, fall, etc)

Signs & Symptoms
-Fractures
-Evident soft tissue injury
-Dysrhythmias and EKG changes (peaked T waves, loss of P waves) from electrolyte imbalances
-Red-brown urine
-Compartment syndrome: tight, shiny, pain out of proportion to exam, pallor, pulselessness, paresthesias, paralysis

Workup
-Electrolytes: hyperkalemia, hyperphosphatemia, hypocalcemia
-High myoglobin
-Elevated CK (officially rhabdo if > 5x ULN)
-Elevated Cr due to AKI from trying to clear myoglobin

Management
-ABCs
-Cardiac monitoring
-Fluid resuscitation
-Pain management

Management
-Can give bicarb before extrication to shift K+ intracellularly
-Can give Ca carbonate for K+ cardiac membrane stabilization post-extrication (different IV from bicarb!)
-Give insulin with D50W to shift K+ intracellularly
-Albuterol to raise insulin level → more intracellular K+ shift
-Kayexalate to reduce K+ via GI tract (slower onset of action)
-Remove any constrictive clothing, jewelry, or splints
-Avoid large boluses of fluid if pt is hemodynamically stable
-Mannitol: a non-osmotic diuretic to help wash myoglobin out of renal tubules to protect kidneys
-Compartment syndrome: fasciotomy, hyperbaric oxygen

Prognosis
-Degree of physiologic dysfunction is not related to time elapsed before extrication

EYES, EARS, NOSE, AND THROAT

EYE DISORDERS

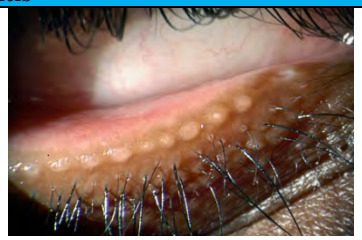
Blepharitis

-Chronic eye condition characterized by lid inflammation with intermittent acute exacerbations

Causes
-Anterior blepharitis: staph colonization or seborrhea
-Posterior blepharitis: meibomian gland dysfunction, rosacea, or seborrheic dermatitis

Signs & symptoms
-Anterior blepharitis: red, itchy eyes with scales along lash bases
-Posterior blepharitis: inflammation of the inner portion of the eyelid at the level of the meibomian glands
-Burning or gritty eyes
-Mattering in the morning

Workup
-Evaluate for sebaceous cell malignancy with unilateral or other unusual symptoms



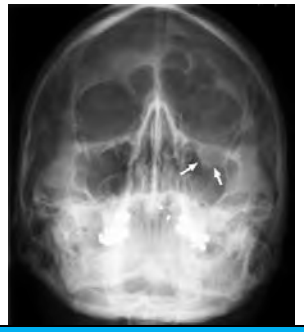
Management
-Regular lid hygiene
-Warm compresses
-Scrub eyelids with baby shampoo

Blowout Fracture

-The most common orbital fx, occurring with blunt force to the globe or orbit rim

Signs & Symptoms
-Anesthesia of the infraorbital area, maxillary teeth, and/or upper lip
-Diplopia
-Rarely enophthalmos (posterior displacement of eyeball)

Workup
-Assess visual acuity and EOM function
-Slip lamp exam to assess for any involvement of the eyeball
-Plain films may show teardrop sign from herniation of orbital fat into the maxillary sinus or open bomb-bay door sign from bone fragments in the sinus
-Confirm abnormal radiograph with CT



Management
-Ophtho consult
-Surgical repair if enophthalmos or persistent diplopia present
-Antibiotic prophylaxis for any sinus involvement

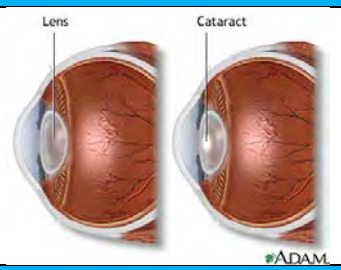
Cataracts

-Opacification of the lens

Etiologies
-Age
-Steroids
-Diabetes
-Electrocution
-Congenital anomaly
-Trauma

Signs & symptoms
-Gradual loss of vision
-Blurred or smoky vision
-Glares
-Decreased vision in bright light or at night

Management
-Surgical removal when it interferes with ADLs with replacement with an artificial lens



Chalazion & Hordeolum

Hordeolum
-Infection of sebaceous or apocrine gland
-Internal or external (= stye, on lid margin)
-Acute onset, lasting 7-10 days
-Tend to be smaller and more painful
-Causes scratchy sensation and blurred vision
-Treat with warm compresses, consider bacitracin or erythromycin ointment
-I&D if not resolving in 48 hours

Chalazion = subacute or chronic
-Sebaceous gland cyst from inflammation of blocked gland
-May follow an internal hordeolum
-Usually point inside the lid
-Usually painless but can become acutely inflamed
-Tend to not resolve without intervention
-Refer to ophtho for surgical excision or corticosteroid injection if not resolved in a few months

Conjunctivitis

Etiologies
-Kids & adults: adenovirus, *Strep pneumo, Haemophilus, Moraxella, Pseudomonas*
-Infant: think *Neisseria gonorrhoeae* or *Chlamydia trachomatis*
-Allergic = conjunctivitis verno
-Conjunctivitis sicca is chronic dry eye related to rheumatic disease

-Hard to distinguish bacterial from viral, all etiologies can cause eyes to be stuck together in the morning
-Bacterial tends to be consistently purulent throughout the day and is usually unilateral
-Viral tends to feel more gritty and usually affects the 2nd eye 24-48 hours later
-Allergic will be ITCHY = pathognomonic

Workup
-Culture if extremely purulent

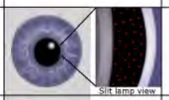
Treatment
-All etiologies are usually self-limiting
-Throw out contact lenses, wash sheets and hands, will be contagious for 2 weeks
-Antibiotics → erythromycin ointment, sulfacetamide drops, FQ drops in contact lens wearers (*Pseudomonas*)
-OTC antihistamine drops for viral causes → Ocuhist, Naphcon-A, Visine AC
-Acute allergy → short-term antihistamine/vasoconstrictor drops like Naphcon-A, Opcon-A, Visine-A
-Chronic allergy → antihistamine + mast cell stabilizer drops like Patanol or Pataday, Optivar, Alocril, Ketotifen, Alamast, Elestat
-Severe allergy: lodoxamide or cromolyn drops
-If no response in 2 days or need for steroid drops refer to ophtho

Hyphema

-Layering of RBCs in the anterior chamber due to blunt or penetrating trauma

Signs and symptoms
-Vision loss
-Eye pain
-N/v
-Microhyphema visible on slit lamp exam if not evident immediately
-Photophobia
-Elevated IOP
-Corneal blood staining

Traumatic hyphema: grading and prognosis

Grade	Anterior chamber filling	Diagram	Best prognosis for 20/50 vision or better
Microhyphema	Circulating red blood cells by slit lamp exam only		90 percent
I	<33 percent		90 percent
II	33-50 percent		70 percent
III	>50 percent		50 percent
IV	100 percent		50 percent

Workup
-Emergent ophtho referral if bleeding dyscrasia, sickle cell, or suspected open globe
-Orbital CT for suspected open globe
-Complete orbital and ocular evaluation required
-Severity determined by grading

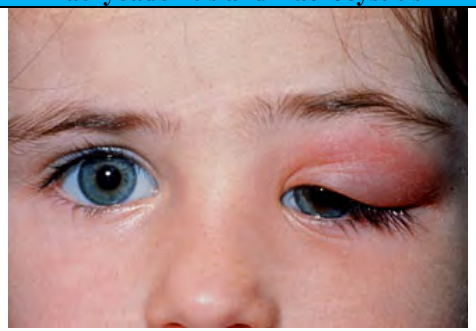
Management
-Eye shield and reading restriction for 1 week or until hyphema resolves
-Elevate head 30° (prevent settling of blood)
-Pain control: cycloplegic eye drops
-Emesis control
-Steroid drops
-Consider antifibrinolytics
-Decrease IOP if needed
-Surgical clot evaluation for large persistent hyphemas > 10 days, early corneal staining, or difficulty controlling IOP

Prognosis
-Can result in vision loss

Dacryoadenitis and Dacrocystitis

-Dacryoadenitis = inflammation or infection of the lacrimal gland from which tears are secreted
-Dacryocystitis = infection within the lacrimal drainage system due to an obstructed lacrimal duct and sac

Etiologies
-Dacryoadenitis: autoimmune diseases, thyroid eye disease, orbital pseudotumor
-Dacryocystitis: EBV, mumps, *Staph*, gonorrhea



Signs & symptoms
-Lid pain
-Excess tearing or discharge
-Swelling of preauricular nodes
-Acute dacryocystitis: swelling of upper lid, erythema, warmth

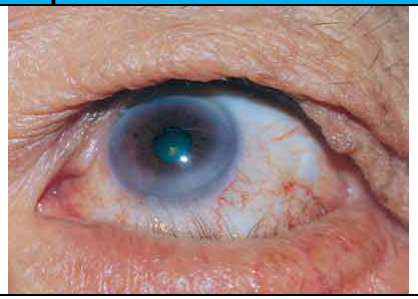
Management
-Simple nasolacrimal duct obstruction → lacrimal duct massage, warm compresses, referral to ophtho for probing if not improving
-Acute dacryocystitis → emergent referral to ophtho and clindamycin or vancomycin due to risk of MRSA
-Think malignancy if no improvement

Entropion

-Folding of the eyelid in ward

Causes
-Genetic
-Congenital
-Infection: *Chlamydia*
-Aging
-Scarring

Signs & symptoms
-Eye redness and pain
-Photophobia
-Sensitivity to wind
-Decreased visual acuity

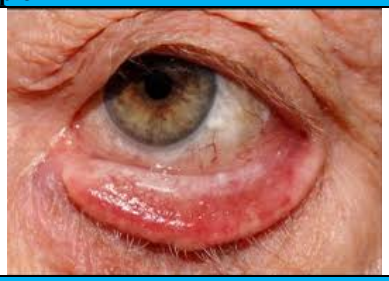


Management
-Surgical repair before permanent corneal damage is done

Ectropion

Causes
-Weakening of tissue or aging
-Allergic
-Facial nerve palsy
-Chemo treatments
-Congenital

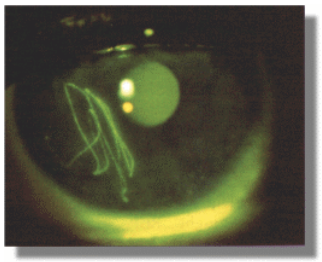
Signs & symptoms
-Sagging lid
-Dull light reflex
-Eye irritation



Management
-Surgical repair

Corneal Abrasion

Corneal ulcer = infected corneal abrasion



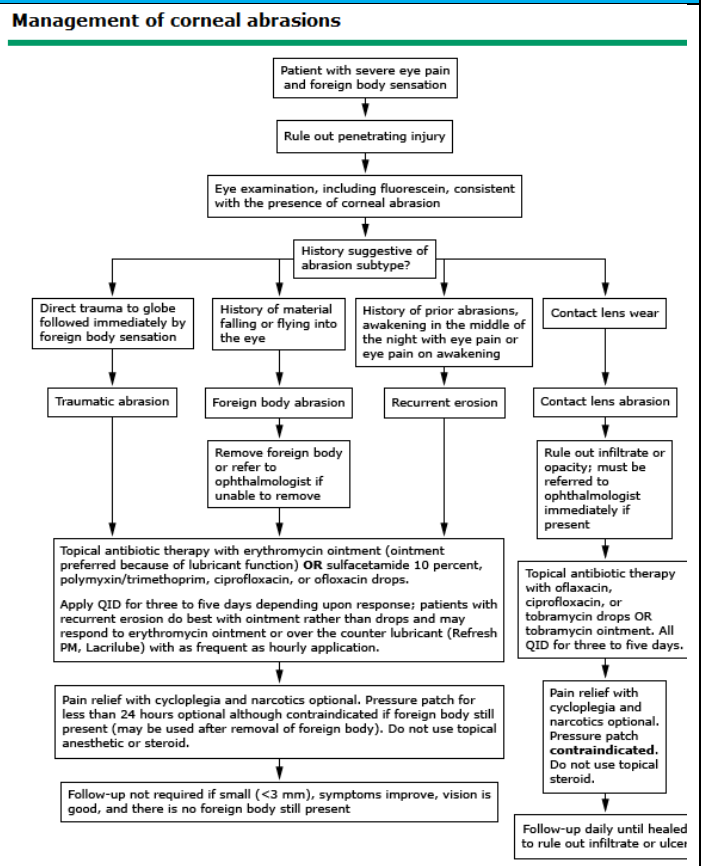
Etiologies
-Traumatic
-Foreign body
-Contact lens
-Spontaneous defect in corneal epithelium

Signs & symptoms
-Severe eye pain
-Foreign body or gritty sensation
-Photophobia
-Excessive tearing
-Blurred vision
-Headache
-Blepharospasm
-Hazy cornea
-Conjunctival injection
-↓ visual acuity

Differential
-Corneal FB
-HSV keratitis

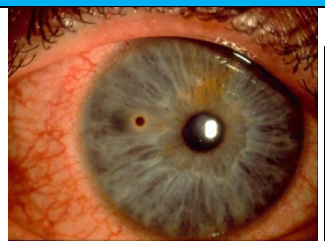
Management
-If abrasion is suspected but not visible, need to do fluorescein stain under UV light
-Rule out FB: flip eyelids-No contact lenses
-No steroids
-Pain control: cycloplegic, opioids, topical anesthetics
-Don't leave pressure patch on > 24 hours
=Any corneal infiltrate, white spot, or opacity needs ophtho referral
-F/u not necessary for most small abrasions as long as symptoms improve and vision remains good (except contact lens wearers)

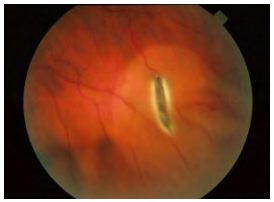
Prognosis
-Most abrasions heal regardless of therapy in 24-72 hours, with return of vision to normal



Foreign Body

Signs & symptoms
-Surface FB: pain and irritation with eye movement
-Intraocular FB: discomfort, blurred vision, h/o striking metal or explosion





Workup
-Visual acuity testing
-Flip eyelids
-Slit lamp exam with fluorescein
-CT or plain film if no findings on slit lamp

Management
-Ophtho consult for intraocular FBs
-Anesthetic eyedrops
-Fine gauge needle to remove FB
-Metal objects can cause rust rings around them, but these usually resorb so don't try to remove or you could cause damage
-Erythromycin ointment and patching following removal
-F/u with ophtho in 48 hours

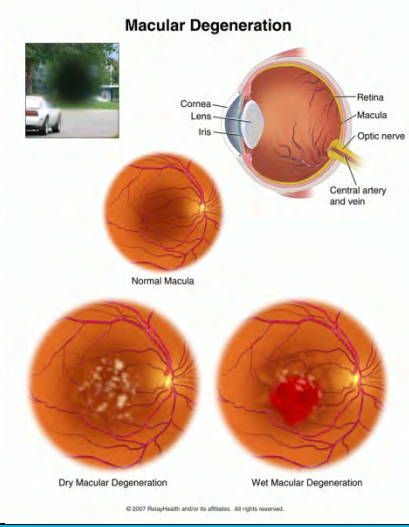
Macular Degeneration

-Damage to the retina causes loss of central vision

Forms
-Non-exudative (dry): when drusen accumulates between the retina and the choroid → loss of cones; accounts for 90% of cases
-Exudative (wet): when blood vessels grow up from the choroid behind the retina

Risk factors
-Age
-Genetics
-Smoking
-HTN
-Micronutrient levels

Management
-Smoking cessation
-Vitamins and supplementation with zinc, beta carotene, copper
-Lasering or surgical extraction for neovascularization or macular translocation
-Antiangiogenesis therapy



Nystagmus

-Regular rhythmic oscillation of the eyes

Jerk nystagmus
-Alternating phases of a slow drift in one direction with corrective quick jerks in the opposite directly
-Caused by imbalance in semicircular canals due to peripheral vestibular disease or disruption of central vestibular pathways in the brainstem

Pendular nystagmus
-Slow, sinusoidal oscillations to and fro
-May be acquired or congenital

Signs & symptoms
-Vertigo
-Oscillopsia
-Abnormal head position
-Blurred vision

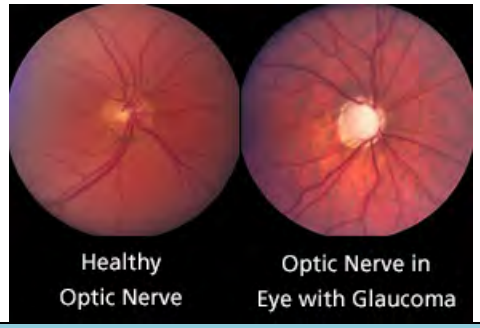
Differential
-Meds: Li, anticonvulsants
-EtOH intox
-Demyelinating disease
-Vertebrobasilar insufficiency
-Brainstem stroke
-Head trauma
-Positional vertigo
-Thiamine or Mg deficiency
-Neoplasm
-Arnold-Chiari malformation
-Encephalitis
-Extreme lateral gaze
-Normal opticokinetic nystagmus

Management
-Treatment is symptomatic
-Treat underlying cause
-Baclofen for periodic alternating nystagmus
-Gabapentin for pendular nystagmus
-Botox injections
-Special prism contact lenses or glasses
-Surgical correction of muscle attachments

Glaucoma

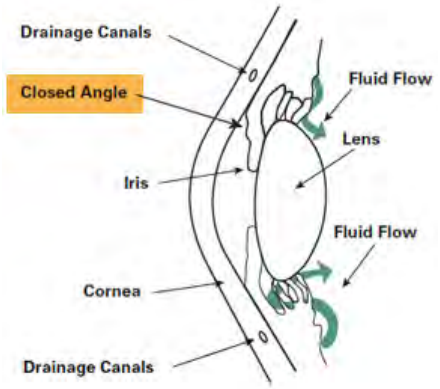
Preventative Eye Exams

Age	Black Patients	White Patients
20-40	Every 2-4 years	No guidelines
40-54	Every 1-3 years	Every 2-4 years
55-64	Every 1-2 years	Every 1-3 years
65+	Every 1-2 years	Every 1-2 years



Closed Angle Glaucoma (Narrow Angle Glaucoma or Acute Angle Glaucoma)

-Fluid builds up behind the lens due to malformed iris and trabecular network contacting each other → sudden blocking of drainage canal
-Most common in Asians and older folks

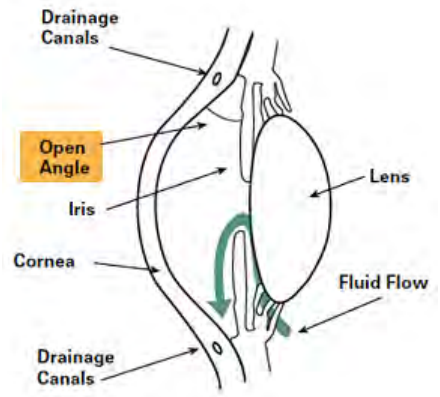


Signs & symptoms
-Occurs after being in a dark place or after anticholinergic use
-Prior episodes of blurred vision
-Halos around lights
-H/o recent eye surgery or uveitis
-Nausea/vomiting
-Eye redness
-Nonreactive pupil
-Hazy cornea
-Shallow depth of anterior chamber
-Optic disc cupping

Management
-Give acetazolamide and timolol, apraclonidine, and pilocarpine
-Emergent referral to ophtho, monitoring of IOP q hour until seen

Open Angle Glaucoma (Wide Angle Glaucoma)

-Fluid builds up in front of the lens as a result of slowly clogging drain → sequential damage to optic nerve with progressive loss of visual field
-Most common type of glaucoma
-Most prevalent in blacks over 40 and others over 65 → screen with visual field confrontation



Signs & symptoms
-Bilateral eyes affected
-Colored halos around lights
-Progressive peripheral vision loss
-May be asymptomatic until severe visual field loss
-Pupil dilation

Management
-Regular aerobic exercise to reduce IOP
-Increase outflow with intraocular prostaglandin analogues
-Suppress production with intraocular β-blockers

Optic Neuritis

-Acute inflammatory demyelinating injury to the optic nerve

Signs & Symptoms
-Painful monocular visual loss over several hours to days
-May have papillitis on funduscopic exam
-Chronic: afferent pupillary defect, color desaturation, optic atrophy

Differential
-Ischemia
-Infection: meningitis, syphilis, Lyme
-Inflammatory: sarcoid, neoplasm, SLE
-Genetic
-Neoplastic
-Compression: pseudotumor cerebri
-Toxic/metabolic: drugs, nutritional deficiency, radiation
-Trauma

Workup
-Gadolinium-enhanced MRI of brain and orbits

Clinical features of more common optic neuropathies*

	Optic neuritis	Non-arteritic ischemic optic neuropathy	Arteritic ischemic optic neuropathy	Leber's hereditary optic neuropathy	Neuroretinitis
Age	20 to 50 years	>50 years	>70 years	25 to 40 years	Children
Gender	2:1 female	Equal	3.5:1 female	80 to 90 percent male	Equal
Pain	Present in >90 percent	Present in <10 percent	Headache, scalp tenderness, jaw claudication	Not present	Variable
Onset	Hours to days	Sudden	Sudden	Weeks to months	Hours to days
Unilateral or bilateral	Usually unilateral	Usually unilateral; low chance may recur in other eye years later	May occur in both eyes in rapid sequence	Bilateral - but presentation often unilateral	Often bilateral
Funduscopic examination	Papillitis present in one-third	Papillitis present in most	Pale swelling of disc; fundus may also be normal (posterior ischemic optic neuropathy - indicates giant cell arteritis)	Disc hyperemia but no swelling; peripapillary telangiectasia	Papillitis, macular edema, exudates
Visual field defect	Central scotoma	Altitudinal (usually inferior) defect	Altitudinal or generalized constriction	Central or cecocentral defect	Variable
Magnetic resonance imaging: optic nerve	Inflammation of optic nerve in most (one-third to one-half will have other demyelinating lesions)	Often normal	May show enhancement	Normal	Variably abnormal
Prognosis	Begins within two to four weeks, most achieve 20/40 or better	Over several months, only 40 percent improve by three or more lines	Poor once vision loss has occurred; may cause rapid blindness untreated	One-third achieve some improvement	Most recover fully

Management
-IV methylprednisone if severe vision loss of 2+ white matter lesions on MRI (NOT oral as it has no benefit)
-Interferon treatments if MRI shows white lesions (may delay development of MS)

Prognosis
-Visual recovery within a few weeks
-30% will go on to develop MS within 5 years

Pterygium

-Triangular wedge of fibrovascular conjunctival tissue extending into the corneal surface

Causes
-UV light exposure
-Abnormal angiogenesis
-HPV

Signs & Symptoms
-Redness and irritation
-Growth over months to years
-Vision impairment
-Often bilateral

Differential
-Conjunctival neoplasia
-Pinguecula (no corneal involvement)
-Conjunctivities

Management
-Artificial tears to reduce irritation
-Surgical removal if visual impairment, restricted eye movement, significant cosmetic impact, or intractable irritation

Orbital Cellulitis

-Usually occurs in kids as a result of untreated sinusitis or trauma

Agents
-*Strep pneumo*
-*H. flu*

Signs & symptoms
-Eyeball proptosis or chemosis
-Edema, erythema, hyperemia, periorbital pain
-Limited eye movements
-Reduced vision

Workup
-CT or MRI

Management
-Admit for IV antibiotics (amoxicillin or ceftriaxone + vanco for severe cases)

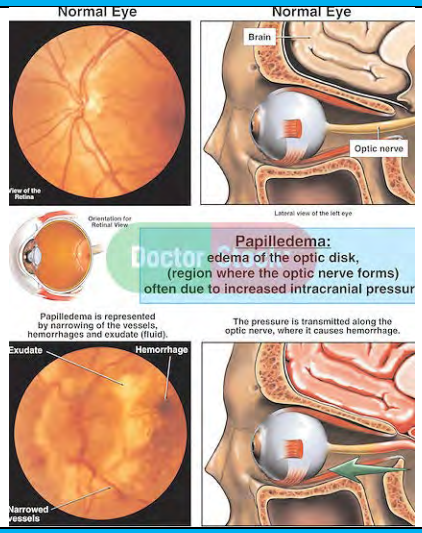
Papilledema

-Optic disc swelling due to ↑ ICP

Signs & symptoms
-Will usually be bilateral
-Headache
-N/v
-Diplopia
-Pulsatile machinery-like sound in ear
-Intermittent/brief visual symptoms

Differential
-Intracranial mass lesion
-Cerebral edema
-↑ CSF production or ↓ absorption
-Obstructive hydrocephalus
-Obstruction of venous outflow
-Pseudotumor cerebri

Workup
-Funduscopic exam and visual acuity testing
-Brain MRI
-LP



Retinal Detachment

-Occurs when retina peels away from underlying support tissue

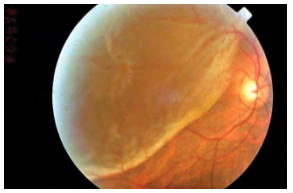
Causes
-Increased risk with myopia, trauma, FH, cataract surgery, diabetic retinopathy

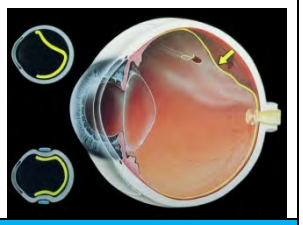
Signs & Symptoms
-Sudden painless loss of vision "like a curtain covering the eye"
-May see flashes or floaters or cuts or lines in vision
-Defect in confrontational visual fields

Workup
-Emergent ophtho consult if suspecting

Management
-Immediate surgical repair

Prognosis
-Will lead to complete vision loss without surgical repair



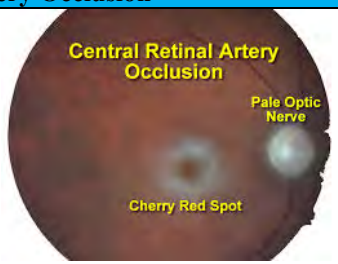


Central Retinal Artery Occlusion

-Loss of blood supply to the retina via embolus
-Risk factors: a-fib, endocarditis, coagulopathies, CAD, hypercoagulable states, temporal arteritis

Signs & symptoms
-Painless, severe loss of vision in one eye
-Funduscopic exam shows cherry red spot, pale or swollen optic nerve with splinter hemorrhages, ground-glass retina

Workup
-CV exam for bruits and temporal arteritis
-Carotid imaging
-EKG



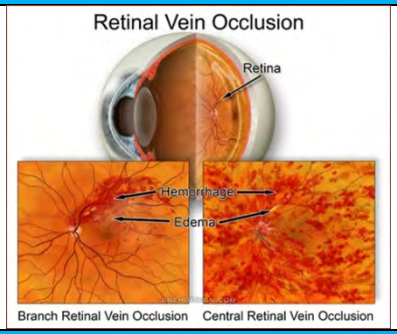
Management
-Emergent ophtho consult with interventions to lower IOP
-Antiplatelets

Prognosis
-Poor, no treatments proven to improve visual outcomes

Central Retinal Vein Occlusion

-When occluded retinal vein backs up and fills the retina with blood
-Risk factors: HTN, mechanical compression, glaucoma, inflammation of the optic nerve, orbital disease, hyperviscosity disorders

Signs & symptoms
-Painless loss of vision in one eye



Management
-Treat underlying medical disorders
-Aspirin therapy
-Lasering of ischemic retina
-Treat associated glaucoma and macular edema

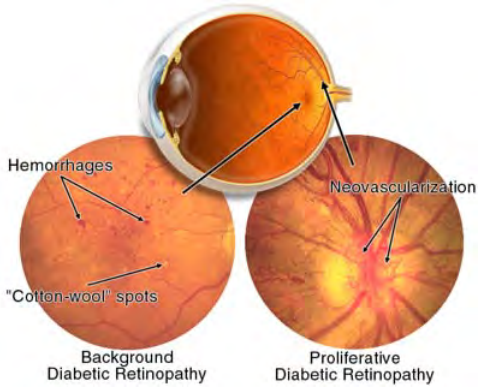
Diabetic Retinopathy

-Occurs when hyperglycemia damages the basement membrane of retinal capillaries → loss of pericytes and microaneurysm formation → leakage of capillaries and macular edema with proliferation of weak blood vessels
-Associated with DM1 and DM2 but not gestational diabetes

Screening
-Yearly eye exams for diabetics
-Maintain A1c < 7%
-HTN control

Signs & symptoms
-Most are asymptomatic

Management
-Nonproliferative → panretinal photocoagulation if severe
-Proliferative → panretinal photocoagulation if high risk or severe, early vitrectomy if severe and DM1, intravitreal VEGF inhibitors as therapy adjunct



Strabismus (Tropia)

Etiologies
-Congenital: pseudostrabismus, prenatal drug exposure, nerve palsy, familial external ophthalmoplegia
-Acquired: accommodative strabismus, intermittent exotropia, cataracts, tumors, increased ICP, orbital injury, head trauma, vascular disorders, botulism, myasthenia gravis, nerve palsy, Guillain-Barre, ocular myopathy, multiple sclerosis, infection, drug or toxin, DM, hypoglycemia, thyrotoxicosis


Pseudostrabismus. Although the eyes appear misaligned in this photograph, the light reflection is symmetrical in both eyes.

Differential
-Pseudostrabismus
-Ocular instability of infancy (normal in first few months of life)

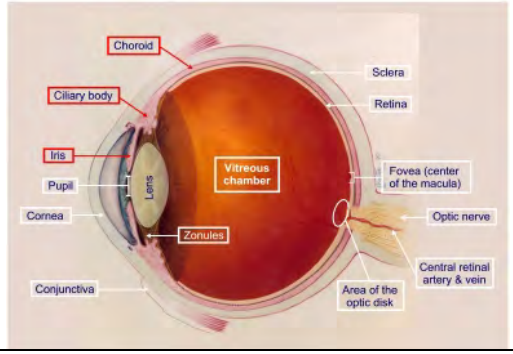
Workup
-Affected eye will drift when covered, then moves quickly back if cover is removed
-Differentiate congenital from acquired (may be vision-threatening or life-threatening)

Management
-Refer to ophtho for consistent strabismus at any age, persistent strabismus after 4 months of age, altered light reflex, deviation with cover test, deviation that changes depending on position of gaze, torticollis, parental concern

Sequelae
-If untreated may lead to amblyopia (vision reduction) or diplopia

Uveitis (Iritis)

-Inflammation and leukocyte infiltration of the iris, ciliary body, and/or choroid and vitreous humor of the eye



Causes
-Infections: CMV, toxoplasma, syphilis, HSV, cat scratch disease
-Systemic immune-mediated disease: spondyloarthropathies, IBD, sarcoidosis, MS
-Syndromes confined to the eye
-Masquerade syndromes: CNS lymphoma

Signs & symptoms
-Depends on area of eye affected
-Anterior chamber → pain and redness
-Posterior chamber → painless floaters or vision loss

Workup
-Slit lamp and funduscopic exam

Management
-Ophtha consult
-Treat any infectious cause
-May need steroids

EAR DISORDERS

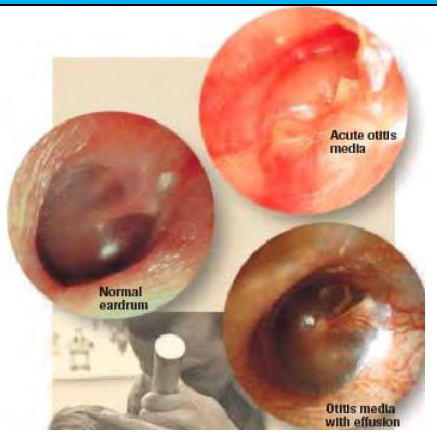
Otitis Media

Acute OM
-Agents: *Strep, H. flu, M. cat,* or viral (can't distinguish)
-Signs & symptoms: hearing loss is hallmark, ear pain, ear fullness, drainage with relief if ear drum is perforated, prior URI, pulling at ears, fever, irritability

Chronic suppurative OM = frequent AOM with otorrhea as a result of TM perforation or tube placement

OM with effusion = fluid behind TM without presence of infection, a result of chronic eustachian tube dysfunction, previous AOM, or barotrauma

Management
-If mild, can watchfully wait with NSAIDs for pain relief as long as patient is > 2 years
-If infection is obvious or there is a fever, treat with 10-14 d of high dose amoxicillin, erythromycin, Augmentin, Septra, ceftriaxone
-Refer for surgical management if there is bilateral effusion > 3 months and bilateral hearing deficiency
-Chronic → tx with 10 d of FQ, consider chronic therapy with daily amoxicillin during winter and spring with monthly f/u



Otitis Externa

Etiology
-Bacterial 90% of the time: *Pseudomonas, Strep, Staph*
-Fungal: *Aspergillus, Actinomyces, Candida*
-Eczema if chronic
-Malignant otitis externa = osteomyelitis of temporal bone as a result of chronic infection in DM, not cancerous!

Signs & symptoms
-Pain with manipulation of tragus
-Hearing loss
-Otorrhea
-Fullness
-Itching
-Recent exposure to water

Management
-Bacterial → neo/poly/HC only if TM intact, FQ (use a wick if canal is swollen), systemic therapy if canal is swollen shut or pt is immunocompromised
-Fungal → acetic acid/HC drops, clotrimazole drops
-Bacterial vs fungal? → CASH powder covers both
-Chronic → treat eczema with steroid cream, then use vinegar/water washes and avoid Q-tips
-Malignant → emergent referral to ENT

Ear Barotrauma

Causes
-Water diving
-Ascent into the atmosphere
-Mechanical ventilation
-URI

Signs & symptoms
-External ear barotrauma: ear pain, bloody otorrhea, petechiae, hemorrhagic blebs, or rupture of TM
-Middle ear barotrauma: pain and fullness on descent in airplane with failure to pop ears, or may have URI symptoms

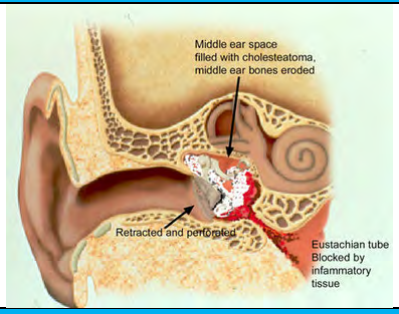
Management
-Keep ear dry
-Pain control
-Decongestants or antihistamines
-Prevent recurrence with decongestants prior to airplane travel

Cholesteatoma

-Keratinized, desquamated epithelial collection in the middle ear or mastoid

Causes
-Primary lesion
-Secondary to TM perforation

Signs & symptoms
-May be asymptomatic
-Hearing loss
-Dizziness
-Otorrhea
-White mass visible behind TM
-TM retraction
-Obstructive polyp



Workup
-CT or MRI if complications present or to better characterize extent

Management
-Surgical removal

Eustachian Tube Dysfunction

-Occurs when blockage of the eustachian tube allows air to exit middle ear but not come back in → creation of negative pressure atmosphere in middle ear

Signs & symptoms
-TM retraction
-Ear pain
-Hearing loss
-Ear fullness

Differential
-Allergies
-URI
-Nasopharyngeal mass
-Abnormal anatomy

Management
Acute: nasal steroid spray, decongestants
-Chronic: tube placement bilaterally

Prognosis
-Risk of cholesteatoma if untreated

Ear Foreign Body

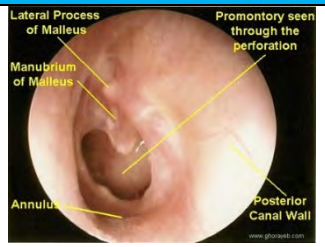
Causes
-Embedded earring
-External ear FB: beads, pebbles, tissue paper, insects

Management
-Local anesthesia with removal for embedded jewelry
-Office removal of small inorganic objects or insects with irrigation (95% ethanol, mineral oil, or 1% lidocaine), followed by forceps or superglue on q-tip for further extraction
-Systemic antibiotics if perichondritis or chondritis present
-ENT referral for button batteries, vegetable matter (will swell with irrigation), penetrating FB, or associated injury to TM

Tympanic Membrane Perforation

Causes
-Penetrating trauma
-Noise trauma

Signs & Symptoms
-Acute onset pain and hearing loss ± bloody otorrhea
-May have tinnitus or vertigo



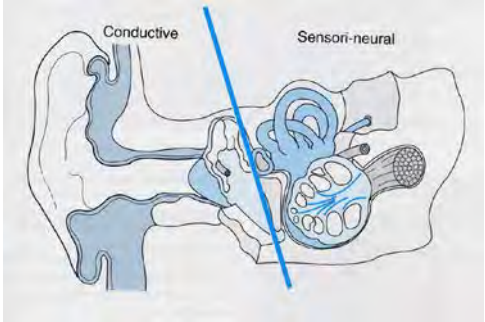
Workup
-ENT consult for serious hearing loss, otorrhea, facial nerve paralysis, or nausea/vomiting, nystagmus, or ataxia

Management
-Most cases will heal on their own
-Need f/u hearing testing
-Avoid water in ear

Hearing Loss

-Conductive, sensorineural, or mixed

Etiologies
-Conductive, outer ear: congenital, external otitis, trauma, squamous cell carcinoma, exostosis, osteoma, psoriasis, cerumen
-Conductive, middle ear: congenital, otitis media, cholesteatoma, otosclerosis, tympanic membrane perforation, temporal bone trauma, glomus tumor
-Sensorineural: hereditary, congenital, presbycusis, meningitis, thyrotoxicosis, viral cochleitis, ototoxic drugs, otologic surgery, Meniere disease, noise, barotrauma, penetrating trauma, acoustic neuroma, meningioma, autoimmune disease, multiple sclerosis, cerebrovascular ischemia

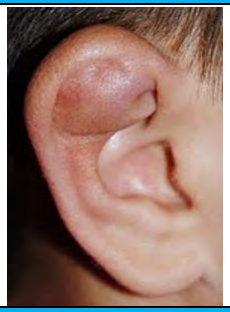


Workup
-Office hearing evaluation with Rinne and Weber tests
-Formal audiologic testing if there is no obvious etiology
-Contrasted MRI or CT if there is progressive asymmetric sensorineural hearing loss
-Labs for unexplained sensorineural hearing loss: glucose, CBC, TSH, RPR
-Urgent referral to ENT for sudden hearing loss

Management
-Standard air conduction hearing aid
-Bone conduction device for congenital atresia, chronic ear infections, or single-sided deafness
-Cochlear implant

Hematoma of External Ear

-Blood collection after direct trauma to the ear



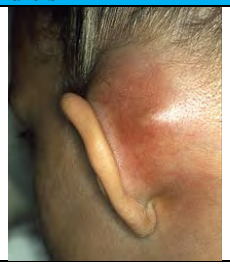
Management
-Regional auricular block followed by I&D or indwelling catheter placement for large hematomas or needle aspiration if small
-Refer to ENT for draining if > 7 days old
-Give 7-10 days of abx to cover *Pseudomonas* given tenuous blood supply of cartilage
-F/u in 3-5 days to eval for infection or reaccumulation
-At least 1 week off from contact sports

Prognosis
-Will lead to necrosis, infection, and permanent cauliflower ear deformity if not drained

Mastoiditis

-Complication of acute or chronic OM
-Usually occurs in kids
-Can be serious due to proximity to the posterior cranial fossa, lateral sinuses, facial nerve canal, semicircular canals, and tip of temporal bone

Signs & symptoms
-Fever
-Posterior ear pain
-Local erythema over the mastoid
-Edema of the pinna
-Posteriorly and downward displaced auricle



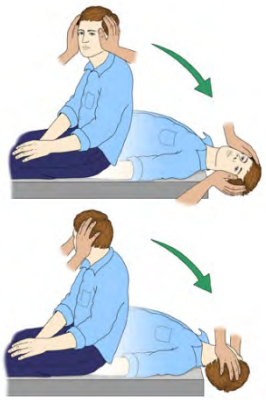
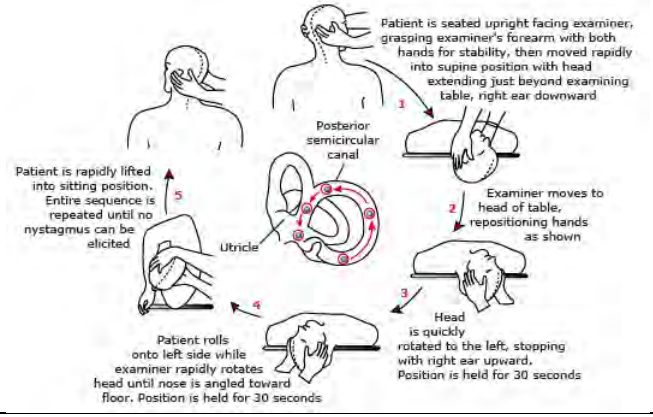
Workup
-CT scan

Management
-Hospital admission with IV abx
-May need mastoidectomy

Vertigo

Differential

-CV: orthostatic HTN, arrhythmia, CAD
-Neuro: acoustic neuroma, TIA, stroke, Parkinson's, neuropathy, migraine
-Anemia
-Psych: panic, anxiety
-Metabolic: hyperthyroid, menopause
-Orthopedic: cervical disc disease, lower extremity arthritis
-Geriatric: decreased proprioception, off center of balance, polypharmacy

Cause	Information	Signs & Symptoms	Management
Benign Paroxysmal Positional Vertigo	-Otolith dislodgement into semicircular canals	-Intermittent vertigo lasting < 1 minute -NO hearing loss -Better with head held still, worse with R/L movements when lying down -Vertical or horizontal nystagmus + Dix-Hallpike maneuver: affected ear will cause nystagmus when it is downward 	
Meniere's Disease	-A result of increased endolymphatic fluid	-Sudden unilateral SNHL, roaring tinnitus, and vertigo for hours -Ear fullness	-Diuretics -Low salt diet -Anti-vertigo meds
Acute Labyrinthitis & Vestibular Neuritis	-Infection or inflammation of the inner ear, usually due to latent virus -Neuritis = only semicircular canals affected -Labyrinthitis = vertigo + hearing loss	-Severe, disabling vertigo for 24-48 hours followed by weeks of imbalance -Vomiting -Pts think they are dying	-Steroids -PT
Central Vertigo	-Issue with balance centers of the brain	-Symptoms with gradual onset but are constant -May have nausea or diaphoresis -Vertical nystagmus -Usually no hearing loss	

Tinnitus

-Abnormal perception of sound in the middle ear in the absence of a corresponding sound in the external environment

Subjective tinnitus

-A sound only the patient can hear
-Caused by aberrant neurological signalling in the brain
-May be a neurologic response to hearing loss; high freq. loss will cause high freq tinnitus, low freq. loss will cause low freq. tinnitus
-Can also be caused be aspirin

Objective tinnitus

-When a clinician can perceive an abnormal sound emanating from the pt's ear
-May have clicking if due to pharyngeal muscle spasm
-May have breathy sounds if due to abnormally open eustachian tube
-May be pulsatile or bruit-like with referred vascular sounds or tumor

Differential

-Excessive noise exposure
-Meniere's disease
-Labyrinthitis
-Otitis media
-Eustachian tube dysfunction
-Ototoxicity: ASA, aminoglycosides, loop diuretics, cisplatin
-Glomus tumor
-Any cause of sensorineural hearing loss
-Intracranial AV malformation

Workup

-Audiometry
-Refer for imaging for pulsatile tinnitus

Management

-Cochlear implants if due to sensorineural hearing loss
-Stop offending meds
-Avoid caffeine and nicotine
-Use background noise
-Tinnitus retraining therapy
-Resolve underlying problem if due to conductive hearing loss

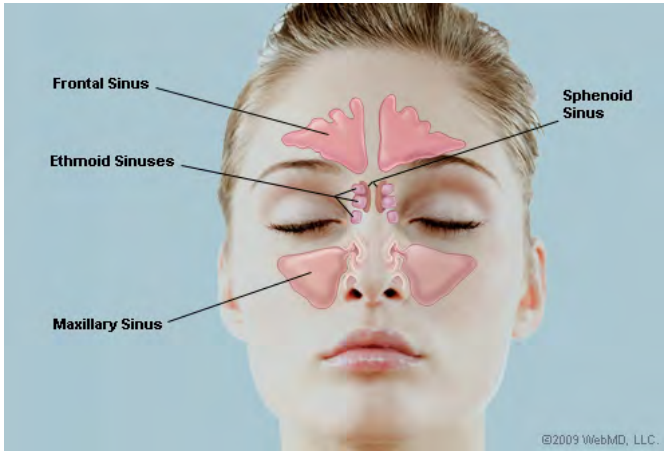
NOSE & SINUS DISORDERS

Sinusitis

-Acute = symptoms < 4 weeks
-Subacute = symptoms for 4-12 weeks
-Chronic = > 3 months

Agents
-Usually viral
-Bacterial is secondary to prior URI: Strep pneumo, M. cat, H. flu, Staph aureus
-Fungal possible if immunocompromised

Signs & Symptoms
-Headache, localized sinus pain and pressure (esp unilateral), foul or purulent nasal discharge, fever, upper tooth pain, cough, fatigue
-No correlation between report of "sinus headache" and sinusitis found on CT scan
-Double sickening more likely to be bacterial
-Peds: unusual as their sinuses are not fully formed, but may have bad breath, subacute or abrupt onset



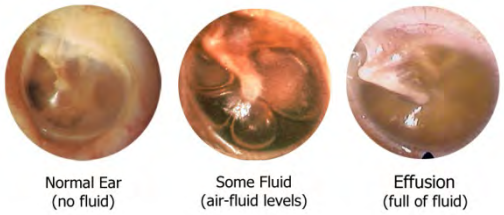
Management
-Mild cases can be observed for 7 days as 80% of all cases improve without any AB within 2 weeks
-Treat with AB if mod-severe symptoms have failed to improve after 10-14 days or there is double sickening: amoxicillin or Septra or doxycycline first, Augmentin or levofloxacin if severe, recent AB use, or failed first-line therapy
-Nasal saline rinses
-Nasal steroid spray
-Antihistamine
-Decongestant
-Mucolytics
-Afrin short-term
-If chronic, consider allergenic causes, CF, anatomic abnormalities, unusual organisms, may need to refer
-Send to ED if there is facial cellulitis, proptosis, vision change, mental status changes

Complications: orbital cellulitis or abscess, osteomyelitis (Pott's puffy tumor), intracranial extension, cavernous sinus thrombosis

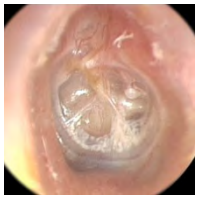
Allergic Rhinitis

-Typically does not occur in infants under 6 mos
-Seasonal or perennial

Samter's triad = syndrome of aspirin sensitivity, nasal polyposis, and asthma often seen with allergic rhinitis, frequently leading to severe pansinusitis



Signs & symptoms
-Repetitive sneezing
-Pruritus of nose, eyes, palate, ears
-Clear rhinorrhea
-Nasal congestion
-Postnasal drip
-Epistaxis
-Allergic shiners or Dennie's lines
-Allergic salute
-Retracted TMs
-Serous effusions
-Swollen or boggy turbinates
-Hyperplasia of palate or posterior pharynx



Differential
-Sinusitis
-Rhinitis medicamentosa (Afrin!)
-Polyps
-Deviated septum
-Adenoid hypertrophy
-FB
-Vasomotor rhinitis

Management
-Instruct patients in allergen avoidance: closed windows, bed cases, washing linens weekly, removing stuffed animals, cockroach poison, mold precautions, HEPA filters
-Nasal saline sprays or rinses
-Oral decongestants
-Nasal steroids: fluticasone, flunisolide
-1st or 2nd gen antihistamines: cetirizine and fexofenadine ok for infants > 6 mo
-Leukotriene inhibitor
-Refer to allergist for kids with mod-severe disease, prolonged rhinitis despite intervention, coexisting asthma or nasal polyps, recurrent otitis media or sinusitis

Epistaxis

-Almost always anterior (Kiesselbach plexus)

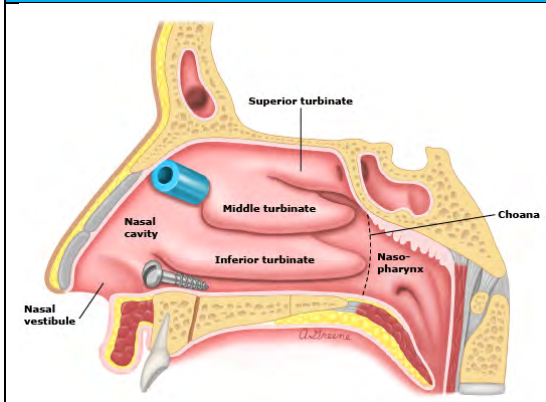
Causes
-Trauma
-Infection
-Allergic rhinitis
-Atrophic rhinitis
-Coagulopathy
-Tumors
-Arteriosclerosis in the elderly

Signs & symptoms
-Anterior bleed is usually unilateral with no sensation of blood down the back of the throat
-Posterior bleeding is profuse with strong sensation of draining down posterior pharynx

Workup
-CBC and coags for profuse bleeds

Management
-Anterior: compress nose for 10-15 min, consider vasoconstrictive agents, cautery with silver nitrate or electrically, nasal packing last resort
-Posterior: nasal packing (high morbidity risk), embolization, ligation

Nasal Foreign Body



Signs & symptoms
-Unilateral purulent and foul-smelling nasal drainage
-Black drainage or epistaxis if button battery present

Workup
-Plain radiographs if unable to visualize FB

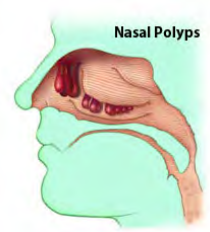
Management
-Urgent ENT referral for button batteries, magnets attached across the nasal septum, penetrating FB, impacted FB, or posterior FB
-Nose blowing or mouth-to-mouth blowing using the parent for occlusive FBs
-Instrumentation for non-occlusive FBs

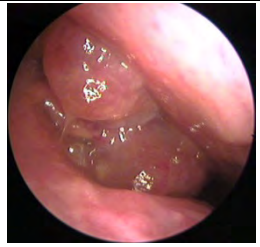
Nasal Polyps

Etiologies
-Usually a reaction to bacterial infection in kids
-Allergies
-Chronic sinusitis

Signs & symptoms
-Stuffiness
-Feelings of pressure or fullness in the face
-Trouble smelling

Management
-Steroid nasal spray
-Saline rinses
-Refer for surgical excision but may recur





MOUTH & THROAT DISORDERS

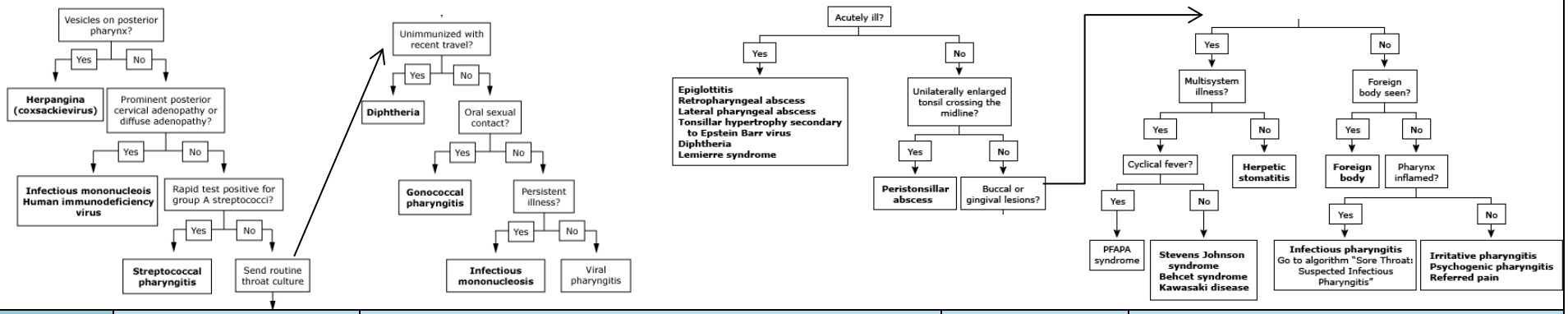
Laryngitis

-Etiology is typically viral

Hoarseness differential
-Acute: postnasal drip, viral laryngitis, hypothyroidism, vocal fold paralysis, recent intubation, vocal hemorrhage
-Chronic: smoking, vocal strain, GERD, cancer, vocal nodules (vocal misuse) or polyps (GERD)

Treatment
-Rest, fluids, smoking cessation
-Never use antihistamines or steroids because they can cover up the injury → permanent damage
-Refer to ENT for laryngoscopy for hoarseness > 2 weeks

Acute Pharyngitis



Differential	Agents	Signs & symptoms	Workup	Treatment
Viral Pharyngitis	-Adenovirus, coronavirus, rhinovirus, influenza, parainfluenza, Coxsackie	-Concurrent rhinorrhea -Erythema, edema, dysphagia, pain, fever, lymphadenopathy, diffusely pink throat, cough, fever		-Salt water gargles -Lozenges or hard candy -Acetaminophen or ibuprofen -Oral rinse with equal parts lidocaine, diphenhydramine, and Maalox -Benzydamine HCl mouth rinse
***Strep* Pharyngitis**	-GAS	-Uncommon in kids under 2-3 -Sore throat, dysphagia, odynophagia, erythema, airway obstruction, bright beefy red demarcated splotches -Centor criteria: tender cervical adenopathy, fever > 100.4, no cough, tonsillar exudate -Abdominal pain and vomiting in peds	-Distinguish from viral by rapid *Strep* test ± culture	-Penicillin VK -Cephalosporin -Erythromycin: increasing macrolide resistance
Acute Tonsillitis	-Viral: Mono -Bacterial: GAS	-Swollen tonsils with white plaques	-Rapid *Strep* -Monospot	-Antibiotics
Peritonsillar Abscess		-May follow tonsillitis -Bulging, asymmetrical soft palate, hot potato voice, severe throat pain, dysphagia, trismus, deviated uvula, salivation, fever, severe malaise		-Urgent referral to ENT for I&D
Mononucleosis	-EBV or CMV	-Fatigue, malaise, sore throat with tonsillar edema, erythema, and shaggy white-purple tonsillar exudate, lymphadenopathy, hepatosplenomegaly -Many will have 2° *Strep* tonsillitis	-Monospot (not + early in disease) -CBC to look for atypical lymphocytes	-OTC pain control -? steroids -Splenic precautions -Treat tonsillitis but avoid ampicillin due to rxn with mono → rash
***Fusobacterium* Pharyngitis**	-*Fusobacterium necrophorum*	-Adolescents -Severe pharyngitis -Cervical adenopathy -Headache -May have fever -Unilateral neck pain or swollen neck	-Very high CRP -↑ WBCs with leukocytosis	-Treat to avoid Lemierre's syndrome (septic emboli thrown from internal jugular) with penicillin + clindamycin

Aphthous Ulcers (Aphthous Stomatitis or Canker Sore)

-Intermittent formation of non-contagious ulcers on the mucous membrane of the oral cavity in otherwise healthy individuals
-Cause not completely understood but may involve a T-cell-mediated response after initiation of a trigger

Common triggers
-Nutritional deficiency
-Local trauma
-Stress
-Hormonal influences
-Allergies
-Genetic predisposition

Signs & symptoms
-Minor aphthous ulceration = lesions < 10 mm
-Major aphthous ulceration = lesions > 10 mm, may have scarring
-Herpetiform ulceration: crops of vesicles that resembles HSV but is not caused by this!

Differential
-HSV
-Erythema multiforme
-Drug reaction
-Pemphigus or bullous pemphigoid
-SLE
-Lichen planus
-Herpangina or hand-foot-mouth
-Acute HIV
-Parvovirus
-Varicella zoster
-Syphilis
-Oral candidiasis
-Behcet's síndrome
-Reactive athritis
-IBD
-SCC
-Necrotizing ulcerative gingivostomatitis

Management
-Topical barriers
-Topical analgesics
-Topical hydrocortisone + antibiotic ointment

Prognosis
-Large aphthae may take 20-30 days to heal

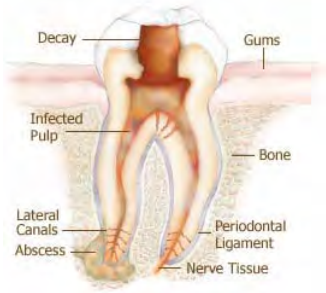
Dental Caries

Prevention
-Early referral to dentist for kids with breast or bottle feeding > 12 months, frequent consumption of sugary beverages and snacks, prolonged use of sippy cups, use of bedtime bottles, use of liquid meds > 3 weeks, insufficient fluoride exposure, visible plaque on upper front teeth, enamel pits or defects, exposure to second-hand smoke
-AAP recommends referral to dentist at age 1, Medicaid begins at age 3
-Screen for plaque, white spots, and cavities as soon as first teeth erupt
-Instruct parents to clean infant's gums with soft cloth starting at birth, and to begin brushing teeth when they first appear twice per day
-Fluoride varnish: providers in NC may apply from eruption of first teeth up to age 3
-Stop pacifiers by age 3, thumb sucking by age 6

Signs & symptoms
-Initial presentation is a white spot

Dental Abscess

-Caused by entrapment of plaque and debris in the periodontal pocket



Signs & Symptoms
-Painful, red fluctuant swelling of the gingiva
-Exudate from affected area on probing

Management
-Warm saline rinses and oral penicillin or erythromycin for small abscesses
-I&D for large abscesses

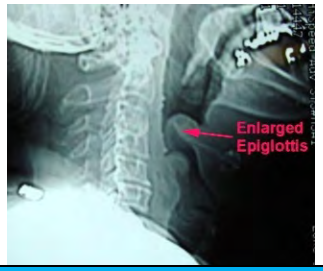
Acute Epiglottitis

Agents
-*H. flu*
-*Strep pneumo or Strep pyogenes*
-*Staph aureus*
-Trauma

Signs & symptoms
-Abrupt onset of high fever, sore throat, stridor, dysphagia, drooling, trismus
-Sitting child that won't lie down, head leaning forward (sniffing or tripod position)

Differential
-Croup
-Peritonsillar abscess
-Foreign body
-Diptheria

Workup
-Lateral x-ray for "thumb sign"



Management
-Send to ED for inpatient management and antibiotics as any manipulation of glottis could result in airway obstruction

Sialadenitis & Parotitis

-Inflammation of a salivary gland
-Most commonly affects the parotid or submandibular gland
-Acute, chronic, or recurrent
-Agent is usually *Staph aureus* but can be polymicrobial

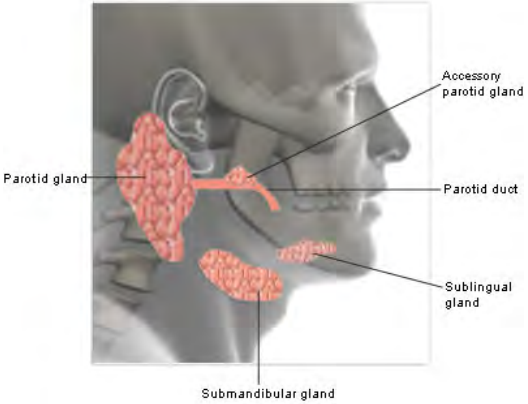
Risk factors
-Decreased salivary flow
-Dehydration
-Poor oral hygiene

Signs & symptoms
-Painful swelling and edema of the cheek, especially with meals
-Reddened skin
-Malaise
-Purulent exudate from duct punctum

Workup
-Culture Stensen's duct drainage if present

Management
-10-14 days antibiotics (may need IV if severe): clindamycin + cipro, or Augmentin
-Warm compresses

Prognosis
-Complications: abscess, Ludwig's angina, cellulitis



EENT NEOPLASMS

Other Ear Neoplasms

Squamous cell carcinoma
-Suspect with nonresolving otitis externa
-High mortality rate

Nose & Sinuses Neoplasms

Inverted papilloma
-Benign tumor caused by HPV
-S/s: obstruction, hemorrhage
-Tx: excise as can be associated with SCC

Juvenile angiofibroma
-Benign vascular tumor
-S/s: nasal obstruction and hemorrhage
-Tx: surgical excision

Squamous cell carcinoma
-Nasopharynx or sinuses
-S/s: eustachian tube obstruction, serous otitis media

Nasopharyngeal carcinoma
-Associated with EBV

Salivary Gland Neoplasms

-Most arise in parotid
-Most are benign

Laryngeal Neoplasms

Vocal fold nodules
-Benign "Singer's nodules"
-Need speech therapy

Vocal fold polyps or cysts
-Benign
-Related to vocal fold trauma

Laryngeal leukoplakia
-Associated with smoking
-May be associated with SCC → biopsy

Laryngeal squamous cell carcinoma
-The most common malignancy of the larynx
-S/s: new and persistent hoarseness in a smoker, persistent throat or ear pain, neck mass, hemoptysis, stridor
-W/u: CT, laryngoscopy with biopsy, esophagoscopy, bronchoscopy
-Tx: radiation or partial laryngectomy, chemo if late stage

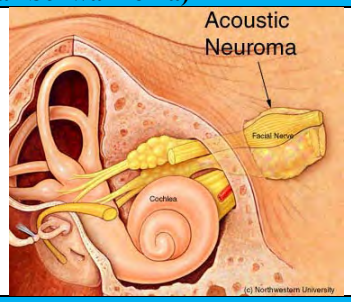
Acoustic Neuroma (Vestibular Schwannoma)

-Account for 85% of all cerebellopontine angle tumors in adults

Risk factors
-Neurofibromatosis type 2
-Exposure to loud noise
-Childhood radiation exposure
-H/o parathyroid adenoma
-Cell phone use

Signs & symptoms
-Unilateral sensorineural hearing loss
-Tinnitus
-Facial nerve symptoms

Workup
-MRI



Management
-Surgical or radiation therapy for large tumors, young age, or significant hearing impairment
-Observation with serial imaging and audiometry for older patients with small tumors and limited hearing loss

Oral Leukoplakia

-An early dysplasia of the squamous epithelium
-May be malignant or inflammatory
-Association with HPV

Risk factors
-Smoking

Signs & symptoms
-White patches or plaques on the oral mucosa

Workup
-Biopsy

Prognosis
-Between 1-20% of lesions will progress to carcinoma within 10 years
-Lesions arising in trauma-prone areas of the oral cavity are less likely to be dysplastic

REPRODUCTIVE SYSTEM

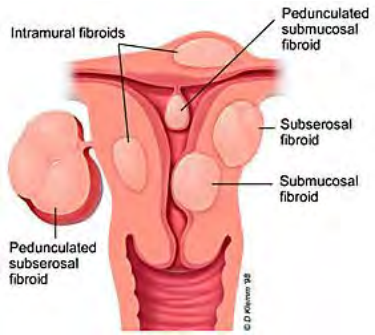
UTERUS

Leiomyomas (Uterine Fibroids)

-Benign tumors arising from the myometrium
-Varying locations

Risk Factors
-Black
-Obese
-Over age 40
-Nulliparity

Protective
-Multigravida
-Postmenopausal
-Smoker
-Prolonged OCP use
-Depo use



Signs & Symptoms
-Dysmenorrhea and AUB
-Menorrhagia and possible subsequent anemia
-Dyspareunia
-Urinary frequency
-Infertility
-Irregular feeling uterus
-Abdominal mass
-Bloating
-Pelvic pain or pressure or feeling of fullness
-Acute pain with torsioned pedunculated fibroid
-Miscarriage with submucosal fibroids intruding on fetus
-Can be asymptomatic
-Symptoms improve after menopause

Workup
-TVUS is diagnostic
-Consider malignancy workout with rapid growth

Management
-Only if symptomatic or pursuing pregnancy
-Surgical myomectomy (should be hysteroscopic if desiring future pregnancy)
-Hysterectomy only for extremely large, painful fibroids with intractable bleeding
-Mirena IUD or Depo injections to reduce bleeding
-Menopause-mimicking agent such as ulipristal
-Uterine artery embolization to starve off fibroids
-Consider shrinking large fibroids with GnRH agonists prior to surgical removal

Prognosis
-Not associated with malignant transformation

Dysfunctional Uterine Bleeding

-Bleeding outside normal parameters of menses (24-35 days, < 80 mL per cycle) found in the absence of demonstrable structural or organic pathology that is unrelated to another underlying illness
-Classified as ovulatory or anovulatory
-Some providers consider DUB to be a subset of abnormal uterine bleeding

Causes
-Usually a hormonal disturbance: menopause, premature ovarian failure, PCOS, prolactinoma, anovulation, immature HP axis (adolescents), perimenopause
-Anovulation causes DUB b/c there is no corpus luteum formation → no progesterone to oppose estrogen-induced hyperplasia of the endometrium

Differential
-Abnormal uterine bleeding (known pathology): miscarriage, gestational trophoblastic disease, IUD, meds, trauma, coagulopathy, adrenal disorder, stress, pituitary adenoma, smoking, infections, fibroids, malignancy, atrophic vaginitis

Workup
-A diagnosis of exclusion
-Pregnancy test
-Malignancy workup if postmenopausal
-Coagulopathy workup: PT/aPTT, CBC,
-Assess ovulatory status: biphasic body temp, progesterone levels, urine LH
-Pelvic exam with pap
-May need endometrial biopsy or hysteroscopy
-Consider US evaluation for fibroids, polyps, or adenomyosis
-Consider testosterone and DHEAS levels in women with signs of virilization

Management
-Treat underlying cause if due to abnormal uterine bleeding
-Adolescent mild DUB can be treated with iron supplements and observation
-Adolescent mod-severe DUB can be treated with OCPs or progestin only regimen
-Patients with contraindication to estrogen therapy can consider symptomatic management with NSAIDs, progestin-only regimen, or Mirena IUD
-Endometrial ablation an option for women not wishing to conceive (although will still need contraception)
-Hysterectomy is the definitive treatment

Endometrial Neoplasms

-Endometrial neoplasia involves proliferation of the endometrial glands that can progress to or coexist with endometrial carcinoma
-Endometrial carcinoma is the most common GYN cancer in the US and is usually adenocarcinoma

Risk Factors
-Age > 50
-Uopposed estrogen use
-PCOS
-DM
-Obesity
-Nulliparity
-Late menopause
-Tamoxifen use
-HNPCC

Signs & Symptoms
-Abnormal uterine bleeding
-Postmenopausal bleeding
-Abnormal pap cytology

Differential for Postmenopausal Bleeding
-Atrophy (59%)
-Endometrial polyps
-Endometrial cancer
-Endometrial hyperplasia
-Hormonal effects
-Cervical cancer

Workup
-Endometrial biopsy can be done in clinic and is 99.6% sensitive in premenopausal women and 91% in postmenopausal women
-Transvaginal US to assess endometrial stripe: thin stripe < 4-5 mm associated with low risk of cancer while stripe > 5 mm warrants biopsy

Management
-Benign pathology on biopsy watched, no action warranted unless bleeding persists
-Endometrial hyperplasia on pathology without atypia is treated with progesterone cream, ovulation induction, or IUD to induce massive menses and endometrial sloughing
-Atypical endometrial hyperplasia needs D&C or hysterectomy + BSO

Endometriosis & Adenomyosis

Endometriosis
-Location of endometrial tissue any place outside of the uterus
-May be caused by retrograde menstruation, where sloughed off endometrial tissue escapes through the fallopian tubes to implant outside of uterus
-Could also be caused by Mullerian cell remnants, direct surgical transplantation, altered immune response, genetics, or increased estrogen stimulation
-Usually occurs in the pelvis, but can occur in the ovary, cul de sac, uterine ligaments, fallopian tubes, bladder, rectum, bowel cervix, vagina, omentum, umbilicus, vulva, ureter, spinal cord, nasopharynx, breast, lung, and kidney

Adenomyosis
-Endometriosis within the uterine muscle

Signs & Symptoms
-Endometriosis typically occurs in young, tall, thin, nulliparous Caucasian women
-Adenomyosis typically occurs in women ages 40-50
-Associated with early menarche and late menopause
-May be asymptomatic
-Dysmenorrhea
-Dyspareunia
-Pelvic pain
-Sacral backache
-Pelvic mass
-Tenesmus and diarrhea with painful BMs
-Urinary frequency
-Infertility
-Lateral displacement of cervix or stenosed os

Workup
-US or MRI
-Laparoscopy for definitive diagnosis (implants will have variable coloration and appearance)

Management
-Endometriosis improves with suppression of ovulation and medical therapy is first line: OCPs, NSAIDs for cyclical pain, GnRH agonists for severe pain (create a hypoestrogenic state)
-Surgical excision for failed medical management
-Adenomyosis is treated with hysterectomy

Prognosis
-Associated with epithelial ovarian cancer but NOT endometrial cancer
-Recurrence is common

OVARY

Functional Ovarian Cysts

-Caused by exaggerations of normal menstrual cycle rather than true neoplasms
-Increased risk with smoking

Types
-Follicular cyst: continued growth of follicle despite failed ovulation
-Corpus luteum cyst: failure of involution with enlargement after ovulation and continued progesterone secretion
-Theca lutein cyst: a result of abnormal pregnancy, uncommon

Signs & Symptoms
-Can be asymptomatic
-Pelvic pain and dyspareunia if large
-Follicular: pelvic pain if rupture
-Corpus luteum: adnexal enlargement, one-sided pain, missed menses
-Torsioned or ruptured cyst will cause acute abdominal pain, rebound tenderness

Differential
-Ruptured ectopic
-Mittelschmerz
-Ovarian torsion
-Degenerating leiomyoma
-PID
-Acute endometritis

Workup
-Must be differentiated from malignancy (benign = mobile, cystic, unilateral, smooth, < 10 cm, minimal septations); get pelvic US

Management
-Will usually regress spontaneously
-Treatment only if recurrent or symptomatic (OCPs, etc)
-Ruptured cyst: expectant management if uncomplicated (no hypotension, tachycardia, fever, leukocytosis, signs of acute abdomen, or US suspicious for malignancy), surgical management if complicated

Prognosis
-Risk of torsion if large or penduculated

Ovarian Neoplasms

-Vary from annoying and benign to invasive and malignant
-Functional ovarian cysts (corpus luteum cyst or follicular cysts) are NOT considered to be neoplasms because they are a result of a normal physiologic process
-Ovarian neoplasms are derived from neoplastic growth of ovarian cell layers

Benign Ovarian Neoplasms
-Mucinous cystadenoma
-Serious cystadenoma
-Endometrioma (chocolate cyst)
-Fibroma
-Brenner tumor
-Thecoma
-Sertoli-Leydig cell tumors
-Dermoid cyst (teratoma): can contain hair, teeth, sebaceous glands, and thyroid cells producing TH
-Uterine leiomyoma

Malignant Ovarian Neoplasms
-Adenocarcinoma
-Granulosa cell tumor
-Dysgerminoma
-Clear cell carcinoma
-Endometrioid carcinoma

Risk Factors
-Nulliparity
-Fertility treatments
-FH of breast or ovarian cancer

Protective Factors
-Prolonged OCP use
-Pregnancy
-Tubal ligation or hysterectomy

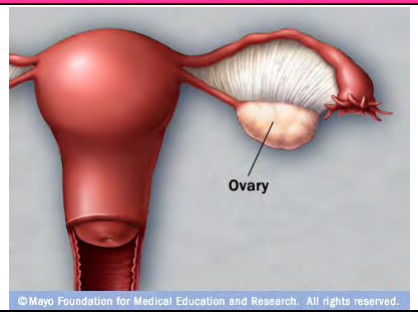
Signs & Symptoms
-Thyrotoxicosis with dermoid tumor
-Torsioned ovary or cyst → signs of acute abdomen
-Malignancy symptoms are nonspecific like pelvic pain and bloating

Workup
-Transvaginal US: signs indicative of malignancy include large amounts of free fluid in the abdominal cavity, solid ovarian enlargement or mixed cystic and solid enlargement, thick-walled or complex ovarian cysts
-Serum CA-125: will also be elevated in infection, endometriosis, ovulation, and trauma
-Staging and grading of malignancies

Management
-Malignancy: local excision vs total hysterectomy and bilateral SO vs partial bowel resection depending on stage of cancer, usually followed by radiation ± chemo
-Benign neoplasms will persist unless excised, which is usually done to prevent ovarian torsion
-Simple cysts in a postmenopausal woman may be followed by serial US and CA-125s

Ovarian Torsion

-A gynecologic emergency caused by ovarian ischemia as a result of complete or partial rotation of the ovary on its ligamentous supports
-Fallopian tube may also be torsioned



Risk Factors
-Ovarian mass
-Ovulation induction for infertility

Signs & Symptoms
-Acute pelvic pain (although rarely can be chronic pelvic pain)
-N/v
-Low grade fever

Workup
-Pelvic US

Management
-Surgical repair with ovarian conservation unless mass or necrosis present

CERVICAL DISORDERS

Cervicitis

Etiologies
-Infectious: chlamydia, gonorrhea, HSV, HPV, trichomoniasis, *Mycoplasma genitalium*, CMV, BV
-Noninfectious: cervical cap, pessary or diaphragm use, chemical or latex allergy, cervical trauma

Signs & symptoms
-Postcoital spotting
-Intermenstrual spotting
-Dyspareunia
-Unusual vaginal discharge
-If chronic → cervical stenosis, leukorrhea, granular redness, erythema, vulvar irritation
-Salpingitis
-Edematous or friable cervix

Workup
-STI testing
-Wet prep
-Pap & pelvic

Treatment
-*Chlamydia* → single azithromycin dose, or doxycycline
-Gonorrhea → ceftriaxone IM or single cefixime oral dose
-HSV → acyclovir
-Trichomoniasis → single metronidazole

Incompetent Cervix (Cervical Insufficiency)

-Painless cervical changes in the 2nd trimester leading to recurrent pregnancy loss, stillbirth, or preterm delivery
-Short cervix is defined as < 25 cm from external to internal os

Causes
-Congenital: short cervix, Mullerian abnormalities, collagen abnormalities, FH
-Trauma: cervical laceration, instrument dilation, cone biopsy, LEEP
-Elevated serum relaxins (higher in multiple gestations)

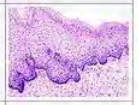
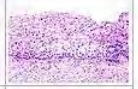
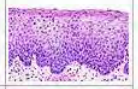
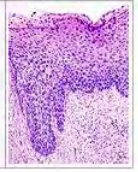
Screening
-UpToDate recommends routine TVUS screening for short cervix in singleton pregnancies at 16-28 weeks

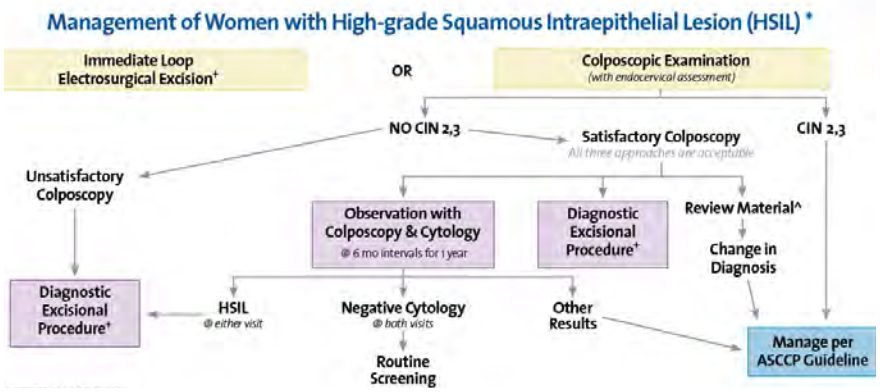
Signs & Symptoms
-Vaginal fullness or pressure
-Spotting or watery or brown discharge
-Vague abdominal and back pain
-Premature cervical effacement and dilation

Workup
-Transvaginal US showing shortened endocervical canal and funneling of fetal membranes into endocervix

Management
-No evidence for bedrest
-Progesterone supplements
-Indomethacin
-Prophylactic cerclage or pessary
-Early US surveillance for women not meeting criteria for cerclage

Cervical Dysplasia

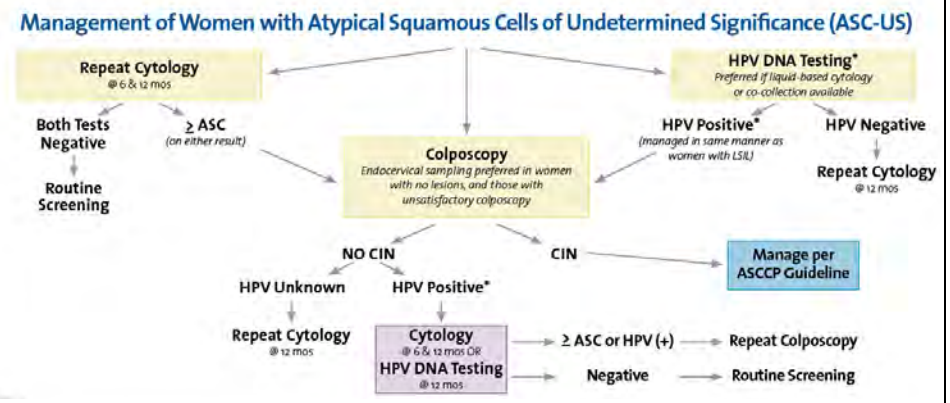
Histology Grade	Corresponding Cytology	Description	Image
–	–	Normal cervical epithelium	
CIN 1 (Grade I)	LSIL[5]	The least risky type, represents only mild dysplasia, or abnormal cell growth.[3] It is confined to the basal 1/3 of the epithelium. This corresponds to infection with HPV, and typically will be cleared by immune response in a year or so, though can take several years to clear.	
CIN 2/3	HSIL	Formerly subdivided into CIN2 and CIN3.	
CIN 2 (Grade II)		Moderate dysplasia confined to the basal 2/3 of the epithelium	
CIN 3 (Grade III)		Severe dysplasia that spans more than 2/3 of the epithelium, and may involve the full thickness. This lesion may sometimes also be referred to as cervical carcinoma in situ.	

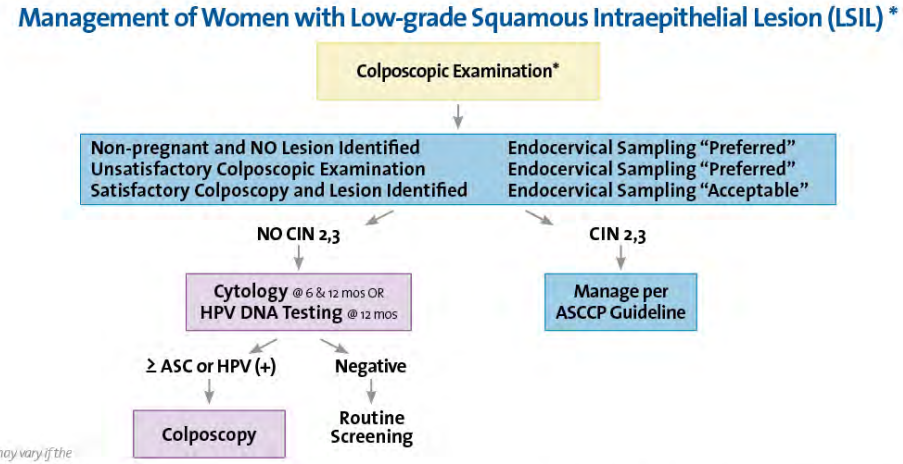


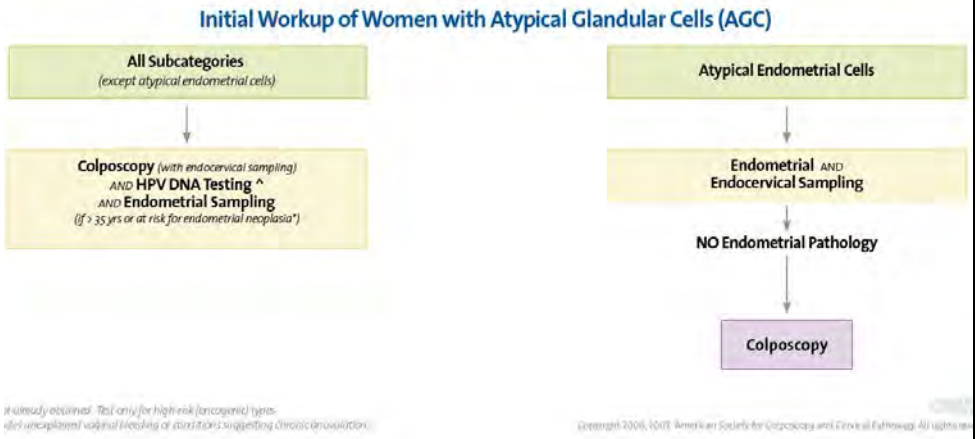
-After HPV infection, epithelia can develop active or latent infection or undergo neoplastic transformation
-HPV types 16 and 18 are more likely to undergo malignant transformation
-Most women will clear HPV infection within 2 years
-CIN = cervical intraepithelial neoplasia (aka cervical dysplasia); premalignant squamous transformation cells (not glandular)
-Bethesda system: ASCUS, LSIL, or HSIL
-Women over 30 who are being screened no more frequently than every 3 years will have HPV testing done automatically with their cytology

Screening
-Begin at age 21 or within 3 years of first sexual contact
-Every 3 years if low risk with 3 consecutive normal paps
-D/c after age 65 if last 3 paps were normal
-Women who have had a total hysterectomy for benign reasons do not need paps

Management
-Differs based on age of patient, as younger women tend to clear the infection before progression to dysplasia
-If ASCUS is detected, specimen will be tested for HPV as well if woman is over 21, or repeat cytology in 6 months if woman is under 21
-LSIL or HSIL or AGC will need referral for colposcopy, where biopsies will be taken
-Biopsies give corresponding histology grade of CIN 1, 2, or 3
-CIN 1 generally observed or may be ablated (cryotherapy or laser)
-CIN 2 or 3 have 5-15% chance of progressing to cervical cancer and these lesions need to be excised via LEEP or conization procedure (↑ risk of preterm labor in future pregnancies)







Cervical Cancer

-80% of women will be infected with a strain of HPV by age 50
-Average age of cervical cancer dx is 51

Cervical carcinoma in situ
-Most common in 25-35 year olds

Cervical squamous cell carcinoma
-The most common form of cervical cancer, accounts for 75% of all cases
-Usually from HPV 16 or 18
-Usually located within 1 cm of the squamocolumnar junction

Cervical adenocarcinoma
-Derived from glandular elements
-Subtypes are mucinous, endometrioid, clear, or serous cell
-Usually from HPV 18, though clear type associated with DES exposure
-More common in women under 35

Screening
-Has resulted in 75% ↓ in cervical cancers
-Pap cytology

Signs & symptoms
-Early disease is asymptomatic
-Abnormal vaginal bleeding or postcoital spotting
-Vaginal discharge
-Foul odor
-Pelvic or flank pain
-Vesicovaginal or rectovaginal fistula
-Weakness
-Weight loss
-Anemia
-Speculum exam shows cervical lesion, enlarged cervix, nodularity, friable tissue, or decreased mobility

Workup
-Biopsy for grossly visible invasive disease
-Imaging for mets

Management
-Carcinoma in situ: LEEP, CKC, simple hysterectomy
-Early stage: radical hysterectomy + pelvic lymphadenectomy, primary radiation with concurrent chemo
-Locally advanced: primary radiation with chemo
-Mets or persistent or recurrent cancer: chemo, palliative radiation,
-Central pelvic recurrence: total pelvic exenteration

Prognosis
-Depends on stage, mets, tumor volume, invasion, and histology
-Good prognosis for early disease responsive to treatment
-Nontreatment or nonresponse yields 5% 2 year survival

VAGINAL & VULVAR DISORDERS

Vulvar Neoplasms

-Vulvar intraepithelial neoplasia (VIN) is a premalignant lesion that is difficult to distinguish or may exist in association with invasive squamous cell carcinoma, lichen sclerosus, or lichen planus
-Malignant lesions include squamous cell carcinoma (90% of vulvar cancers), melanoma, and basal cell carcinoma

Risk Factors
-HPV infection
-Immunosuppression
-Cigarette smoking
-Lichen sclerosus (can transform to SCC)

Differential
-Flesh-colored lesion: sebaceous gland, vestibular papillae, skin tag, cyst, wart, molluscum contagiosum
-White lesion: lichen sclerosus, lichen simplex chronicus, vitiligo
-Brown, red, or black lesion: could be anything, need to biopsy

Signs & Symptoms
-Vulvar pruritus and pain
-Visible or palpable abnormality, may be in multiple locations
-Dysuria

Workup
-Any lesion not previously known on the vulva warrants biopsy via physical exam or colposcopy

Management
-Wide local excision of VIN if high risk based on lesion characteristics and pt age
-Laser ablation or topical therapy with imiquimod for VIN lesions that would cause significant vulvar mutilation if excised
-Excision of malignant lesions with inguinofemoral lymph node evaluation ± chemo or radiation

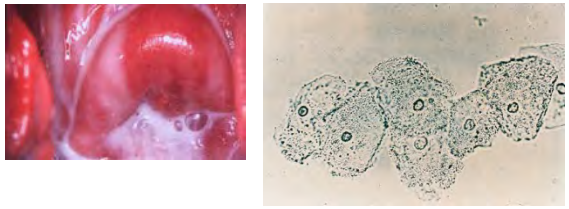
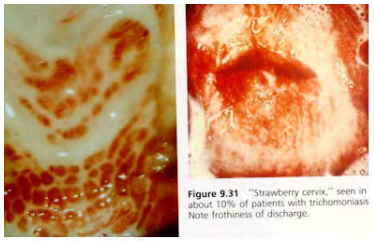
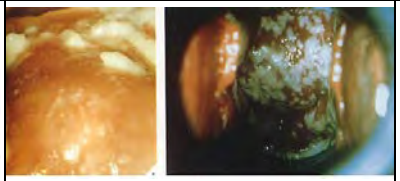
Prognosis
-VIN recurs in 30% of women and 4-8% will go on to develop locally invasive vulvar cancer

Pediatric Vaginitis

Etiologies
-STI
-Vaginal polyp or tumor
-Atrophic prepubertal tissue (more susceptible to irritants)
-*Strep pyogenes* and other respiratory pathogens
-Foreign body
-Pinworms

Urethral prolapse: treat with topical estrogen cream for 2 weeks
-Lichen sclerosus: treat with topical steroids
-Labial adhesions: treat with topical estrogen cream
-Systemic illness: measles, varicella, scarlet fever, EBV, Crohn's, Kawasaki disease
-Nonsexually transmitted vulvar ulcers
-Urethral prolapse
-Ectopic ureter

Management
-Treat underlying cause
-Wear nightgowns to allow air circulation
-Cotton underwear
-Avoid tights, leotards, and leggings
-Bathe in water only for 15 minutes and limit soap to non-genital areas
-Dry genital area well after bathes, can use a cool hair dryer
-No bubble baths or perfumed soaps
-Cool compresses
-Use wet wipes instead of toilet paper
-Avoid sitting around in wet swim suits
-Antibiotic therapy for purulent discharge that does not respond to hygiene measures

Adult Vaginitis				
Etiology	**Bacterial vaginosis**	**Trichomoniasis**	**Yeast vaginitis**	**Atrophic vaginitis**
Info	-Polymicrobial overgrowth of normal flora	-Usually sexually transmitted	-Agent is Candida albicans -May be ppt by hormonal changes, oral steroids or abx, nylon panties, hot weather, obesity	-Inflammation of the vagina due to thinning and shrinking of tissues and decreased lubrication -Seen in women with decreased estrogen
Signs & symptoms	-Fish odor -Heavy bubbly discharge that is white or gray	-Copious vaginal discharge -Pruritus -Dysuria -Dyspareunia -Abdominal pain -Vaginal and cervical inflammation with punctate hemorrhages = strawberry cervix	-Pruritus -Burning -Nonfoul cottage cheese discharge -Dyspareunia -Vaginal or vulvar erythema -May be asymptomatic	-Pruritus -Burning -Vaginal dryness -Dyspareunia -Spotting -Pale, thin vaginal mucosa
Workup	-Wet prep: clue cells, alkaline pH -Whiff test	-Wet prep: look for motile trichomonads	-KOH wet prep	-Must r/o infectious cause -Negative wet prep
Treatment	-1st line is metronidazole or clindamycin cream	-Single dose of metronidazole	-Single dose of butoconazole or fluconazole -1st trimester pregnancy use itraconazole -OTC Monistat only treats *Candida albicans* and not other 2 species	-Estrogen replacement therapy -Regular sexual activity -Lubricants and vaginal moisturizers
Picture				

MENSTRUAL DISORDERS		
Premenstrual Syndrome		
-More severe form is premenstrual dysphoric disorder **Signs & Symptoms** -Symptoms begin with ovulation and last 2 weeks until menses -Acne, breast swelling, fatigue, GI upset, insomnia, bloating, HA, food cravings, depression, anxiety, irritability	**Workup** -PMDD defined by 5+of the following: sadness, despair, suicidal ideation, tension, anxiety, panic attacks, irritability affecting others, mood swings, crying, disinterest in daily activities, binge eating, cravings	**Management** -Exercise -Regular sleep -Stress management -Healthy eating habits -Avoiding caffeine, sugar, and salt -OCPs for severe symptoms -Consider antidepressants or counseling for PMDD

Amenorrhea

Primary Amenorrhea

-Failure to menstruate by age 16 in presence of 2˚ sex characteristics or failure to menstruate by age 14 in absence of 2˚ sex characteristics

Etiologies
-Chromosomal abnormality → gonadal dysgenesis
-Central: tumors, infiltration of hypothalamus or pituitary, congenital GnRH deficiency, hypoprolactinemia, disrupted GnRH pulsations
-PCOS
-Anatomic abnormality or absence of uterus, cervix, or vagina

Workup
-Physical exam for sex characteristics and normal anatomy (breast development indicates estrogen effects and functioning ovaries)
-US to look for presence of uterus
-FSH level to determine whether cause is central or ovarian
-Karyotype if breast development not present
-Normal FSH, signs of breast development, and presence of uterus indicate further workup for secondary causes of amenorrhea

Management
-Treat underlying pathology
-Achieve fertility if desired
-Prevent complications of disease process

Secondary Amenorrhea

-Cessation of menses for a period of time = to 3 cycles or 6 months in a woman who previously had menses

Etiologies
-Pregnancy
-Functional hypothalamic amenorrhea: excessive exercise, eating disorder, systemic illness, psychological stress
-Hyperprolactinemia: pituitary tumor, medications, hypothyroidism
-PCOS
-Premature ovarian failure
-Endometrial scarring (Asherman's)

Workup
-Physical exam for hirsutism, acanthosis nigricans, vitiligo, galactorrhea, and signs of estrogen deficiency or eating disorder
-Serum hCG, FSH, LH, PRL, TSH, progesterone, ?DHEAS (false negs)
-Serum total testosterone with signs of hyperandrogenism
-Other workup based on clinical findings

Management
-All depend on desire for fertility
-Hypothalamic amenorrhea: sufficient calorie intake, CBT, leptin administration
-Hyperprolactinemia: dopamine agonist or surgical treatment
-Premature ovarian failure: OCPs to prevent bone loss
-PCOS treatment
-Hysteroscopic lysis of intrauterine adhesions

Dysmenorrhea

Primary = painful menses with normal anatomy

-Leading cause of school absences
-Incidence decreases after age 20

Cause
-Usually prostaglandins and uterine vasoconstriction

Signs & Symptoms
-Cramping pain radiating to back or inner thighs
-May have associated heavy flow, n/v/d, HA, dizziness

Management
-NSAIDs beginning 1-2 days before expected menses
-OCPs
-Progesterone
-Mirena IUD
-Acupuncture
-Thiamine supplementation

Secondary = a result of disease or pathology

Causes
-GYN: endometriosis, uterine fibroids, adenomyosis, STIs, endometrial polyps, ovarian cysts, pelvic adhesions, chronic PID, cervical stenosis
-Non-GYN: IBD, IBS, uteropelvic junction obstruction, psychogenic

Signs & Symptoms
-Usually begins well after menarche

Workup
-Refer for laparoscopy

Management
-Treat underlying cause
-NSAIDs
-OCPs
-IUD
-Refer to OB-GYN for uterine artery embolization and evaluation for hysterectomy

BREAST DISORDERS

Breast Abscess

-Can be lactational or nonlactational
-Etiologist is usually *Staph aureus*, with MRSA becoming increasingly more common

Risk Factors
-Obesity
-Smoking
-Black

Workup
-Wound culture

Management
-Needle aspiration if overlying skin is intact
-I&D for compromised overlying skin or failed needle aspiration
-Antibiotics: dicloxacillin, cephalexin, or clindamycin
-Bactrim, clindamycin, or linezolid if suspecting MRSA
-Continue breastfeeding

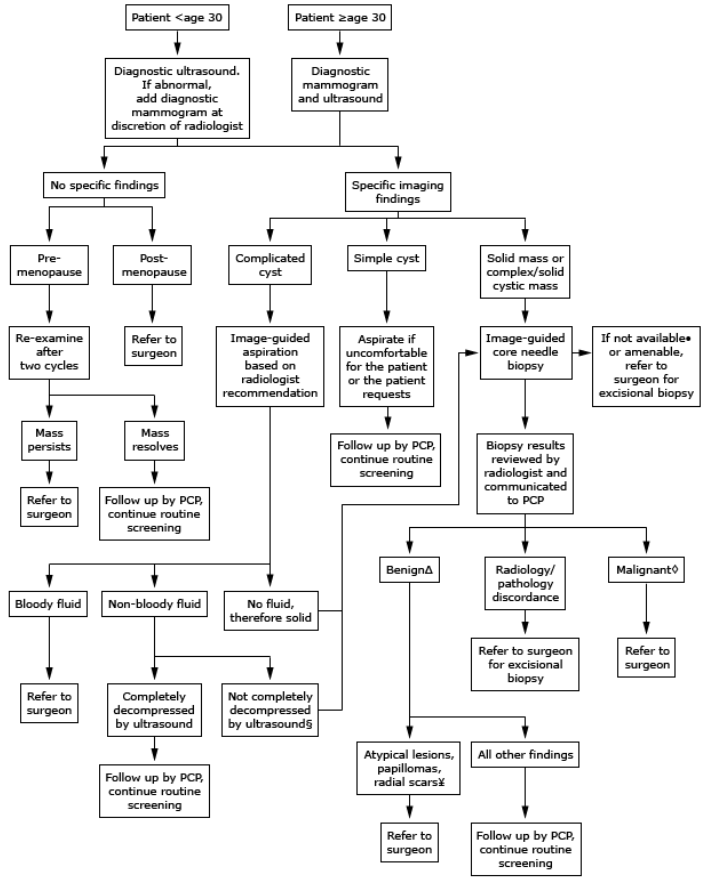
Breast Mass

Screening
-Can assess risk for breast cancer using Gail Model
-Screening mammo for ages 40-49 is grade C
-Screening mammo recommended for women 50-74 every 2 years
-Breast self-exam is grade D
-Dedicated breast MRI for select high risk patients

Signs & symptoms
-Mastalgia is rarely associated with breast cancer
-Nipple discharge: only 2-3% will have malignancy
-Concerning: single, nontender firm mass with ill-defined margins, skin or nipple retractions, axillary adenopathy, breast enlargement, erythema, peau d'orange, edema, pain, fixation of mass to chest wall

Differential
-Fibrocystic changes
-Fibroadenoma
-Intraductal papilloma
-Fat necrosis
-Abscess
-Malignancy

Workup



Breast Cancer

-Usually arises from ducts or lobules
-Most commonly diagnosed female cancer
-Only 5-10% are due to genetic mutations

Risk Factors
-Obesity or inactivity
-Use of hormone therapy
-Nulliparity
-First birth after age 30
->1 alcoholic drink per day
-Not breastfeeding
-Increasing age
-White
-Hx of chest irradiation
-Hx of atypical hyperplasia on previous biopsy
-FH of breast cancer or inherited mutations

Prevention
-Women with high risk can consider chemoprevention with tamoxifen or raloxifene

Screening
-Mammography is USPSTF grade C for women 40-49, grade B for women 50-74 every 2 years
-Clinical breast exam
-Breast self-exam is USPSTF grade D
-Dedicated breast MRI for high risk populations

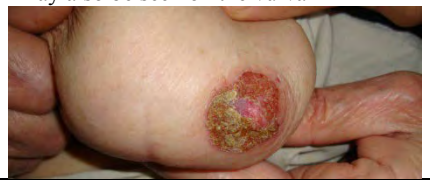
Signs & Symptoms
-Most commonly found on the upper outer quadrants
-Early: single, painless firm mass with ill-defined margins or possibly no palpable mass but an abnormality is detected on mammogram
-Later: skin or nipple retractions, axillary adenopathy, breast enlargement, erythema, peau d'orange, edema, pain, fixation of mass to chest wall
-Very late: ulceration, supraclavicular adenopathy, arm edema, mets to bone, liver, lung, brain, or adrenal glands

Differential
-Fibrocystic disease
-Fibroadenoma
-Intraductal papilloma
-Lipoma
-Abscess
-Fat necrosis
-Phyllodes tumor

Workup
-Biopsy of suspicious lesion
-Pathology and genomic marker assay

Management
-TNM classification
-Tumor marker profiling
-Surgical management: lumpectomy, sentinel node biopsy, or mastectomy
-Chemo is typically 3-6 months and is initiated for visceral mets, failed endocrine therapy, or ER-/PR- tumors
-Endocrine therapy with tamoxifen (premenopausal)or aromatase inhibitors (postmenopausal)
-Radiation therapy as an adjuvant

Prognosis
-Surgical complications: long thoracic nerve injury, lymphedema

Breast Cancer Type	Info	S/S	Management
Ductal Carcinoma in Situ	-Some consider this to be a pre-malignant lesion -Arises from ductal hyperplasia and fills ductal lumen -Very early malignancy without basement membrane penetration -Less than 30% recurrence rate following lumpectomy	-Typically asymptomatic and discovered on screening mammogram as calcifications -Usually not palpable on PE	-Lumpectomy followed by radiation is most common -Tamoxifen or aromatase inhibitor therapy for 5 years if receptor+ tumor
Invasive Ductal Carcinoma	-The most common breast cancer -Worst and most invasive	-Pt is typically postmenopausal -Mammogram detects spiculated margins -Firm, fibrous, rock-hard mass with sharp margins and small, glandular, duct-like cells -Likes to metastasize	-Chemo with Herceptin and Tykerb for HER2+ tumors
Lobular Carcinoma in Situ	-Some consider this to be a pre-malignant lesion -Contains signet ring cells -Will progress to invasive lobular carcinoma in 10%	-Usually not palpable and hard to detect on mammo -Often bilateral	
Invasive Lobular Carcinoma	-2nd most common breast cancer	-Orderly row of cells in stroma that are fluid and mobile -Often bilateral	-Assessment with US preferred over mammography
Medullary Carcinoma	-Fleshy, cellular, lymphocytic infiltrate -Good prognosis although it is a rare subtype of invasive ductal carcinoma	-Mammogram detects linear crystallization pattern	
Comedocarcinoma	-Subtype of DCIS -Ductal caseating necrosis		
Paget's Disease of the Breast	-Subtype of ductal carcinoma	-Presents as eczematous lesions on the nipple -May also be seen on the vulva 	

Fibroadenoma

-Common benign neoplasm in young women composed of fibrous and glandular tissue

Signs & Symptoms
-Pt is usually in teens to 30s
-Firm, round, nontender, mobile 1-5 cm nodule that is solitary and unilateral
-Growth is hormonally affected and can be rapid during pregnancy

Workup
-Consider malignancy or fibrocystic changes in women > 30
-US for younger women
-FNA or needle biopsy for confirmatory diagnosis

Management
-Observation if malignancy has been ruled out
-Surgical excision if unable to r/o malignancy or if large

Prognosis
-Can recur after excision

Fibrocystic Breasts

-The most common benign condition of the breast
-Uncommon in postmenopausal women unless on HRT

Signs & Symptoms
-Patients are usually ages 30-50
-Pain or tenderness in the breasts
-Cysts or multiple transient lumps that are firm, mobile, and tender
-Changes are related to menstrual cycle and can be worsened by caffeine

Workup
-Differentiate from fibroadenoma or malignancy by the presence of multiple transient lesions
-Further workup via US or mammogram indicated for lesions that persist throughout menstrual cycle

Management
-Counseling to wear supportive bra
-Avoiding trauma and caffeine
-Danazol for severe persistent pain
-Evening primrose oil

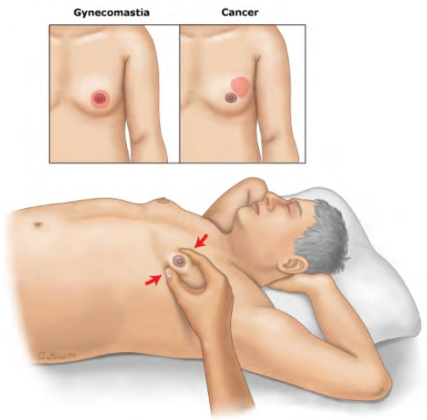
Gynecomastia

-Benign proliferation of glandular tissue of the male breast
-Imbalance between stimulatory effect of estrogen and inhibitor effects of androgens

Etiologies
-Physiologic: common and self-limiting in infant and adolescent males
-Drugs: finasteride, spironolactone, ketoconazole, metronidazole, omeprazole, ACEI, amiodarone, digoxin, methyldopa, HAART, street drugs, anabolic steroids, diazepam, TCAs
-Cirrhosis or malnutrition
-Male hypogonadism
-Testicular tumor
-Cryptorchidism
-Hyperthyroidism
-CKD
-Idiopathic

Signs & symptoms
-Rubbery or firm mass extending concentrically from the nipple

Differential
-Male breast cancer
-Pseudogynecomastia: fat deposition without glandular proliferation, seen in obese men



Workup
-Indicated for recent-onset or painful gynecomastia
-Not necessary for pts with underlying contributing illness or use of drugs associated with gynecomastia
-Serum hCG
-LH
-Testosterone
-Estradiol

Management
-Observation with reevaluation in 3-6 months or brief SERM trial if tender or cosmetic concern

Galactorrhea

-Non-pathologic physiologic nipple discharge in the absence of childbirth or nursing
-Considered normal when secreted for a short period of time from newborns

Etiologies
-Excessive nipple stimulation
-Pituitary adenoma
-Acromegaly
-Other causes of hyperprolactinemia
-Hyperthyroidism
-Meds: methyldopa, opiates, SSRIs, antipsychotics
-Licorice
-Dopamine release from surgery, trauma, or anesthesia

Signs & symptoms
-Usually bilateral
-Can appear in a variety of colors

Differential
-Non-physiologic nipple discharge such as breast cancer
-Pregnancy

Workup
-Breast exam
-Hemoccult testing of discharge
-Visual field exam
-Mammogram for women > 30
-US if concern for pathologic discharge
-Urine bHCG
-Bilateral discharge: prolactin, BMP, TSH

Management
-Reassurance if cause is benign
-Surgical terminal duct excision if concern for pathologic discharge

Mastitis		
Agents -*Staph aureus*, increasingly MRSA **Signs & Symptoms** -Usually unilateral -Fever and flulike symptoms -Erythema, warmth, tenderness, and hardness of affected breast	**Workup** -Malignancy workup if occurring in a non-lactating woman -US to differentiate from abcess if needed	**Management** -Continue breastfeeding or pumping -Ibuprofen -Cold compresses -Oral dicloxacillin, 1st generation cephalosporin, or erythromycin if not suspecting MRSA -Severe infection with MRSA risk → Bactrim, clindamycin, or linezolid

CONTRACEPTIVE METHODS			
Misc. Methods			
	Info	**Failure Rate**	**Cost**
Withdrawal		4-27%	
Fertility Awareness	-Includes rhythm method, natural family planning, and symptothermal method -Based on consistent symptoms of ovulation -Effective if regular cycles -Must be committed, motivated, vigilant -Control of fertility -No chemicals, hormones or foreign objects -Inexpensive -Accepted by religious organizations -Use alternate methods or alternate forms of pleasure during 'unsafe' days -Decreases spontaneity -Unreliable if irregular cycles -Perimenopausal years more difficult	9-25%	
Barrier Methods			
	Info	**Failure Rate**	**Cost**
Spermicide	-Only kind available in US is nonoxyl-9 -Natural alternatives: lemon juice, vinegar, neem oil -Comes as a vaginal film, suppository, cream, gel, or lubricant -No STI protection, can acually cause allergies an irritation → ↑ risk of STIs	10-29%	$0.50-$1.50 per application
Cervical Cap	-Silicone cap filled with spermicide -Only kind approved in US is the FemCap (others associated with abnormal paps) -Requires prescription and fitting -Can be inserted up to 24 hours before sex and worn for up to 48 hours -No STI protection -Increased risk of nonmenstrual toxic shock	7.6-14%	$89 + exam & fitting Free for insured under new ACA legislation
Diaphragm	-Rubber that is filled with spermicide -Requires prescription and fitting -No STI protection -Increased risk of UTIs, vaginitis, and nonmenstrual toxic shock	10-20%	$15-$75 + exam & fitting Free for insured under new ACA legislation
Female Condom	-Synthetic nitrile	5-20%	$2-$4 each
Male Condom	-Latex, polyurethane, natural, or "spray on" -Often prelubricated with spermicide -Can cause UTIs in female partners -No STI protection with natural condoms	3-15%	$0.25-$2 each
Sponge	-Polyurethane with spermicide -Does not prevent STIs		$13-$19 for 3

Hormonal Methods

-Absolute contraindications to all estrogen-containing BC (per CDC):

- Age > 35 and smoking > 15 cigs/day
- Known CAD
- Multiple risk factors for CAD: DM, HTN, smoking
- HTN
- H/o DVT, PE, stroke, or migraine with aura
- Known coagulopathy
- Complicated valvular heart disease: pulm HTN, afib, h/o bacterial endocarditis
- SLE
- Breast cancer
- Cirrhosis, hepatocellular adenoma, or malignant hepatoma

-Relative contraindications to all estrogen-containing BC:

- Gall bladder disease
- H/o cholestatic jaundice in pregnancy
- Epilepsy
- Clot risks: leg injury or cast, elective surgery, sickle cell disease
- Obesity

	Info	Failure Rate	Cost
Combined OCPs	-Estrogen portion suppresses the FSH surge by negative feedback → ovulation inhibition, also alters endometrium and causes degeneration of the corpus luteum -Progestin portion suppresses LH surge → inhibited ovulation, also thickens cervical mucus to inhibit implantation -Benefits: improvement of acne, DUB, mittelschmerz pain, endometriosis, ovarial failure, ovarian cysts, uterine fibroids, fibroadenomas or fibrocystic breasts, iron deficiency anemia; decreases risk of ovarian and endometrial cancers, ectopic pregnancy, and acute PID -Adverse effects: nausea, vomiting, weight changes, spotting, migraines, edema, rash, depression, decreased libido, ? ↑ risk of breast cancer, ↑ risk benign liver tumors, worsening gallbladder problems, blood clots, stroke -Need to adjust strength and estrogen/progesterone formulation if adverse effects are present -Most to least androgenic progestins: norgestrel, levonorgestrel, norethindrone, norethindrone acetate, ethynodiol, norgestimate, desogestrel, drospirenone -Ethynodiol is the only highly estrogenic estrogen, all others have lower estrogenic effects -No protection against STIs	3-9%	$15-$30 per month Free for insured under new ACA legislation

Adverse Effect	Causes	Management
Breakthrough bleeding	Need higher progestin content to increase endometrial support	- Monophasic formulation with a higher progestin dose - Triphasic formulation with increasing dose of progestin - higher dose of estrogen
Acne, oily skin, and hirsutism	Side effects from progestins	Product with lower risk of androgenic effects
GI complaints	Estrogen and progesterone	- Estrogen – induces nausea and vomiting via the CNS - Progesterone – slows peristalsis, causing constipation and feelings of bloating and distention
Headaches		- discontinue the oral contraception - lower the dose of estrogen - lower the dose of progestin - eliminate the pill-free interval for 2-3 consecutive cycles
Decreased libido and depression	Low levels of estrogen ↓ vaginal lubrication	Use of the NuvaRing may help with lubrication disorders
Dyslipidemias	Estrogen	Replace an androgenic progestin with a more estrogenic progestin
Mastalgia	Estrogen component	- lower-dose estrogen pills - if tenderness occurs prior to menses, switch to a contraceptive that offers extended cycle length
Weight gain	High estrogen content	Switch to an estrogen product with <35 mcg of ethinyl estradiol
Visual changes/ contact lens disturbances	Estrogen stimulation of melanocyte production	- progestin-only products - use sunscreen - refer to ophthalmologist if normal saline eye drops do not help

Info		Failure Rate	Cost
Progestin-Only Pill	-Must be taken with obsessive regularity -Can have irregular bleeding -A good option for breastfeeding women, smokers > 35, or those who can't tolerate estrogen	1-13%	Free for insured under new ACA legislation
Vaginal Ring	-May be removed for up to 3 hours during intercourse without backup protection -Adverse effects: vaginitis, HA, leukorrhea, FB sensation, device expulsion, feeling it during sex	1-2%	$15-$70 per month Free for insured under new ACA legislation
Transdermal Patch	-Can bathe, swim, or exercise with patch in place -Must use back-up if patch falls off > 1 day	0.3-8%	Free for insured under new ACA legislation
Medroxyprogesterone Injection	-IM injection q 3 months -Results in amenorrhea after a year or so of use -Can use if smoker or nursing -Decreased risk of PID and endometrial cancer -AEs: bleeding abnormalities, weight gain, lipid changes, depression, acne, HA, delay in return to fertility -Black box warning for ↑ risk osteoporosis related to duration of use = should only use < 2 years -No protection against STIs	1-2%	$35-$75 per injection Free for insured under new ACA legislation
Progesterone Implantable Rod	-Must be trained by company-approved provider to insert and remove -Good option for smokers or those who have contraindications to estrogen -May be less effective in obese patients -AEs: menstrual irregularity, amenorrhea, weight gain, acne, depression	1-4%	$400-$800 for insertion $75-$150 for removal Free for insured under new ACA legislation
Mirena IUD	-Changes mucus and sets up hostile environment for sperm -Questionable use in individuals at risk for STIs -Often used in later reproductive years before menopause -Decreased risk of endometrial cancer -Can be in place up to 5 years -Women may become amenorrheic after a year of use -Less bleeding and cramping than with copper IUD -Increased risk of ovarian cysts -May want to culture IUD for *Actinomyces* after removal	0.2%	
Surgical Methods			
Info		**Failure Rate**	**Cost**
Vasectomy	-Cutting and sealing the vasa deferentia -Clinic procedure under local anesthesia -Recovery period of 2-3 days -Men will still be fertile for several ejaculations afterwards, need to have semen analysis in 1 month to confirm sterility	0.15%	$350-$1000
Tubal Ligation	-An outpatient surgery under general anesthesia -Recovery period of 1 week -Benefits: ↓ risk ovarian cancer and possibly breast cancer, can be done immediately postpartum -Increased risk of ectopic pregnancy -Need to confirm blockage with hysterosalpingogram	0.5%	$1500-$6000

Other Methods			
Other Methods	**Info**	**Failure Rate**	**Cost**
Paragard IUD	-Changes mucus and sets up hostile environment for sperm -Questionable use in individuals at risk for STIs -Often used in later reproductive years before menopause -Decreased risk of endometrial cancer -Can be in place for up to 10 years -Can cause heavy bleeding and cramping -May want to culture IUD for *Actinomyces* after removal	0.6-1.0%	
Lactation	-Most effective if infant is not taking any supplemental formula and mother is nursing at least every 4 hours	10%	
Emergency Methods			
	Info	**Failure Rate**	**Cost**
Morning After Pill (Plan B One-Step, Next Choice)	-Not an abortifacient = won't work if already implanted -No evidence of teratogenic effects -Best if initiated within 72 hours of unprotected sex but can be taken for up to 5 days afterward -Rare risks or AEs, but may need prophylactic antiemetics before taking -Available without a prescription for ages 17+		
Ulipristal acetate (Ella)	-Selective progesterone receptor modulator -Rx only		
Mifepristone (RU486)	-Use within 72 hours of unprotected sex -An abortifacient = will dislodge implanted embryo -Also inhibits ovulation and changes endometrium	15%	

UNCOMPLICATED PREGNANCY		
Normal Labor & Delivery		
Stages of Labor	**Signs & Symptoms**	**Management**
-1st stage: onset of labor to full dilation of 10 cm -2nd stage: interval between full dilation and delivery of fetus -3rd stage: time from fetal delivery to expulsion of placenta **Factors Influencing Course of Labor** -Powers: uterine contractions -Passenger: fetal size and number, lie, presentation, station, presence of any fetal anomalies (ideally fetus is small and in vertex position, longitudinal lie, with head flexed and in anterior position and passing through pelvic inlet) -Passage: pelvis and surrounding soft tissues	-Sequential changes within the myometrium and cervix take place over days to weeks -Loss of mucus plug → "bloody show" -Progressive cervical dilation and effacement: should dilate at > 1.2 cm/hr for nulliparous women and > 1.5 cm/hr for multiparous women -Fetal head can be observed to rotate as it navigates the birth canal ("cardinal movements of labor")	-Measurement of uterine contractions via tocodynamometer -Continuous fetal HR monitoring -Adequate labor for delivery is 3-5 contractions in a 10 minute period -IV placement -Pain management: parenteral analgesics vs epidural anesthesia -Clear liquid diet during labor -Consider C-section for labor dystocia ("failure to progress") -Consider operative vaginal delivery (use of forceps or vacuum assistance) for fetal distress, maternal exhaustion, or prolonged 2nd stage of labor -Routine episiotomy not recommended, instead repair lacerations if they present -Deliver placenta within 30 minutes of birth of fetus and examined to be sure it is intact

Prenatal Care												
Week(s)	**Initial visit: 8-12**	**16**	**20**	**24**	**28**	**30**	**32 & 34**	**36**	**37**	**38 & 39**	**40+**	**Postpartum**
Discussion highlights	-History -Counseling -Anticipatory guidance -Genetic screening options		-Begin fetal movements -Round ligament spasms → flank pain	-Importance of daily fetal movements from here on -Discuss preterm labor		-Pregnancy ROS: cramping, bleeding, n/v, constipation, fetal movement, leakage, contractions, preeclampsia sx (HA, vision Δ, edema, RUQ pain, ↓ urine output)			-Signs of true contractions -Loss of mucus plug -Pregnancy ROS	-Pregnancy ROS	-Postterm once > 42 weeks, discuss induction	-1 week incision check for C-sections -2 week check for vaginal deliveries -4-6 week f/u for everyone -Adjustment, breastfeeding, postpartum depression, return to sex, contraception, bowel movements, lochia
Complete PE	✓											
Pap & pelvic	✓											✓
Weight, BP check, fetal heart tones	→	→	→	→	→	→	→	→	→	→	→	
Measure fundal height			*Follow up with US for height > 3 cm discrepancy from gestational age* →									
Leopold's maneuvers					→	→	→	→	→	→	→	
Cervical checks								→	→	→	→	
Imaging	*TVUS for dating*		*20 week US to assess fetal anatomy and size*	*Consider additional US for select high risk pregnancies or inability to measure accurate fundal heights: h/o preterm labor (cervical length), obesity, DM, multiple gestation*								
NST				*Consider for high risk women: IDDM, AMA, maternal heart defect, intrauterine growth restriction, multiple gestation*							✓ *Biweekly*	
Genetic screen	*10-13 weeks: CVS* *11-14 weeks: PAPP-A, NT*	*15-22 weeks: window for quad screen and amniocentesis*										
bHCG	✓											
CBC	✓				✓							
T&S	✓											
GC/C	✓							✓				
RPR	✓							✓				
HIV	✓							✓				
Hep B surface antigen	✓							✓				
Varicella & rubella titers	✓											
Vit D level	✓											
Glucose tolerance test	*Consider for select high risk individuals*				✓							
HSV, TB, TSH, urine drug screen		*If hypothyroid need to follow TSH q 8 weeks with goal TSH 2-3*										
Urine dip	✓	*Consider repeat or frequent UAs for certain high risk individuals: UTI at initial visit, h/o pyelonephritis or kidney problem, symptoms of preeclampsia or diabetes → culture if + and f/u with test of cure* *F/u proteinuria with preeclampsia labs: 24 hour urine, CMP, PT/PTT, uric acid*										
Rhogam administration	*Only give for abnormal bleeding during this time*				✓							✓ *Before leaving hospital*
GBS swab								✓				

COMPLICATED PREGNANCY

Induced Abortion

-98% of unsafe induced abortions occur in the developing world
-Many US states have limits on abortions after 20 weeks

Methods
-Surgical: D&C, vacuum
-Medical: for women < 63 days since LMP

Workup
-Confirm pregnancy and gestational age
-CBC and T&S

Management
-Antibiotic prophylaxis: doxycycline
-Rhogam if indicated

Prognosis
-Surgical complications: cervical laceration, hemorrhage, uterine perforation, incomplete abortion, sepsis
-Psychological complications? Studies show women post-abortion have no higher incidence of mental health disorders

Spontaneous Abortion

-Pregnancy that ends spontaneously before fetus has reached age of viability (= before 22 weeks)
-80% occur in the first trimester
-Occurs in up to half of all pregnancies, although only half of these are diagnosed

Causes
-Chromosomal abnormalities, esp trisomy 16
-Fibroids, polyps, or scarring
-Thrombosis or other placental complication
-Infection
-Fetal exposure

Risk Factors
-Maternal or paternal age
-Increasing parity
-Smoking
-Cocaine or caffeine
-High BMI
-Submucosal fibroids or other uterine abnormality
-Asherman's syndrome
-DM or PCOS
-Thyroid disease
-H/o spontaneous abortion

Signs & Symptoms
-Vaginal bleeding
-Abdominal pain or cramping
-Open cervical os
-Products of conception visualized in the vagina or cervical os
-Signs of hemodynamic instability and fever if septic

Differential
-Physiologic bleeding from implantation
-Ectopic pregnancy
-Cervical polyp
-Cervical infection or neoplasia
-Recent sex

Workup
-US: no cardiac activity in a fetus with CRL > 6 mm or no growth of pregnancy over one week are diagnostic for miscarriage; bad signs indicated miscarriage include yolk sac abnormalities, fetal HR < 100, and large subchorionic hematoma
-Serial quantitative hCGs: normal doubling is reassuring

Management
-Follow quantitative hCG to zero
-May need surgical intervention: D&C
-Medical management (90% efficacy): mifepristone or misoprostol
-Expectant management is an option as long as there is minimal bleeding or discomfort, pt is < 13 weeks, stable VS, and no evidence of infection (80% efficacy but can take days to weeks)
-Administer Rhogam if mother is Rh-
-Methylergonovine maleate to control bleeding
-Broad spectrum abx if septic abortion (clindamycin + gentamicin or Zosyn)
-Grief counseling
-Pelvic rest for 2 weeks
-No evidence for avoiding pregnancy for 2-3 cycles
-Contraception if desired

Cesarean Section

Indications
-Indicated when clinician and patient feel that abdominal delivery is likely to provide a better maternal or fetal outcome vs vaginal delivery
-Failure to progress
-Nonreassuring fetal status
-Fetal malpresentation
-Maternal infection
-Multiple gestation
-Fetal bleeding diathesis
-Cord prolapse
-Suspected macrosomia

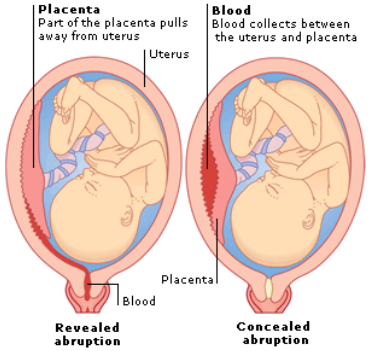
Scheduled C sections
-Should be done at 39-40 weeks, or at 39 weeks if prior C section
-Higher risk of increased hospital stay, neonatal respiratory problems, abnormal placentation in future pregnancies
-Lower risk of fetal injury than planned vaginal deliveries

Technique
-Pre-op abx: cefazolin
-SCDs for DVT prophylaxis
-Transverse abdominal incision preferred over vertical (less post-op pain, greater wound strength, better cosmetic results)
-Transverse hysterotomy rather than vertical (less blood loss, lower risk of rupture)
-Spontaneous placental extraction preferred

Post-operative risks
-Infection: increased over vaginal
-Hemorrhage
-Injury to pelvic organs: urinary, GI
-Ileus
-Thromboembolism
-Future abnormal placentation
-Abdominal adhesions
-Numbness or pain from ilioinguinal nerve laceration
-Increased risk of uterine rupture in subsequent pregnancies
-Wound dehiscence

Abruptio Placentae (Placental Abruption)

-Partial or complete separation of the placenta from the uterine wall prior to delivery of the fetus



Risk Factors
-High: cocaine use, trauma, polyhydramnios, eclampsia, prior abruption, chronic HTN, PROM, chorioamnionitis, fetal growth restriction, smoking
-Moderate: AMA, multiparity, male fetus

Signs & Symptoms
-Painful vaginal bleeding
-Tender uterine fundus
-Contractions
-Abdominal pain
-Can be asymptomatic
-Can be chronic: light, intermittent vaginal bleeding, oligohydramnios, fetal growth restriction, and preeclampsia

Workup
-US to eval for retroplacental hematoma but sensitivity is only 25-50% = diagnosis is clinical
-Blood type and Rh status
-NST

Management
-Fetal HR abnormality on NST suggests impending distress and emergency management
-Stabilization of maternal hypovolemia with large bore IV access with blood replacement
-Expeditious delivery for nonreassuring fetal HR, maternal instability, or gestational age > 36 weeks (should be C-section if unstable or with malpresentation)
-Expectant management of select cases in pregnancies < 36 weeks with administration of glucocorticoids in fetuses 23-34 weeks

Prognosis
-Separation > 50% usually leads to acute DIC and fetal death
-Increased risk of abruption in all future pregnancies

Cesarean Section

Indications
-Indicated when clinician and patient feel that abdominal delivery is likely to provide a better maternal or fetal outcome vs vaginal delivery
-Failure to progress
-Nonreassuring fetal status
-Fetal malpresentation
-Maternal infection
-Multiple gestation
-Fetal bleeding diathesis
-Cord prolapse
-Suspected macrosomia

Scheduled C sections
-Should be done at 39-40 weeks, or at 39 weeks if prior C section
-Higher risk of increased hospital stay, neonatal respiratory problems, abnormal placentation in future pregnancies
-Lower risk of fetal injury than planned vaginal deliveries

Technique
-Pre-op abx: cefazolin
-SCDs for DVT prophylaxis
-Transverse abdominal incision preferred over vertical (less post-op pain, greater wound strength, better cosmetic results)
-Transverse hysterotomy rather than vertical (less blood loss, lower risk of rupture)
-Spontaneous placental extraction preferred

Post-operative risks
-Infection: increased over vaginal
-Hemorrhage
-Injury to pelvic organs: urinary, GI
-Ileus
-Thromboembolism
-Future abnormal placentation
-Abdominal adhesions
-Numbness or pain from ilioinguinal nerve laceration
-Increased risk of uterine rupture in subsequent pregnancies
-Wound dehiscence

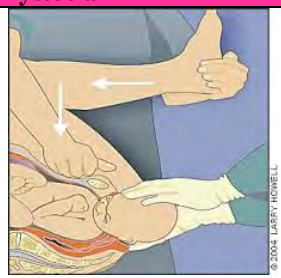
Shoulder Dystocia

-When shoulders of infant can't fit through pubic symphysis because they are wider than the pelvic outlet

Risk Factors
-Maternal obesity
-DM
-H/o or current macrosomic infant
-H/o shoulder dystocia

Prevention
-Routine prophylactic C-section not indicated for suspected macrosomia but can be considered in mothers with h/o shoulder dystocia and brachial plexus injury

Signs & Symptoms
-Prolonged 2nd stage of labor
-Recoil of infant head on perineum ("turtle sign")
-Lack of spontaneous restitution (translation: no natural head turning)



Management
-Get help
-Episiotomy
-McRobert's maneuver
-Drain bladder and disimpact bowel

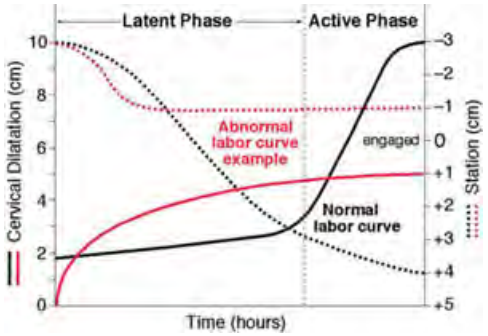
Prognosis
-Fetal complications: brachial plexus injury, clavicular or humeral fracture, increased risk of asphyxia
-Maternal complications: hemorrhage, 4th degree tear

Labor Dystocia

-Failure of labor to progress as anticipated

Causes and Risk Factors
-Hypocontractile uterine activity
-Inadequate pelvis
-Fetal malpresentation or macrosomia
-AMA
-Maternal medical issues: DM, HTN, obesity
-Prolonged rupture of membranes
-Chorioamnionitis
-Short maternal stature
-High station at complete dilation

Signs & Symptoms
-Labor not following the norms of the Friedman curve (although these values are now debated)
-Uterine contractions < 200 Montevideo units



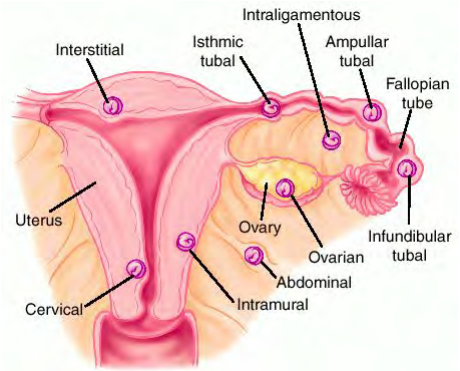
Median and 95th percentile hours for cervical dilation dilation during labor by parity

Cervical dilation (cm)	Parity 0	Parity 1
4 to 5	1.3 (6.4)	1.4 (7.3)
5 to 6	0.8 (3.2)	0.8 (3.4)
6 to 7	0.6 (2.2)	0.5 (1.9)
7 to 8	0.5 (1.6)	0.4 (1.3)
8 to 9	0.5 (1.4)	0.3 (1.0)
9 to 10	0.5 (1.8)	0.3 (0.9)
Second stage with epidural analgesia	1.1 (3.6)	0.4 (2.0)
Second stage without epidural analgesia	0.6 (2.8)	0.2 (1.3)

Management
-Administer oxytocin and monitor for 4-6 hours before considering operative delivery
-Intervention not indicated as long as labor is progressing and fetal HR reassuring

Ectopic Pregnancy

-Most occur in the fallopian tube
-Others are cornual (interstitial), cervical, fimbrial, ovarian, abdominal
-Rarely heterotrophic (intrauterine and ectopic at the same time)
-The leading cause of pregnancy-related deaths in the 1st trimester



Risk Factors
-High: tubal obstruction or injury (PID, tubal ligation), previous ectopic, DES use, current IUD use
-Moderate: infertility, smoking, older age, non-white ethnicities, previous cervicitis

Signs & Symptoms
-Abdominal or pelvic pain
-Amenorrhea or vaginal bleeding
-Usual pregnancy symptoms
-Shoulder pain from blood pooling under diaphragm
-Urge to defecate from blood pooling in cul-de-sac
-Orthostatic BP
-Fever
-Rebound tenderness
-Adnexal pain on bimanual exam
-Cervical motion tenderness

Workup
-Quantitative serum hCG
-Transvaginal US to examine uterine contents: diagnostic if true gestational sac, yolk sac, or embryo is detected inside or outside of the uterus (should be able to visualize if hCG > 1500 which is the limit of US detection)

Management
-If hCG is < 1500 and US is nondiagnostic, need to repeat US and hCG in 3 days or when hCG level reaches US limit
-Surgical if unable to comply with nonsurgical management, ruptured, or hCG > 5000: best outcome with salpingostomy, but will need salpingectomy if ruptured
-Medical management is the treatment of choice for women who are hemodynamically stable with hCG < 5000 and tubal size < 3-4 cm: methotrexate IM followed by serial hCG measurements
-Expectant management only for asymptomatic women with small tubal pregnancy and low hCG levels who are willing to accept the risk of rupture or hemorrhage

Fetal Distress

Causes
-Cord compression
-Placental abruption
-Cord prolapse
-Maternal medication
-Rapid descent of fetal head

Prevention
-Continuous fetal HR monitoring vs intermittent auscultation during labor: no evidence one is better than the other
-High risk women should have continuous fetal monitoring during labor

Signs & Symptoms
-Prolonged abnormalities on fetal HR monitoring

Workup
-Outpatient: NST
-Inpatient: fetal scalp stimulation (FHR acceleration in response is reassuring), fetal ST analysis, fetal scalp blood sampling

Management
-Correct underlying abnormality
-Outpatient: monitor with repeat NSTs
-Improve fetal oxygenation
-Rapid operative intervention if needed

Gestational Diabetes

-Carb intolerance induced by human placental lactogen
-Occurs in 3-5% of all pregnancies
-Classification via White's classification (A1 = nutritional controlled; A2 = insulin requiring

Screening
-Oral glucose tolerance test administered at 28 weeks, when HPL begins to have most effect
-Positive results followed up with 3 hour glucose tolerance test

Management
-Diet + exercise, insulin if needed (preferred over orals)
-BG monitoring 4x daily, with goal FBG < 95 and 1 hour postprandial BG < 130
-Early NSTs with amniotic fluid index for fetal monitoring of insulin-requiring mothers d/t higher rates of placental insufficiency, with biweekly NSTs after 32 weeks for type A2
-Single 3rd trimester US to screen for macrosomia
-Deliver by 40 weeks or earlier if fetus is nearing 8.8 lb
-Rescreen mother at 6 weeks postpartum for glucose intolerance

Prognosis
-Increased risk of having DM postpartum, as well as preeclampsia, bacterial infection, macrosomia, neonatal complications, polyhydramnios, preterm labor, and ketoacidosis
-Child will be predisposed to developing DM later in life

Intrauterine Growth Restriction

-Fetal growth < 10%ile for gestational age and gender
-Multiples share the same growth curve as singletons up to 22-24 weeks

Causes
-Congenital malformations
-Chromosomal abnormalities
-Damage during organogenesis
-Infection: rubella, CMV
-Placenta previa
-Placental infarction or single umbilical artery
-Small placenta
-Multiple gestation

Risk Factors
-Chronic maternal vascular disease
-Smoking
-Fetal abnormalities
-Poor maternal weight gain or malnutrition
-Vaginal bleeding during pregnancy
-Low pre-pregnancy weight
-Prior fetal growth restriction
-Prior stillbirth
-Alcohol, cocaine, or heroin use
-Elevated AFP during 2nd trimester screen

Screening
-Fundal height measurement

Signs & Symptoms
-Fundal height consecutively < 2 than expected

Workup
-US to evaluate fetal growth and %ile, with Doppler of umbilical cord to assess blood flow
-Symmetrically small growth may just indicate small baby
-Asymmetrically small growth indicates placental insufficiency (brain will be larger than body)
-NST and biophysical profile
-Fetal karyotyping if polyhydramnios present

Management
-Delivery with maturity or by 37 weeks if evidence of compromise or poor growth

Prognosis
-High infant mortality within first 2 years of life
-Risk of intellectual deficits

Molar Pregnancy (Hydatiform Mole) and Gestational Trophoblastic Disease

-Occurs when an extra set of paternal chromosomes is incorporated into a fertilized egg, transforming the placenta into a growing mass of cysts
-A complete molar pregnancy means there is no embryo or normal placental tissue
-A partial molar pregnancy means there is an abnormal nonviable embryo and possible some normal placental tissue
-Can coexist with a viable fetus

Risk Factors
-Extremes of age
-Prior molar pregnancy

Signs & Symptoms
-Vaginal bleeding
-Enlarged uterus excessive for gestational age
-Pelvic pressure or pain
-Theca lutein cysts
-Anemia
-Hyperemesis gravidarum
-Hyperthyroidism
-Preeclampsia before 20 weeks' gestation
-Vaginal passage of hydropic vesicles

Workup
-Quantitative hCG: will be higher than expected
-Pelvic US showing "snowstorm pattern"

Management
-Suction uterine curettage with testing of tissue by a pathologist
-Weekly hCG levels until normal
-May need prophylactic chemotherapy for high risk disease

Prognosis
-Risk of developing malignancy with uterine invasion or metastatic disease if tissue is retained: persistent or invasive gestational trophoblastic neoplasia, choriocarcinoma, or placental site trophoblastic tumor

Pregnancy-Induced Hypertension

Signs & Symptoms
-Defined as BP > 140/90 after 20th week of pregnancy WITHOUT proteinuria in a previously normotensive woman
-No symptoms of preeclampsia, such as HA, vision changes, RUQ pain

Workup
-Differentiate from preeclampsia: urine dip for protein may not be 100% reliable, so need to do 24 hour urine or spot urine:creatinine
-Assess fetal wellbeing with biophysical profile or NST with amniotic fluid estimation

Management
-Weekly prenatal visits
-Delivery at 37-39 weeks for frequent mildly elevated BPs, or earlier for severe HTN
-Antihypertensives if severe to reduce risk of maternal stroke

Prognosis
-Generally favorable, not associated with morbidity and mortality of preeclampsia, however women with gestational HTN are at increased risk of developing preeclampsia
-Associated with development of HTN later in life

Preeclampsia (Toxemia) & Eclampsia

-Pregnancy-induced HTN with significant proteinuria ± pathologic edema
-Can also have preeclampsia superimposed on chronic HTN

Risk Factors
-Multiple gestation
-Obesity
-Chromosomal or congenital fetal anomalies
-Pregestational DM
-First pregnancy
-Age < 20 or > 40

Screening
-Urine dip for symptomatic women

Signs & Symptoms
-Lies on a spectrum from mild & asymptomatic to severe
-Only appears after 20 weeks, with majority of cases after 28 weeks
-Irritability
-Hyperreflexia
-End-organ damage: frontal HA, photophobia and visual changes, epigastric pain, oliguria, nondependent edema
-Eclampsia: all s/s of preeclampsia + seizures due to neurologic irritability
-HELLP syndrome: preeclampsia + signs of hemolysis, elevated liver enzymes, and low platelets

Differential
-Exacerbation of underlying renal disease
-Acute fatty liver of pregnancy
-TTP/HUS
-Exacerbation of lupus

Workup
-24 hour urine
-CBC
-CMP
-Uric acid
-Coags: PT, aPTT
-NST
-Diagnose with BP > 140/90 + proteinuria > 0.3 g in a 24 hour urine specimen

Management
-Deliver if severe preeclampsia or eclampsia
-Expectant management with frequent monitoring with delivery at 37 weeks if mild
-Seizure prophylaxis with mag sulfate if severe
-Labetalol or hydralazine only for BPs > 150/100 to reduce risk of stroke

The presence of one or more of the following criteria upstages preeclampsia from mild to severe

Symptoms of central nervous system dysfunction:
Visual disturbance (photopsia, scotomata, cortical blindness, retinal vasospasm)
Severe headache (ie, incapacitating, "the worst headache I've ever had") or headache that persists and progresses despite analgesic therapy
Altered mental status
Symptoms of liver capsule distention:
Right upper quadrant or epigastric pain
Nausea, vomiting
Hepatocellular injury:
Serum transaminase concentration ≥ twice normal
Severe blood pressure elevation:
Systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥110 mm Hg on two occasions at least six hours apart
Thrombocytopenia:
<100,000 platelets/microL
Proteinuria:
≥5 grams in 24 hours
Oliguria <500 mL in 24 hours
Fetal growth restriction
Pulmonary edema or cyanosis

Multiple Gestation

-More common developed countries secondary to use of fertility drugs
-Natural incidence of triplet pregnancy is 1/6000 births

Screening
-First trimester US

Risks of multiple gestation
-Increased preterm delivery
-Intrauterine growth restriction
-Discordant growth
-Twin-twin transfusion
-Increased risk of cerebral palsy
→ Higher infant mortality rates than in singleton pregnancies

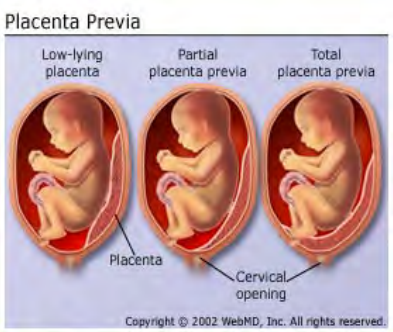
Workup
-Determine chorionicity with US

Management
-Weight gain for twins should be 35-45 lbs
-No intervention proven to reduce risk of preterm labor
-Frequent US to follow fetal growth
-Weekly NSTs beginning at 32 weeks for triplets
-Suggested delivery for twins is 36-37 weeks, 35 weeks for triplets

Prognosis
-Single fetal death after 20 weeks gestation occurs in 5% of twin pregnancies
-Average duration of gestation for twins is 35 weeks, 32 weeks for triplets

Placenta Previa

-When placenta implants abnormally in the lower uterine segment → partial or total blockage of cervical os



Risk Factors
-Multiparity
-AMA
-Asian
-Prior placenta previa
-Smoking
-H/o C-section
-Multiple gestation

Screening
-Usually detected in 1st or 2nd trimester US

Signs & Symptoms
-Painless vaginal bleeding

Workup
-Avoid pelvic exam which can rupture the placenta
-Transvaginal US to assess placental location

Management
-Total placenta previa → refer to high risk OB
-Marginal previa → f/u with serial US, avoid cervical US and sex, activity restrictions, deliver via C-section at 36-37 weeks
-Active bleeding → hospitalization with close monitoring, may need emergency C-section

Postpartum Hemorrhage

-Defined as blood loss > 1000 mL (or > 1500 for C-section)
-Avg vaginal delivery EBL is 500 mL (or 1000 mL if section)
-Occurs in 5% of deliveries
-Can be early (within 24 hours of delivery) or late (up to 6 weeks after delivery)

Causes
-Uterine atony (causes 70% of cases)
-Retained placental tissue
-Infection
-Blood vessel damage during C-section
-Congenital coagulopathy

Risk Factors
-Chorioamnionitis
-Uterine distension
-Prolonged or induced labor
-Use of mag sulfate
-General anesthesia
-Multiparity
-Previous hemorrhage
-Placenta previa or abruption
-Operative delivery

Prevention
-Active management of 3rd stage of labor
-Use of oxytocin after delivery of the anterior shoulder

Signs & Symptoms
-Signs of shock and hypovolemia
-Delivery of placenta > 30 min after infant
-Uterine atony
-Signs of uterine rupture: hypotension greater than expected for EBL, increasing abdominal girth

Workup
-Check for retained placenta: inspect delivered placenta for missing parts, explore uterus
-Look for traumatic cause of hemorrhage: tear, hematoma, uterine inversion
-Coagulopathy workup: PT/aPTT, fibrinogen, antithrombin III

Management
-Treat underlying cause
-Uterine atony → uterine massage, oxygen, large-bore IV access, oxytocin, methylergonovine
-Uterine inversion → manual reduction of uterus, laparotomy
-Uterine rupture → surgical intervention
-Embolization of uterine or hypogastric arteries
-Hysterectomy is last resort

Preterm Labor

-Regular, painful uterine contractions with cervical dilation or effacement before 37 weeks

Possible Etiologies
-Dental disease
-Bacterial vaginosis
-Inflammatory response

Risk Factors
-Smoking
-Black
-Extremes of age
-Low SES, poor housing, or other social stress
-Multiple gestation
-Intergestational period < 6 mos
-H/o cervical surgery or short cervix
-Infection: bacteriuria or UTI, BV

Prevention
-Treating infections has not been shown to improve outcomes
-ID of high risk women with early care and enhanced prenatal services also has failed to improve outcomes
-Can follow women with h/o preterm labor with frequent US to assess cervical length
-ACOG recommends offering progesterone to women with cervical length < 15 mm or with h/o preterm delivery
-Cervical cerclage or pessary an option

Signs & Symptoms
-Contractions: back pain, abdominal pain, cramping
-Diarrhea
-Leaking fluid

Workup
-Check fetal fibronectin, has good NEGATIVE predictive value for assessing risk of delivering in next 7-14 days (can be inaccurate with recent cervical disruption like sex or TVUS)
-US measurement of cervical length; preterm labor likely if < 20 mm
-Evaluation of fetal lung maturity (amniotic fluid specimen): lecithin/sphingomyelin ratio, foam stability index, phosphatidylglycerol, or fluorescence polarization

Management
-Progesterone: maintains cervical integrity, opposes oxytocin, and is anti-inflammatory
-Tocolytics (anti-contractants like terbultaline, mag sulfate, CCBs, indomethacin): no evidence that they improve outcomes but they do buy time to administer steroids or transport to NICU facility
-Steroids to mature fetal lungs
-GBS prophylaxis if needed or if culture not recently done
-Bed rest, pelvic rest, and hydration have no evidence to back them up
-Avoid sex and strenuous physical activity
-Outpatient follow-up feasible for reliable patients

Premature Rupture of Membranes (PROM) & Preterm Premature Rupture of Membrans (PPROM)

PROM
-Rupture of membranes at full term but before onset of labor (normally amniotic sac ruptures well into labor)
-Occurs in 10% of normal pregnancies

PPROM
-Refers to rupture of membranes before 37 weeks
-Usually caused by maternal infection
-Risk factors: intra-amniotic infection, prior h/o PPROM, lower SES, teen mom, smoker, h/o STD, h/o cervical cerclage, multiple gestation, polyhydramnios

Signs & Symptoms
-Feeling "leaking urine" or increased vaginal secretions
-Sx of chorioamnionitis: odor, fundal tenderness, low grade fever, fetal tachycardia

Workup
-Visual exam for pooling of amniotic fluid in vagina with test for ferning of sampled fluid
-GC/*Chlamydia* testing

Management
-If term, good evidence that labor should be induced after this in order to prevent NICU placement; GBS prophylaxis if > 18 hours since rupture, or with colonization or fever
-If preterm, need inpatient monitoring, treatment of infection if present, deliver if > 34 weeks or with fetal distress, otherwise expectant management with steroids if needed for fetal lung maturation

Rh Incompatibility

-Maternal immunization can occur as a result of transplacental fetomaternal hemorrhage or blood transfusion with Rh+ blood

Screening
-Maternal Rh status and antibody screening done at first prenatal visit and at delivery

Prevention
-Rhogam given to all Rh- mothers at 28 weeks, again just after delivery if neonate is determined to be Rh+, and anytime during pregnancy when there is risk of fetomaternal hemorrhage

Signs & Symptoms
-Rh incompatibility causes a spectrum of disease from hyperbilirubinemia to hydrops fetalis

Differential
-RBC membrane defects: hereditary spherocytosis
-RBC enzyme defects: G6PD deficiency, pyruvate kinase deficiency
-Gilbert's syndrome

Workup
-Maternal and infant blood T&S
-Coombs test
-Infant peripheral smear
-Antibody titers during pregnancy for mothers with known Rh sensitization and Rh+ fetus

Management
-Emergent neonatal transfusion at delivery for infants with signs of shock
-Later transfusions for symptomatic anemia
-EPO and iron for mild anemia

OTHER REPRODUCTIVE SYSTEM TOPICS

Menopause

Climacteric = a phase in women transitioning from a reproductive state to a non-reproductive state; includes perimenopause as well as a time before and after

Perimenopause = ~4 years before menopause when cycles become irregular and there are increased climacteric symptoms

Menopause = time during cessation of menses for 1 year; can be natural due to loss of ovarian estrogen activity, induced via surgery or radiation, temporary due to diet or GnRH therapy, premature if before age 40, or late if after age 55

Postmenopause = time following cessation of menses for 1 year

-Average age of natural menopause in US is 51.4 years

Signs & Symptoms
-Begin up to several years before cessation of menses and can last for 2-9 years after menopause
-Dry hair and mouth, facial hirsutism
-Menstrual irregularlity, postcoital bleeding, intermenstrual spotting
-Loss of adiposity and collagen in vulva, loss of protective covering of clitoris, thinner vaginal surface, vaginal dryness and atrophy, genital itching or burning, dyspareunia, pale or shiny vaginal epithelium with loss of rugae, spare pubic hair, introital stenosis, fusion of labia minora, pelvic organ prolapse, vulvar dermatoses, stress incontinence, urinary frequency, decreased libido
-Hot flashes, vasomotor instability, sleep and mood disruptions
-Reduced breast size and loss of ligamentous supports

Workup
-Diagnosis is usually clinical
-FSH > 35 is diagnostic (FSH is ↑in response to low estrogen)
-Check TSH if there are symptoms of hyperthyroidism
-For women under 45, do oligo/amenorrhea workup: hCG, prolactin, TSH, FSH
-Women under 40 need comple premature ovarian failure workup

Management
-Dressing in layers, avoiding food triggers, regular exercise
-Estrogen replacement therapy for moderate to severe symptoms of vasomotor instability: use lowest dose for shortest amount of time possible
-Vaginal moisturizers and lubricants for vaginal atrophy symptoms, may need vaginal estrogen
-SSRIs
-Biofeedback
-Acupuncture

Pelvic Organ Prolapse

-Herniation of pelvic organs to or beyond the vaginal walls
-Can be a cystocele, rectocele, enterocele, uterus, vaginal vault, fibroid
-Classified via the Pelvic Organ Prolapse Quantitation system

Risk Factors
-Multiparity
-Advancing age
-Obesity
-Hysterectomy
-Chronic constipation
-Heavy lifting

Signs & Symptoms
-Feeling a bulge or something falling out of vagina
-Urinary, defecatory, or sexual dysfunction

Workup
-Pelvic and rectovaginal exam to determine location and source of prolapse
-Neuromuscular exam: bulbocavernosus and anocutaneous reflexes, sharp/dull touch, strength

Management
-No treatment needed if asymptomatic
-Symptomatic prolapse may be treated conservatively (pessaries or Kegels) or surgically

Pelvic Inflammatory Disease

-Inflammation of the uterus, fallopian tubes, and/or ovaries, and possibly surrounding pelvic organs
-Usually polymicrobial, with STIs + endogenous organisms

Risk Factors
-Multiple sex partners
-Douching
-Smoking

Signs & Symptoms
-Pelvic or abdominal pain
-Painful defecation
-Abnormal vaginal bleeding
-Dyspareunia
-Uterine, adnexal, or cervical motion tenderness
-RUQ pain (from perihepatitis)
-Signs of STI infection

Workup
-Testing for GC, *Chlamydia*, HIV, hep B, syphilis
-Cervical cultures
-hCG
-Pelvic US if concern for abscess
-CBC
-UA

Management
-If no other cause of pelvic or abdominal pain can be found in a sexually active woman at risk for STIs, always treat for PID
-Begin antibiotic before cultures come back
-Admit for inpatient management if there is pregnancy, nonresponse to oral antibiotics, inability to take PO, severe illness, or tubo-ovarian abscess
-Outpatient treatment of mild-mod PID: ceftriaxone IM + doxycycline
-Inpatient treatment of severe or complicated PID: IV cefoxitin + PO doxycycline
-Treat partners

Prognosis
-Risk for infertility increases with each episode

Infertility

-Failure to achieve pregnancy within one year of frequent, unprotected sex if < 35 or within 6 months if > 35

Etiologies
-Male issues: 1° hypogonadism (androgen insensitivity, cryptorchidism, meds, varicocele, genetic defect), 2° hypogonadism (androgen excess, infiltrative disorder, meds, pituitary adenoma)
-Female issues: ovulatory dysfunction, tubal damage, endometriosis, cervical factor

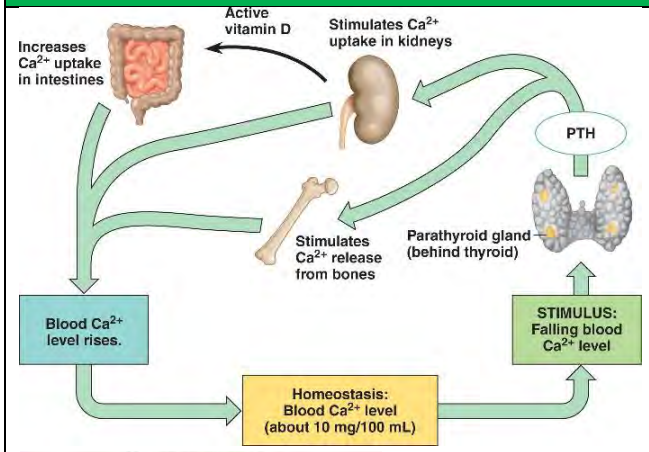
Signs & Symptoms
-Men: genital infection, hernia, absence of vas deferens, signs of androgen deficiency, testicular mass, varicocele
-Women: breast formation, galactorrhea, genitalia, signs of hyperandrogenism

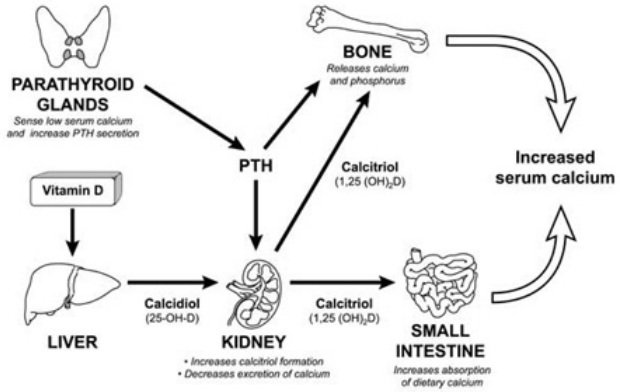
Workup
-CBC and CMP for both partners
-GC/*Chlamydia*
-UA
-Men: consider post-ejaculatory UA for retrograde ejaculation, scrotal US, FSH and testosterone levels, sperm studies, transrectal US
-Women: consider FSH, prolactin, TSH levels, antral follicular count via US, hysterosalpingography, pelvic US, hysteroscopy, laparoscopy

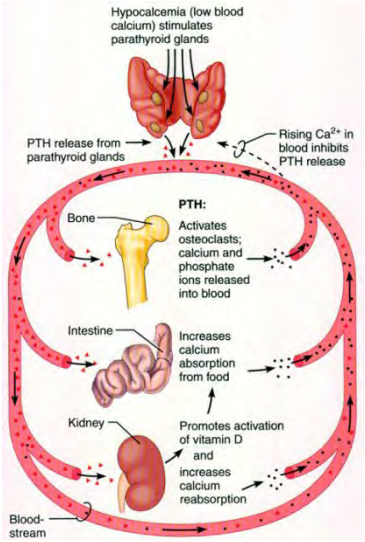
Management
-Treat underlying problem
-Bromocriptine for hyperprolactinemia
-Treat ED
-Varicocele repair
-Referral to fertility specialist for semen abnormality
-Ovulatory dysfunction treatment: ovulation-inducing meds or hormone injections
-Tubal repair
-Laparoscopic ablation of endometriosis
-Fertility monitoring: timed intercourse with fertility awareness methods will result in pregnancy in 90% of couples
-For unexplained infertility, 3-4 cycles of clomiphene followed by intrauterine insemination is recommended
-IVF results in the highest per cycle pregnancy rate in the shortest time interval but is most costly and has a high rate of high order multiple pregnancy

Prognosis
-Overall likelihood of successful treatment is 50%

ENDOCRINE SYSTEM
DISEASES OF THE THYROID GLAND
Hyperparathyroidism







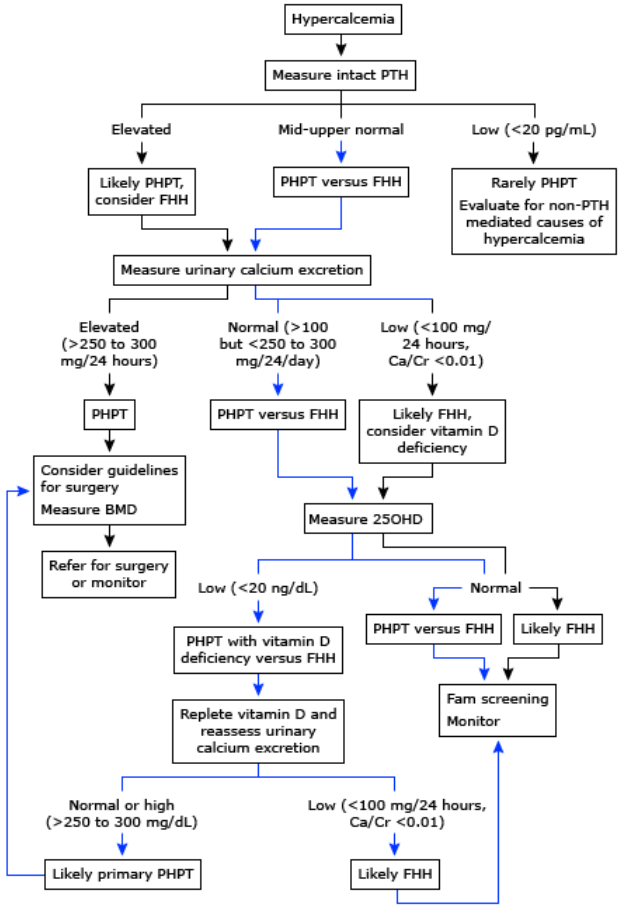
Etiologies
-Primary hyperparathyroidsm: adenoma or other hyperplasia
-Secondary hyperparathyroidism as a response to low Ca levels (vit D deficiency → impaired abs of Ca, or CKD → failure to activate vit D)

Signs & symptoms
-Incidental hypercalcemia finding on blood test
-Low bone mineral density screen
-Weakness and fatigue, depression, bone pain, myalgias, decreased appetite, n/v, constipation, polyuria, polydipsia, cognitive impairment, kidney stones and osteoporosis
-Rickets
-Osteomalacia

Differential
-Malignancy

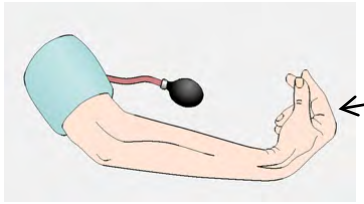
Management
-Primary hyperparathyroidism → surgical parathyroidectomy if symptomatic, otherwise monitor Ca and Cr annually and DEXA every 2 years
-Calcium mimics if unable to undergo surgery

Workup



Hypoparathyroidism

Etiologies
-Genetic
-Parathyroidectomy
-Autoimmune
-Hemochromatosis
-Mg deficiency



Signs & symptoms
-Paresthesias in the mouth, hands, and feet
-Muscle cramps in the hands and feet
-Fatigue
-Headaches
-Bone pain
-Insomnia
-Abdominal pain
-Chvostek's sign
-Trosseau's sign
-Seizures
-Arrhythmias
-Respiratory failure

Ask the patient to relax his facial nerves. Next, stand directly in front of him and tap the facial nerve either just anterior to the earlobe or below the zygomatic arch and the corner of the mouth. A positive response varies from twitching of the lip at the corner of the mouth to spasm of all facial muscles, depending on the severity of hypocalcaemia.

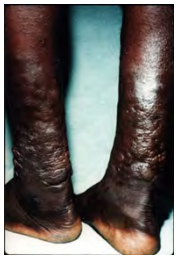
Differential
-Pseudohypoparathyroidism
-Vit D deficiency
-Meds
-Kidney disease
-Malabsorption

Workup
-Labs: low Ca, PTH, albumin to correct

Management
-IV Ca gluconate if severe
-PTH supplementation

Hyperthyroidism

Signs & symptoms: weight loss, hyperphagia, heat intolerance, increased sweating, frequent stools, oily hair or skin, exercise intolerance due to heart changes, proximal muscle weakness, nervousness, irritability, sleep disturbance, tremor, palpitations, decreased menstrual flow, onycholysis, exophthalmos, lid lag, goiter, thyroid bruits, brisk DTRs, muscle cramps, osteoporosis, pretibial myxedema



Differential
-Grave's disease
-Toxic multinodular goiter
-Single toxic nodule (Plummer disease)
-Subacute (de Quervain) thyroiditis: a result of viral infection, eventually leads to hypothyroid
-Initial destructive phase of Hashimoto thyroiditis
-Postpartum thyroiditis
-Amiodarone-induced thyrotoxicosis
-Rare: gestational trophoblastic disease, increased iodine intake, thyrotoxicosis factitia (consumption of TH), ovarian tumor secreting TH, pituitary tumor secreting TSH, thyroid carcinoma

Workup
-Depressed TSH with elevated free T4
-↑ LFTs and Ca
-↓ Lipids

Management
-Referral for endocrine workup and imaging, possible ablation
-Antiadrenergics: propranolol or diltiazem
-Antithyroids: propylthiouracil, methimazole
-Untreated thyrotoxicosis can progress to a thyroid storm that can be fatal!

Hypothyroidism

-TSH screen recommended for all elderly with depression and all elderly entering long-term care

Signs & symptoms: cold intolerance, fatigue, heavy menstrual bleeding, weight gain, dry skin, constipation, bradycardia, delayed DTRs, hoarseness, coarse hair, hair loss, myalgia, cognitive impairment, depression, decreased concentration, decreased hearing, periorbital or facial edema, non-pitting ankle edema, ± goiter

Differential
-Post-Hashimoto thyroiditis (most common cause)
-Congenital hypothyroidism
-Post thyroid ablation or neck radiation
-Transient causes: post-postpartum thyroiditis, post-de Quervain thyroiditis
-Goiterous hypothyroidism: due to impairment of TH synthesis (lack of iodine or genetic)
-Lithium or other drug therapy
-Central hypothyroidism: not enough TSH being made

Workup
-Elevated TSH
-↑ LDL, TG, LFTs, CK

Initial therapy
-Thyroxine with recheck of TSH in 6 weeks
-For patients still having symptoms 2-3 weeks into therapy, recheck TSH and free T4

Maintenance therapy
-Once TSH reaches reference range, recheck annually
-Dose needs to be increased in pregnancy and decreased with aging

Myxedema Coma

-Severe hypothyroidism

Causes
-Severe longstanding hypothyroidism
-Adrenal insufficiency
-Precipitation by acute infection, MI, cold, or sedatives

Signs & Symptoms
-AMS
-Hypothermia
-Hypotension, bradycardia, hyponatremia, hypoglycemia, hypoventilation
-Edema of hands and face
-Thickened nares, swollen lips, or enlarged tongue

Workup
-TSH and free T4
-Cortisol level

Management
-If clinical suspicion, begin treatment without waiting for labs
-Stress dose steroids until adrenal insufficiency has been excluded
-Administer loading dose T4 followed by daily dose
-Also give T3 (more rapid onset) until pt is stabilized with clinical improvement
-ICU admission
-Supportive care: passive rewarming (don't use active rewarming device), mechanical ventilation for respiratory distress, fluid replacement, correction of chemistry abnormalities

Prognosis
-Mortality 40%

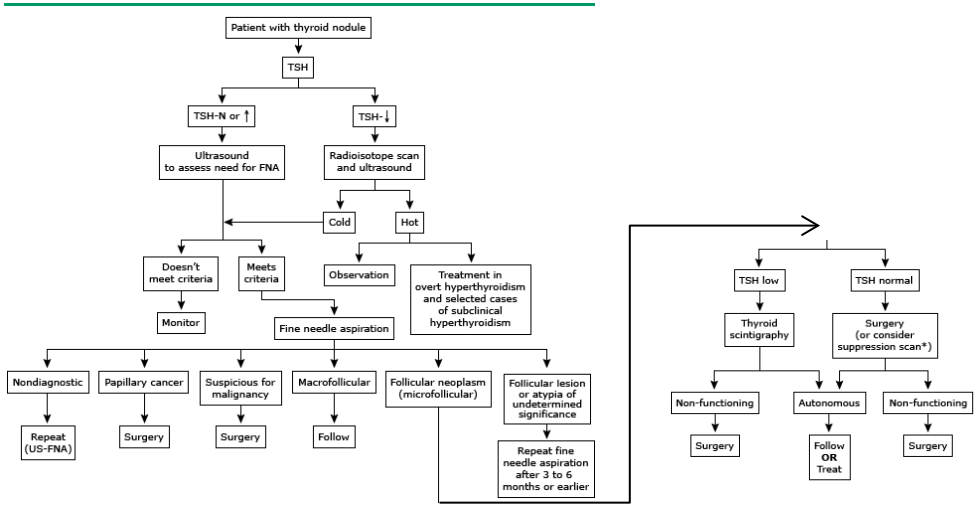
Thyroid Neoplasms and Thyroid Nodules

-More common in women
-Not associated with hypo or hyperthyroidism
-Characteristics suggesting malignancy: age < 20 or > 70, solid or complex, cold nodules, single nodule, nodule that grows with TH replacement, hoarseness or obstruction symptoms, hx of neck or head radiation

Workup
-Check TSH for all patients
-Can check autoantibodies

Management
-Follow low-risk nodules every 6 months with palpation and US
-Benign nodules may disappear over time
Surgical removal if concern for malignancy
-Refer for neck US to assess for size and shape
-Low TSH → radionuclide scan to check hot/coldness of nodule
-Refer for FNA if US results show risk of malignancy
-Refer for resection if FNA cytology is suspicious

Prognosis
-10% of palpable nodules will be malignant
-Surgical complications: recurrent laryngeal nerve damage, parathyroid damage



Benign Thyroid Neoplasms	Malignant Thyroid Neoplasms	
Follicular cell adenoma	**Papillary adenocarcinoma** -Most common type of thyroid cancer **Follicular adenocarcinoma** -Diagnosis usually occurs during evaluation of a cold thyroid nodule -Treatment is through radioactive iodine ablation with hormone replacement to suppress TSH **Hurthle cell thyroid cancer**	**Medullary Adenocarcinoma** -Arises from C-cells of the thyroid -Age > 40 -Associated with MEN type 2 -Regional lymph node involvement with mets to the lung, bone, and liver -Evaluate serum calcitonin, CEA, Ca, and plasma fractionated metanephrines -Very deadly

Thyroiditis

Subacute Thyroiditis

-AKA de Quervain's thyroiditis

Causes
-Radioiodine therapy
-Viral or infectious cause
-Trauma

Signs & Symptoms
-Painful thyroid, neck pain, or goiter

Workup
-TSH, free T3 & T4, ESR
-Radioiodine imaging (uptake will be low)

Management
-Pain control with NSAIDs or prednisone if refractory
-Monitor thyroid panel every 2-8 weeks to confirm resolution of thyroid imbalance and normalization of function
-β-blockers for palpitations

Painless Thyroiditis

Causes
-Usually autoimmune: Hashimoto's (aka chronic lymphocytic thyroiditis) or a variant, can be postpartum
-From exposure to drugs like Li
-Precipitating factors: infection, stress, sex steroids, pregnancy, iodine intake, and radiation exposure

Signs & Symptoms
-Painless thyroiditis; typically involves 2-6 weeks of hyperthyroidism followed by transient hypothyroidism, then euthyroid
-Postpartum presentation can occur up to 1 year after giving birth and has a longer course than typical painless thyroiditis
-Hashimoto's typically causes a goiter

Workup
-TSH, free T3 & T4
-Differentiate from Grave's disease with technetium scan or radioiodine uptake scan

Management
-Monitor thyroid panel every 2-8 weeks to confirm resolution of thyroid imbalance and normalization of function
-β-blockers for palpitations
-Hyperthyroid phase is generally mildly symptomatic and does not require treatment
-Treatment of symptomatic hypothyroid phase with thyroxine

Prognosis
-May have recurrent episodes
-20% develop permanent hypothyroidism

Acute (Suppurative) Thyroiditis

-Rare!

Cause
-Suppurative bacteria

Signs & Symptoms
-Painful, red, tender thyroid

Management
-Antibiotics
-Surgical drainage if needed

Riedel's (Fibrous) Thyroiditis

Signs & Symptoms
-Painful, stony, hard thyroid
-Dysphagia
-Dyspnea
-Hoarseness

Management
-Short-term steroids
-Long-term tamoxifen therapy

Thyroid Storm

-Rare, severe form of thyrotoxicosis

Causes
-Stressful illness or surgery
-Thyroid surgery
-Radioactive iodine
-Longstanding hyperthyroidism
-Childbirth

Signs & symptoms
-Delirium
-Tachycardia
-Vomiting, diarrhea, dehydration
-Fever

Workup
-Thyroid panel
-Diagnosis is clinical

Management
-ICU admission
-β-blocker, thionamide to block new hormone synthesis (PTU or methimazole), iodine to block release of TH, iodinated contrast and steroids to block peripheral conversion of T4 → T3

DISEASES OF THE ADRENAL GLANDS

Adrenal Insufficiency

Signs & symptoms
-Chronic fatigue, lack of appetite, unintentional weight loss
-Joint pain
-Abdominal pain, nausea, diarrhea
-CV instability, hypoglycemia in times of stress, hyponatremia, hypotension unresponsive to fluids or pressors

Workup
-Morning cortisol levels: levels > 18 high enough to rule out AI, levels ≤ 3 high enough to rule in AI
-Synthetic ACTH simulation test
-Lastly check the ACTH levels

Management
-If acute crisis, treat with IV dexamethasone and IVF for hypotension
-If chronic, glucocorticoid maintenance therapy is needed
-Primary AI will also need mineralocorticoid replacement
-MedicAlert bracelet and emergency steroid injections
-Stress dose steroids needed for surgeries

	Primary Adrenal Insufficiency (Addison's Disease): occurs when adrenal gland does not respond to ACTH or make adrenal hormones (including aldosterone) due to damage	**Secondary Adrenal Insufficiency:** failure of pituitary to secrete ACTH	**Tertiary Adrenal Insufficiency:** failure of hypothalamus to secrete CRH	**Adrenal Crisis:** acute, life-threatening low levels of cortisol
Etiologies	-Most causes are autoimmune -Infection -Adrenal hemorrhage -Drugs	-Pituitary tumor -Radiation -Surgery -Long-term steroid therapy -Megace (appetite stimulant drug) -Sarcoidosis	-Usually due to suppression of CRH and ACTH by exogenous cortisol use	-Underlying adrenal condition
Signs & symptoms	-Presentation can be slow or abrupt -Skin hyperpigmentation -Salt craving -Hyponatremia -Hyperkalemia -Vitiligo -Pallor -Autoimmune thyroid disease	-Slow onset -No skin hyperpigmentation (because there is no excess ACTH) -Intact RAAS		-Vomiting -Abdominal pain -Weakness -Fever -Confusion -Syncope
Workup	-Labs showing hypoglycemia, hyponatremia, hyperkalemia, low aldosterone, and high renin due to increased renal sodium losses	-Insulin tolerance test -Metyrapone test		-Labs showing low cortisol, low glucose, hyperkalemia, hyponatremia, elevated BUN

Cushing's Syndrome

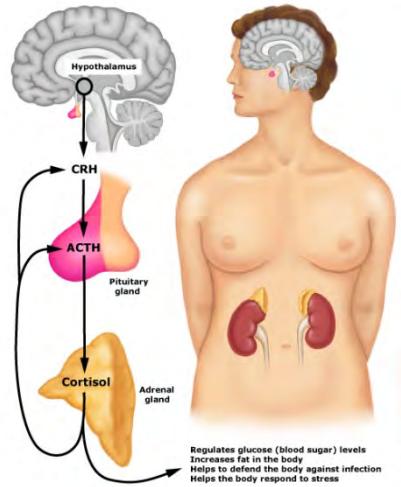
-A general term for hypercortisolism at any level, including adrenal, ectopic, or pituitary source

Etiologies
-Exogenous steroid use
-Excess pituitary ACTH production: includes Cushing's disease (refers specifically to an ACTH-secreting pituitary adenoma)
-Ectopic ACTH production: small cell lung ca, carcinoid tumors, pheochromocytoma, thymoma, pancreatic cell tumors, medullary carcinoma of the thyroid
-Adrenal hypercortisolism: adrenal adenoma or carcinoma or hyperplasia

Signs and symptoms
-Supraclavicular and dorsal fat pads ("buffalo hump")
-Central obesity
-Proximal muscle weakness
-Thinning of the skin, purple striae, spontaneous ecchymoses, skin hyperpigmentation, papular acne
-Osteopenia
-Hypertension
-Early or delayed puberty
-Growth retardation
-Glucose intolerance
-Decreased libido, infertility, amenorrhea
-Nephrolithiasis or polyuria
-Headaches

Workup
-Always remember to check for exogenous glucocorticoid use
-Always do labs first before any imaging to avoid incidentalomas and false negative scans
-Establish presence of cortisol excess: 24 hour urine, dexamethasone suppression test, or saliva cortisol
-Establish ACTH dependence or independence to pinpoint problem to the adrenals vs the pituitary gland

Management
-Surgical resection is first line
-Drugs to block adrenal response: somatostatin analogs, adrenal steroid synthesis inhibitors



Adrenal Neoplasms

-Majority are benign, nonfunctioning incidentalomas
-Imaging characteristics suggesting benignity are homogeneity, smooth border, rapid contrast medium washout
-Imaging characteristics suggesting malignancy are irregular shape, inhomogeneous density, delayed contrast washout, diameter > 4 cm, tumor calcification
-Workup for all incidentalomas: 24 hour urine metanephrines and catecholamines, overnight dexamethasone suppression test, plasma aldosterone-to-plasmin renin activity ratio and plasma K+ if pt is hypertensive
-Surgical resection of incidentalomas associated with pheochromocytoma findings, subclinical Cushing's, > 4 cm, or if suspicious
-Repeat imaging of incidentalomas 3-6 months after discovery, with removal of tumors growing > 1 cm during this time

Benign

Adrenal cortex adenoma
-Cause Cushing's syndrome, primary aldosteronism, virilization, or feminization
-Most are < 4 cm
-Unilateral adrenalectomy if Cushing's or hyperaldosteronism present

Malignant

Adrenal cortex carcinoma
-Rare
-Aggressive
-May cause Cushing's syndrome or virilization or can be nonfunctional
-Present as abdominal mass or incidentally
-Most are > 4 cm
-Half will have advanced disease at initial presentation
-Surgical resection
-Adjuvant mitotane
-Unfavorable prognosis due to presence of micromets in most disease

Pheochromocytoma
-Medullary neuroendocrine tumor
-10% will be malignant
-S/s: HA, swelling, tachycardia, HTN unresponsive to therapy
-Surgical resection

DISEASES OF THE PITUITARY GLAND

Acromegaly (Gigantism)

-Caused by GH-secreting pituitary tumor in adulthood
-If this occurs before puberty, it is called gigantism

Signs & symptoms
-Enlarged soft tissue of the hands and feet
-Teeth splaying, prominent brow
-Hyperhidrosis
-Arthralgias
-Headaches
-Hypogonadal symptoms
-Vision deficits
-Fatigue, weight gain
-Galactorrhea
-CV disease such as HTN, LVH, or cardiomyopathy
-Enlargement of thyroid, liver, kidneys, or prostate

Workup
-Initial screen is IGF-1 test, will be high
-Most sensitive test is ↑ GH after glucose tolerance test
-Labs: abnormal glucose, ↑P and PRL
-Pituitary MRI
-GHRH if MRI is negative

Management
-Surgery
-Somatostatin analogs (inhibit GH)
-GH receptor agonists
-Radiation

Prognosis
-Bony abnormalities usually won't regress

Diabetes Insipidus

Central diabetes insipidus
-Lack of ADH production in the hypothalamus or insufficient release of ADH from the posterior pituitary
-Etiologies: idiopathic, familial, panhypopituitarism, infiltrative diseases, metastatic tumor, trauma or surgery, Wolfram syndrome

Nephrogenic diabetes insipidus
-Kidney is resistant to ADH
-Etiologies: amyloidosis, myeloma, Sjogren's, sickle cell, hypercalcemia, recovery from ATN, Li, foscarnet, methicillin, demeclocycline, colchicine

Signs & symptoms
-5-10 L per day of dilute polyuria
-Polydipsia
-Hypernatremia
-Normal glucose

Differential
-Inpatient: mannitol, post-op diuresis, hyperglycemia, diuretics, overzealous IVF, cured acromegaly
-Outpatient: hyperglycemia, psychogenic polydipsia, osmotic load

Workup
-Direct serum ADH measurement is difficult as it is in extremely low concentration
-Pituitary imaging
-Water deprivation test: restrict water and check Na, urine osmolality, urine output, weight, and orthostatic BPs/HRs every 1-2 hours → + if body wt ↓ by > 5%, serum Na > 145, or > 2 urine osmolalities differ by < 10%
-Differentiate central vs nephrogenic by giving desmopressin (synthetic ADH) → central DI if urine vol ↓ or there is > 50% ↑ in urine osmolality, nephrogenic if there is < 50% change in urine osmolality

Dwarfism

Etiologies
-Achondroplasia: most common, genetic
-Growth hormone deficiency: will see delayed puberty
-Others: congenital dysplasias, Noonan syndrome, Tuner syndrome, osteogenesis imperfecta, hypothyroidism

Workup
-Bone x-rays
-Referral to endocrinology

Management
-Growth or thyroid hormone supplementation
-Distraction osteogenesis

Pituitary Neoplasms

-Differential**:** physiologic pituitary enlargement of pregnancy, sellar cyst or abscess, lymphocytic infiltration of the pituitary
-W/u: MRI, eval of hypothalamic-pituitary axis function

Benign

Pituitary adenoma
-Only tumor that causes hormonal hypersecretion
-May secrete gonadotropins, TSH
-S/s: neuro sx, hormonal abnormalities, incidental
-Resection if causing neuro sx
-Annual monitoring of growth and HPA

Craniopharyngioma

Meningioma

Malignant

Germ cell tumor

Chordoma

Primary CNS lymphoma

Breast or lung mets

DIABETES MELLITUS

Type II Diabetes Mellitus

Prevention
-7% TBW loss, food journals, and visiting dietician regularly better than starting on metformin

Screening
-ADA says do for all patients who are overweight or who have risk factors, and for all patients over age 45 every 3 years
-Peds: universal not recommended by AAP nor ADA, rather screen at risk children with BMI > 85th percentile and 2+ additional risk factors; screen every 3 years
-FBG: prediabetes is 100-125, diabetic is 125+
-Oral glucose tolerance test: prediabetes is 140-199, diabetes is 200+
-Random glucose test: diabetic if 200+
-Hb A1c: diabetic if > 6.5%, want DM to be < 7%, check this every 3 mo if it is not at goal or 1-2 times per year for patients at goal

Signs & symptoms
-Blurred vision
-Fatigue
-Polyuria & polydipsia
-Weight loss
-Hirsutism (DM2)
-Acanthosis nigrans (DM2)
-DKA (more likely DM1)
-In kids often occurs at onset of puberty as this causes increased insulin resistance

Workup
-C-peptide: ↓ in DM1, normal or hi in DM2
-Fructosamine (glycated albumin): gives info about short-term BG in last few weeks
-Differentiate from DM1 by presence of excess weight, acanthosis nigricans, HTN, dyslipidemia, PCOS, FH, ethnic group risk factors

Prevention of complications
-Diabetic retinopathy, cataracts, glaucoma → annual eye exams
-Nephropathy and ESRD → BP and BG control, ACEI, annual urine microalbumin (should be < 30)
-DM is a cardiac risk equivalent → cholesterol control (LDL < 100, HDL > 50, TG < 150), baby aspirin (ADA rec)
-Neuropathy is the most common complication: cardiac denervation, gastroparesis, neurogenic bladder, ED, etc → annual diabetic neuropathy screens

Glycemic control
-If A1c 6.5-7.5 → start on metformin unless contraindicated
-If A1c is 7.6-9% → start on metformin + additional oral
-If A1c is > 9% → start on insulin if symptomatic or metformin + 1-2 other orals if asymptomatic
-Initiate insulin if 3+ orals are needed to control BG or if A1c remains > 8.5 even after dual therapy
-Rebound (Somogyi) hyperglycemia occurs in response to hypoglycemia
-Dawn phenomenon is morning hyperglycemia as a result glucagon response to waning insulin levels around 3-5 am → change insulin or move peak to a more physiologic time

Oral Pharmacologic Treatment: most are not for DM1, they have no insulin to secrete!

Sulfonylureas
-1st gen: chlorpropamide, tolazamide, tolbutamide → more AEs
-2nd gen: glipizide, glyburide, glimepiride → don't work better but have less AEs and less drug interactions
-Hug pancreas all day long = tend to burn out pancreas after 3-5 years of use
-Hypoglycemia risk
-Start on low dose and adjust over 3-4 weeks
-Begin combination therapy with another agent when approaching max dose

Biguanides: metformin
-Decrease hepatic glucose output 1st and then increases uptake by fat and muscle
-May help with weight loss
-Every DM2 should be put on this at time of diagnosis
-Must monitor creatinine @ baseline and annually to screen for kidney disease (from the DM, not from the metformin itself), stop using if Cr > 1.4 in women and 1.5 in men → risk of lactic acidosis
-Contraindicated in kidney or liver disease, elderly, CHF, alcohol abuse or binge drinking, IV contrast

GLP-1 agonists: exenatide pen, long-acting exenatide, liraglutide
-Aid native GLP-1 naturally released by the gut to help insulin secretion while reducing glucagon
-Only respond when BG is high!
-Need to be paired with a DPP-4 inhibitor like sitagliptin, saxagliptin, or linagliptin
-Need to decrease sulfonylurea dose to avoid hypoglycemia
-Contraindications: pancreatitis, gastroparesis, GI issues, CrCl < 30, thyroid cancer
-Many drug interactions, must take 1 hour prior

Meglitinides: repaglinide, nateglinide
-Hug pancreas with one quick squeeze = take with a meal
-Hypoglycemia risk
-May cause weight gain = less favorable
-No renal or liver patients
-Pregnancy C

Glitazones: pioglitazone, rosiglitazone
-Increases glucose uptake in fat and muscle 1st, then decreases hepatic glucose output
-Current FDA restrictions due to risk of bladder ca, fracture risk, CV side effects = try other meds first
-Contraindicated in liver disease, heart failure
-Pregnancy C

Amylin agonists: pramlintide
-Aid amylin, which is co-secreted with insulin, to suppress glucagon secretion and regulate gastric emptying
-Can be used in DM1 but at lower doses
-Rarely used because it only serves to fine-tune blood sugar control while doubling the number of injections needed
-Contraindicated with GI disorders

α-glucosidase inhibitors: acarbose, miglitol
-↓ Glucose abs in the intestine
-Cause farts
-Contraindicated in bowel disorders, liver or renal impairment

Bile acid sequestrants: colesevelam
-Only used if patient needs lipid management as well as DM and can't tolerate a statin

Dopamine agonists: bromocriptine
-Only offer modest decreases in A1c with significant GI side effects

Insulin

Formulations
-Rapid: aspart, glulisine, lispro → 30 minute peak = before each meal
-Short (may be purchased without rx): regular → 2 hour peak = before each meal
-Intermediate: NPH (may be purchased without rx) → 6-10 hour peak = before breakfast + supper
-Long-acting/basal: detemir, glargine, aspart protamine → no peak = 1-2x daily
-Mixes: 70% NPH/30% regular, 75% aspart protamine/30% rapid

-Most syringes are U-100 but U-500's are 5x as concentrated and are manufactured for patients needing > 200 U of insulin daily

Initiating insulin in DM2
-Avg size patient → begin with 10 U basal insulin once daily
-Obese patient → begin with 0.2 U/kg basal insulin once daily
-Insulin mixes → begin with 0.6 U/kg total daily dose, 2/3 of this in the am and the remaining 1/3 in the evening

Initiating insulin in DM1
-Calculate total daily dose, which is 0.5-0.7 U/kg/day in adults
-Individual injections of basal and bolus are best: 50% of TDD should be basal, 50% should be bolus (and further divided into 3 mealtime doses)
-Can also use insulin mixes and administer as in DM2 for patients who are unwilling to do multiple injections per day

Switching from NPH to long-acting insulin: if NPH was once daily, a unit-to-unit change is ok; if NPH was 2x daily, ↓ total dose by 20% and give it as a once daily dose

Rule of 1800: 1800/TDD = how many mg/dL that 1 U of insulin will change your patient's BG

Rule of 500: 500/TDD = how many g of CHO that 1 U of insulin will cover

Type I Diabetes Mellitus

-Accounts for most cases of diabetes in kids under 19
-Genetic and environmental influences

Signs and symptoms
-Polyuria
-Polydipsia
-Weight loss
-Lethargy
-DKA is often the initial presentation

Workup
-Differentiate from DM2 by islet autoantibody screen

Management
-Formal training and education using a diabetes team
-Intensive insulin regimen
-Address depression and anxiety
-Annual urine microalbumin
-Ophtho visits at age 10 or after 3-5 years of diagnosis
-Lipid screens
-Periodic autoimmune thyroid and celiac screening

Diabetic Ketoacidosis

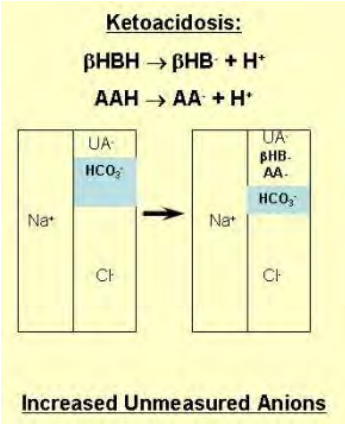
-Typically occurs in DM1 but can happen in DM2

Differential
-Other things that cause anion gap metabolic acidosis (lactic acidosis, aspirin, methanol, ethylene glycol, etc)

Precipitating Factors
-UTI is #1 trigger
-Pneumonia
-Pancreatitis
-MI
-Stroke
-Trauma
-EtOH or drug abuse
-Missing insulin dose
-Dehydration

Signs & Symptoms
-Usually present with early symptoms of SOB and abdominal pain
-Later symptoms of hyperosmolarity: polyuria, polydipsia

Workup
-Serum glucose is usually < 800
-3 ketone bodies are produced: acetoacetic acid, β-OH-butyrate, and acetone, so check dipstick UA and serum ketones
-BMP shows anion gap metabolic acidosis, hyponatremia (intracellular shift), hyperkalemia (shift out of cells)
-VBG to determine severity of acidosis
-May have elevated amylase or lipase even if pancreatitis is not present



Management
-Treat precipitating cause
-Correct deficits gradually to avoid cerebral edema
-Replace fluid deficits: give several L of NS, switching to ½ NS once hyponatremia corrects, and adding dextrose once serum glucose ↓ to 200
-Replace K+ deficits: initial hyperkalemia will rapidly become hypokalemia once insulin is started; may need to supplement K before starting insulin!
-Start insulin drip (preferred over SC insulin unless DKA is mild) ± bolus, continue with titration by protocol until BG is 200, then add on SQ therapy and wean off drip in ~1-2 hours
-Continue to monitor BMP and continue drip until gap closes
-Can monitor serum ketones, however they may persist up to 36 hours after the gap closes as removal takes some time
-Bicarb only given for severe acidosis

Hyperosmolar Hyperglycemic Nonketotic Syndrome (Hyperosmolar Hyperglycemic State)

-DKA without the ketoacidosis

Signs & Symptoms
-May see neurologic changes or coma

Workup
-Differentiate from DKA by absence of ketones
-Serum glucose is usually > 1000

Prognosis
-Associated with higher mortality than DKA

LIPID DISORDERS

Hyperlipidemia

-Most LDLs > 190 have a genetic component
-2° dyslipidemia can be a result of DM, hypothyroid, obstructive liver disease, chronic renal failure, meds, diet, or sedentary lifestyle

-Treating known hyperlipidemia up to age 75 results in significant reductions in morbidity and mortality

Screening
-Once between 2-10 years and q 3-5 years thereafter for pediatric patients with risks (obesity, HTN, FH)
-Every 5 years for patients 20-35 with risk factors (DM, FH, CV risk)
-Otherwise begin at 35 in men and 45 in women

Labs
-Lipid panel: TC, LDL, HDL, TG (TC and HDL can be measured even if patient was not fasting)
-Direct LDL: for nonfasting patients or if TG > 400
-CAD → *LDL goal > 100 or sometimes < 70*
-CAD equivalents (MI, stroke, DM, carotid artery disease, PVD, AAA) → *LDL goal > 100 or sometimes < 70*
-Risks factors (smoking, HTN, HDL < 40, FH premature CAD, men > 55, women > 65) → *one risk factor makes an LDL goal < 190 while two risk factors makes an LDL goal < 130*

Cholesterol Goals

Known CAD		*LDL goal < 100* *Sometimes < 70*
Risk equivalents	MI, stroke, DM, carotid artery disease, PVD, AAA	*LDL goal < 100* *Sometimes < 70*
Risk factors	Smoking HTN HDL < 40 FH premature CAD Men > 55, Women > 65	*1 risk factor makes an LDL goal < 190* *2 risk factors makes an LDL goal < 130*

Management
1.) With abnormal lipids without CAD or CAD risk equivalents, set an LDL goal first and initiate therapeutic lifestyle changes for 3-6 months (unless TG > 500, then address this first to prevent pancreatitis)
2.) If lifestyle changes don't work, consider meds
3.) If meds don't result in LDL goal being met, add a higher dose statin or another med
4.) If TG remain elevated, set another goal for TG that is LDL goal + 30
→ For CAD or CAD equivalent patients, start meds and lifestyle changes right away

Drug Class	Statins	Fibric Acid Derivatives	Bile Acid Sequestrants	Niacin	Ezetimibe	Other Options
Information	-Inhibit HMG CoA reductase -First-line medication to reduce LDL -Taken at bedtime, when cholesterol synthesis peaks -Check baseline LFTs before starting -FDA recs rechecking LFTs into treatment only if patient is symptomatic and d/c of therapy if LFTs have increased to 3x ULN -May also check TSH before starting statin b/c hypothyroidism predisposes to myalgias as well as dyslipidemia -If pt has myalgias, check CK but it isn't always elevated even with statin-induced myalgias; CK ↑ > 10x ULN is a reason for d/c of statin -Drug interactions -Contraindicated in chronic liver disease -Pregnancy category X	-Stimulate transcription factor to promote lipid metabolism -Contraindicated in severe renal or hepatic disease	-Cause liver to break down more cholesterol to make new bile -Good addition to statin therapy to further ↓ LDL -Contraindicated with TG > 400	-Blocks VLDL synthesis → shift in LDL from small and dense to larger and more buoyant -SE of flushing, prevent with aspirin before -Contraindicated with liver disease, gout	-Inhibits cholesterol absorption at the brush border -Better tolerated than a bile acid sequestrant -Contraindicated in liver disease	-Fish oil: ↓ TG by 20-50%, minor ↑ LDL and HDL, increased risk of bleeding -Red rice yeast: has natural HMG CoA reductase activity
Effect on cholesterol	↓ LDL by 20-60% (doubling dose → additional 6% ↓) ↓ TG by 7-30% ↑ HDL by 5-15%	↓ LDL by 5-10% ↓ TG by 20-50% ↑ HDL by 10-20%	↓ LDL by 15-30% No effect on TG ↑ HDL mildly	↓ LDL by 5-25% ↓ TG by 20-50% ↑ HDL by 15-30%	↓ LDL by 15-20% ↓ TG by 5-10% ↑ HDL mildly	
Specific Drugs	-Atorvastatin: ok to use in CKD -Lovastatin -Pravastatin: less muscle toxicity, good 2nd choice for pts with ↑ LFTs on other statins -Fluvastatin: less muscle toxicity, ok to use in CKD -Rosuvastatin -Simvastatin: new FDA warning, don't exceed 20 mg simva with amiodarone, amlodipine, or ranolazine; don't exceed 10 mg simva with diltiazem or verapamil	-Gemfibrozil -Fenofibrate	-Cholestyramine			

OTHER ENDOCRINE TOPICS

Hypoglycemia

Causes	Signs & Symptoms	Workup	Management
-Drugs: insulin or insulin secretagogue, other drugs -Alcohol -Critical illness -Malnutrition -Hormone deficiency: cortisol, glucagon, epinephrine -Nonislet cell tumor -Insulinoma -Post gastric bypass -Insulin autoimmune disorder -Factitious hypoglycemia	-Autonomic response: diaphoresis, weakness, tachycardia, palpitations, tremor, nervousness, hunger, paresthesias -Irritability, confusion, or seizure -Transient focal neuro deficits -Visual disturbance	-Only indicated in nondiabetic patients who exhibit Whipple's triad: symptomatic hypoglycemia confirmed on lab testing with relief of symptoms after plasma glucose is raised -If BG is low in a nondiabetic, also draw insulin level, C-peptide, and oral hypoglycemic agent screen before treating -Insulin and C-peptide elevated in insulinoma, oral agent use -Exogenous insulin use will also cause elevated insulin levels but C-peptide will be low	-Administer rapidly absorbed carb if able to eat or IV glucose, or glucagon SQ if no IV access -Maintenance glucose as needed with monitoring every 10-15 minutes

Hypothermia

Differential	Signs & symptoms	Workup	Management
-Environmental exposure -Hypothyroidism or adrenal insufficiency -Sepsis -Neuromuscular disease -Malnutrition -Thiamine deficiency -Hypoglycemia -EtOH or CO intoxication -Meds: anxiolytics, antidepressants, antimanics, antipsychotics, opioids	-Mild: tachypnea, tachycardia, hyperventilation, ataxia, dysarthria, impaired judgment, shivering, cold diuresis -Moderate: bradycardia, hypoventilation, CNS depression, hyporeflexia, loss of shivering, paradoxical undressing, arrhythmias -Severe: pulmonary edema, oliguria, areflexia, coma, hypotension, bradycardia, arrhythmias, asystole -Pt will be coagulopathic as factors are inhibited below 37°	-POC glucose: insulin does not function below 30° -EKG -BMP -CBC -Lactate -CK -Fibrinogen -ABG -CXR	-ABCs -Head to toe survey for frostbite, etc. -May need to check pulses with a Doppler -Chest compressions only if no signs of life -Warmed crystalloid infusions -Avoid rough movement (heart sensitive to arrhythmias during this time) -Rewarming: passive if mild, active if mod to severe

Polycystic Ovarian Syndrome

-Highly genetic predisposition **Signs & Symptoms** -Oligomenorrhea -Hyperandrogenism → acne, hirsutism, male-pattern hair loss, DUB due to endometrial hyperplasia -Obesity -Glucose intolerance -Dyslipidemia -OSA -NASH	**Workup** -Diagnose with 2/3 Rotterdam criteria: oligomenorrhea, hyperandrogenism, polycystic ovaries on US -Also can check total testosterone -Rule out other causes of irregular menses: bHCG, prolactin, TSH, FSH	**Management** -Weight loss and exercise -Follow fasting lipids and glucose regularly -Assess for depression, eating disorders, and sleep apnea regularly (hi prevalence in this population) -Fertility evaluation if desired -Hirsutism and other androgenic symptoms → OCPs, adding spironolactone later if needed (has antiandrogenic effects) -Endometrial protection against hyperplasia → OCPs or intermittent progestins to induce bleeding -Glucose intolerance → metformin especially helpful if infertility also present -Infertility → weight loss, consider clomiphene

GENITOURINARY SYSTEM

GENITOURINARY TRACT CONDITIONS

Benign Prostatic Hypertrophy (Hyperplasia)

-Risk increases with age, and prostate undergoes growth spurt after age 40
-Only some men are symptomatic

Signs & symptoms
-Irritative symptoms = bladder storage problems like urgency, frequency, nocturia, urge incontinence, stress incontinence
-Obstructive symptoms = bladder emptying problems like hesitancy, poor flow, intermittency, straining, dysuria, dribbling, incomplete bladder emptying

Workup
-DRE +PSA (prostate size will not be correlated to symptoms, rather you are trying to detect malignant cause)
-Abdominal exam for bladder distension
-Neuro exam
-Post-void residual/bladder scan
-UA to exclude infection or hematuria
-Rule out urethral stricture of bladder neck contracture (no instrumentation, urethritis, or trauma)

Management
-Always based on symptoms (calculate AUA symptom index score)
-Watchful waiting only if pt is not bothered
-Natural saw palmetto supplement
-Refer to urology for surgical intervention with recurrent UTIs, recurrent gross hematuria, bladder stones, CKD, urinary retention

Pharmacologic Therapy	MOA	Information
Alpha-1 blockers	-Decrease muscle tone for rapid symptom relief	-Do not decrease prostate size -Nonselective (terazosin, doxazosin, prazosin) not recommended with concomitant HTN due to risk of first dose syncope and orthostatic HTN, take 2-4 weeks for full effect -Selective (tamsulosin, alfuzosin) have no effect on BP and begin to work in days to 1 week, slight risk of ejaculatory dysfunction
5-alpha reductase inhibitors	-Blocks conversion of testosterone → dihydrotestosterone	-Finasteride -For those who can't tolerate alpha- blockers -Shrink prostate -Take 6-9 months -Pregnancy category X -AEs of ED and ejaculatory dysfunction
Saw palmetto		-Not shown to be helpful in clinical trials

Congenital Urinary Tract and Kidney Abnormalities

-Account for 25% of all anomalies identified in the prenatal period
-Caused by genetic and environmental factors

Types
-Renal dysplasia
-Renal agenesis
-Renal tubular dysgenesis
-Polycystic renal disease
-Collecting duct abnormalities → ureteropelvic junction obstruction, megaureter, ectopic ureter, vesicoureteral reflux, bladder exstrophy, posterior urethral valve
-Horseshoe kidney
-Pelvic kidney

Postnatal workup
-Voiding cystourethrography
-Renal scan
-DMSA scan to detect ectopic renal tissue

Management
-Serial US to monitor compensatory growth of unaffected kidneys

Vesicoureteral Reflux

-Currently this is treated as it is thought to promote renal scarring and recurrent pyelonephritis
-Can occur prenatally and may be seen on prenatal US as hydronephrosis
-Graded I-V based on severity

Workup
-Renal US for infants diagnosed with prenatal hydronephrosis
-Contrasted voiding cystourethrogram
-Radionuclide cystogram
-Serum creatinine
-UA
-Screen siblings for reflux

Management
-Grades I and II can be managed with observation
-Kids with > grade III reflux are treated
-Antibiotic prophylaxis: Septra, trimethoprim, nitrofurantoin
-Surgical correction
-Annual imaging for medical or observational therapy
-Annual growth checks, BP, and UA

Cryptorchidism

-Most undescended testes will descend spontaneously by the time an infant is several months old but will rarely occur after 6 months
-Ectopic testes are descended but are in an aberrant position such as the inguinal pouch, suprapubic region, or perineum
-Occasionally descended testes can ascend as child grows
-Retractile testes and located suprascrotally but can descend to the scrotum and remain there as long as the cremasteric reflex is overcome

Management
-Refer for testes not descended by 6 months for surgical orchiopexy due to risk of malignant degeneration, subfertility, torsion, or inguinal hernia

Erectile Dysfunction

Causes
-Usually neurovascular, and may be an early marker of vascular disease
-HTN
-Dyslipidemia
-Smoking
-Hyperglycemia
-Penis curvature
-Diabetic neuropathy
-MS
-Low testosterone
→ It is normal for men to have slowly decreased erection hardness and longer refractory periods in between orgasms as they age

Workup
-Ask if pt is having normal erections, if not then it is an organic issue, if yes than it is a psychogenic issue
-Early morning testosterone with free testosterone
-Lipids
-FBG
-Prolactin if there is nipple discharge

Management
-Helpful book "The New Male Sexuality"
-Refer for surgical options: inflatable prosthesis, balloon dilation, vein ligation
-Smoking cessation has greatest effect when patient is younger with mild symptoms

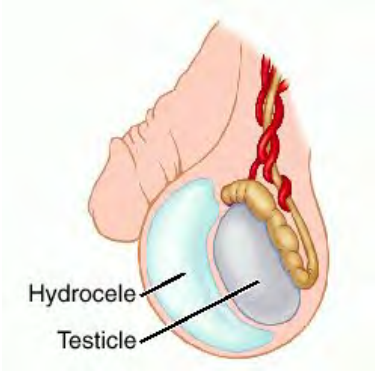
Pharmacologic Therapy	MOA	Information
Phosphodiesterase type 5 inhibitors	-Interfere with cGMP breakdown → continued dilate of inflowing blood vessels	-Sildenafil, tadafil, vardenafil -First line agents -May need 6-8 tries before these meds work -AEs: loss of blue-green color vision, hypotension -Contraindications: concomitant nitrates, severe CV disease
Yohimbe	-Natural alpha-2 adrenergic-R blocker derived from African tree	-Safe and low cost -Not currently recommended by AUA
Alprostadil injection	-Prostaglandin penile injection	-Not a good initial treatment -Issues of fibrosis -Painful -Contraindicated in sickle cell
Prostaglandin intraurethral pellet		-Expensive, does not work for many

Hydrocele

-Collection of peritoneal fluid between the parietal and visceral layers of the tunica vaginalis in the scrotum

Etiologies
-Fluid imbalance between secretion/absorption in the tunica vaginalis
-Injury or inflammation
-Neoplasm
-Torsion

Signs & symptoms
-Soft, cystic scrotal mass
-Usually painless
-Mass transilluminates
-May be bilateral



Workup
-US

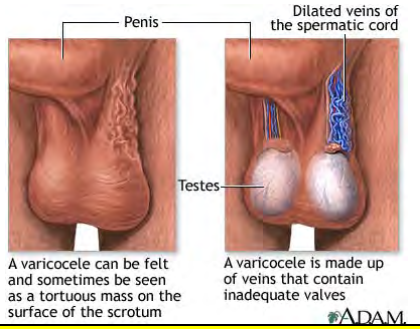
Management
-Treatment not necessary unless symptomatic
-Surgical excision
-Simple aspiration has high recurrence rate
-Surgical repair of patent processus vaginalis

Varicocele

-Dilation of pampiniform vein plexus
-May be due to valve insufficiency in gonadal veins

Signs & symptoms
-More common on the left side
-"Bowl of spaghetti" or "bag of worms" appearance that ↑ with valsalva
-Oligospermia or asthenospermia
-Painful, dull, or heavy sensation in scrotum

Management
-Scrotal support
-Analgesics
-Surgical repair



Incontinence

Type	Stress Incontinence	Urge Incontinence	Overflow Incontinence	Functional Incontinence
Signs & symptoms	-Leakage with coughing, sneezing, standing -No leakage when supine	-Involuntary loss of large amount of urine preceded by intense feeling of having to void but without sufficient warning -Loss of urine with sound of running water or waiting to use toilet -Unrelated to position -Normal PVR	-Continuous or persistent urine loss through day and night due to chronic urine retention -Painful abdomen -Large PVR -Urine dribbling -Unawareness of urine loss	-Inability to toilet due to cognitive impairment, physical disabilities, psychological problem, or environmental barriers
Management	-Topical estrogens if due to atrophic vaginitis -Kegel exercises -Weight loss -Refer to urology/urogyn for sling procedures, vaginal repair, injection of bulking agents, or pessary fitting	-Fluid restriction after 6pm -Frequent, scheduled voids -Relaxation techniques -Bladder antispasmodics: tolterodine, oxybutynin -Anticholinergics -Refer to urology for nerve stimulator, Botox injections	-Alpha blockers to relax bladder neck -Treat BPH if present -Refer to urology for prostate resection -Indwelling, intermittent, or suprapubic catheterization -Scheduled toileting	-Decrease use of sedatives, alcohol, and anticholinergics -Reschedule meds to not act during sleeping hours -Easy access to commode or urinal -Easy-to-remove clothing -Scheduled or prompted toileting

Paraphimosis & Phimosis

Paraphimosis

-Retracted foreskin in uncircumcised male that can't be returned to normal position

Causes
-Over-retraction of foreskin while cleaning
-Sexual intercourse
-Iatrogenic: cystoscopy, bladder catheterization
-Repeated episodes of balanitis

Signs & Symptoms
-Penile swelling and pain
-Blue or black discoloration of glans if ischemic

Management
-Immediate urologic consult if ischemic
-Pain control, may need local anesthesia
-Reduce swelling
-Reduce foreskin back manually
-May need surgical techniques for failed manual reduction

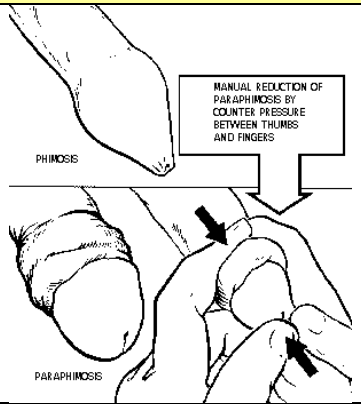
Phimosis

-Foreskin of uncircumcised male can't be retracted

Causes
-Lichen sclerosus
-Balanitis

Management
-Steroid cream
-Manual stretching
-Circumcision

Prognosis
-Untreated phimosis is a risk factor for urinary retention and penile carcinoma



Nephrolithiasis/Urolithiasis

-Southern "stone belt"
-FH incurs 3x greater risk

Types of stones
-Ca oxalate
-Ca phosphate
-Struvite: may be secondary to recurrent infection
-Uric acid: h/o gout
-Cystine
-Medication crystallization
-Staghorn calculi associated with *Proteus*

Signs & symptoms
-Abrupt flank pain that is severe, colicky, may radiate to scrotum/labia or groin
-Nausea and vomiting
-Hematuria
-Prior episodes
-CVA tenderness
-LQ pain on palpation
-Pts will want to constantly move
-Urinary frequency and urgency with stone lodgement at ureterovesicular junction
-Less pain after stone passes into the bladder and through the urethra
-Size of stone does not correlate to severity of symptoms

Differential: AAA, appendicitis, tubo-ovarian abscess, ectopic pregnancy, renal cell carcinoma, intestinal obstruction

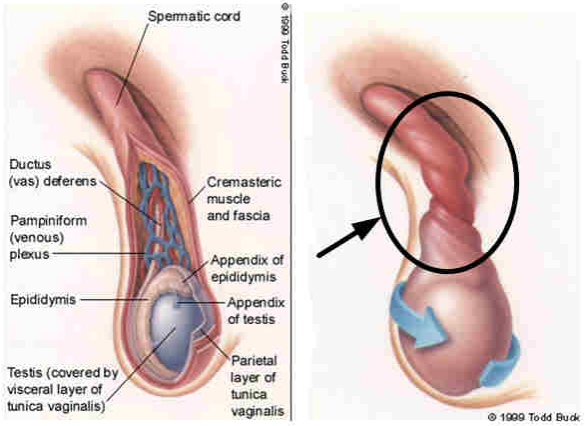
Workup
-Spiral CT (no contrast) for evaluation of flank pain in first-time stoners, initial imaging of choice
-US (less sensitive but good for eval of secondary signs of obstruction)
-KUB will show 85% of stones since most are made of Ca
-UA usually shows microscopic or gross hematuria
-Urine culture to look for *Proteus*
-Urine pH persistently < 5.5 suggests uric acid or cystine stones
-Urine pH persistently > 7.2 suggests struvite stones
-Urine pH between 5.5-6.8 suggests Ca-based stones
-CBC to look for infectious cause
-First-time stoners with uncomplicated stone need BMP and uric acid levels
-Recurrent stoners or FH need extensive workup, do 2 x 24 hour urines and refer to nephrology

Management
-Asymptomatic stones found incidentally are usually left alone unless pt is a pilot, frequent flyer, or only has one kidney
-Send to ED if septic, intractable n/v, solitary kidney, large or proximal stones
-If tolerating PO meds and stone is < 5 mm, can do home management with oral fluids, pain control, tamsulosin, urine straining for 48-72 hours, nephrology referral if no improvement
-Stones > 5 mm may need urology eval for shock wave lithotripsy or other method of stone removal
-Admit for pts with intractable pain, inability to take PO meds and fluids, or fever

Prevention
-Limit sodium intake
-Don't limit Ca supplements or intake unless stones are Ca
-Potassium citrate: regulates urine pH and binds Ca
-Thiazide diuretics reduce Ca
-Allopurinol lowers urinary uric acid
-Tiopronin/D-penicillamine reduces cystine levels
-Acetohydroxamic acid with antibiotics reduces struvite formation

Testicular Torsion

-Twisting of the spermatic cord within a testicle, cutting off blood supply
-A result of inadequate fixation to the tunica vaginalis
-Can be spontaneous or post trauma
-More common in neonates or postpubertal males



Signs & Symptoms
-Scrotal pain and swelling
-N/v
-Abdominal pain
-May wake child up in the middle of the night
-Tender epididymis, elevated testis, and scrotal discoloration
-Absent cremasteric reflex

Management
-Color Doppler US

Management
-Surgical emergency, must be treated within 4-6 hours with irreversible damage and possible infertility after 12 hours
-Manual detorsion if surgery unavailable

INFECTIOUS & INFLAMMATORY CONDITIONS			
Cystitis			
Group	**Women**	**Men**	**Pediatrics**
Info	-Risk factors: sex, spermicide, diaphragms, DM, h/o recurrent UTIs, recent abx -Agents: usually *E. coli* or *Staph saprophyticus*, also *Proteus, Klebsiella*, enterococci -Uncomplicated = healthy young nonpregnant female -Complicated is anything else!	-Rare in men under age 50 -Risk factors: MSM, uncircumcised, low CD4 count	-Agents: 90% are *E. coli*, also *Staph saprophyticus, Enterococcus*, enterics -Uncomplicated = limited to lower urinary tract, child > 2 years, no underlying medical problems, no underlying anatomic or physiologic abnormalities -Complicated = upper tract disease, MDR pathogen, host with malignancy, DM, or anatomic or physiologic abnormality, indwelling catheter -Risk factors: female, sexual activity, vesicoureteral reflux, polycystic kidneys, dysfunctional elimination syndrome, fecal impaction, paraplegia, sickle cell anemia, kidney transplant, DM, bladder stones, immunodeficiency, recent instrumentation
Signs & symptoms	-Lower UTI: dysuria with muscle spasm, frequency with small vol, urgency, suprapubic pain, ± hematuria -Upper UTI: lower symptoms + fever > 100.4, flank pain, CVA tenderness, nausea, vomiting -Elderly may have AMS only	-Elderly may have AMS only	-Infants < 1 month: may only have fever -Older kids: dysuria, frequency, urgency, enuresis, abdominal or suprapubic pain, hematuria (fever, chills, flank pain suggest upper tract infection)
Differential	-Urethritis with STI -Vaginitis -PID -Cervicitis -FB -HSV	-Urethritis -Prostatitis -HSV	-Chemical cystitis -Autoimmune cystitis -Drugs -Bladder dysfunction -Vulvovaginitis, cervicitis, or urethritis -Prostatitis or epididymo-orchitis -Nephrolithiasis -Urethral stricture -Neoplasm -Vaginal foreign body
Workup	-Uncomplicated → proceed to empiric treatment -Complicated = UA with microscopy (look for pyuria, bacteriuria, varying hematuria), culture, wet prep -Hematuria will not be present in cervicitis or urethritis but is common in UTI!	-UA and always culture -DRE	-UA with culture (catheterized specimen for non-toilet trained children) -GC/chlamydia if sexually active -Renal bladder US indicated for first febrile UTI in kids under 2 who did not have normal prenatal screening US, for kids of any age with recurrent UTIs, and kids of any age with UTI, poor growth, HTN, or FH of renal disease -VCUG indicated for evaluation of possible reflux in kids of any age with > 2 febrile UTIs
Treatment	-Uncomplicated: 3-5 days of nitrofurantoin (DOC) or FQ if more severe symptoms, Septra alternative if local *E. coli* resistance is not > 20% -Complicated: home treatment with 7-14 days of FQ or Septra OK as long as there is no n/v, otherwise send to ED for inpatient treatment -Preventive cranberry juice acidifies urine and prevents pathogens from binding to urinary epithelia -Short-term phenazopyridine for dysuria -Consider prophylaxis for women with > 2 UTIs in last 6 mo or > 3 UTIs in last year: clean UA followed by 6 months of Septra, nitrofurantoin, cefaclor, cephalexin, or FQ -Postmenopausal women may benefit from vaginal estrogen cream	-7 days of Septra or FQ	-Admit for infants < 2 months, immunocompromised, vomiting, inability to tolerate orals, lack of outpatient f/u, and failure of outpatient therapy -Ages 2-13 years → 2nd or 3rd generation cephalosporin, add amoxicillin if suspecting enterococcal infection -Age > 13 → Septra or cephalosporin -In general, treat for 3-5 days if afebrile, 10 days if febrile -First episode in uncomplicated female should be treated 5-7 days -Young children, male adolescents, and children with recurrent, febrile, or complicated cystitis should be treated for 7-14 days

Pyelonephritis

-Agent is usually uropathic strain of *E. coli*

Signs & symptoms
-Same manifestations as lower UTI + fever > 100.4, flank pain, CVA tenderness, n/v, rigors
-Sepsis
-Multi-organ dysfunction
-ARF
-Alternative presentation of weeks to months of insidious, nonspecific symptoms such as malaise, fatigue, nausea, or abdominal pain

Workup
-UA with microscopy & culture
-Blood cultures if hospitalized

Management
-If mild with no n/v → outpatient treatment with cipro
-Mod-severe → inpatient treatment with initial empiric IV ceftriaxone, cipro, or imipenem, f/u culture 2 weeks after therapy

Epididymitis

Etiologies
-Men under 40 → STI
-Men over 40 → seeding UTI or prostatitis, gram negs

Signs & symptoms
-Fever
-Irritative voiding symptoms
-Painful enlargement of epididymis with scrotal swelling
-Urethritis
-May have tender prostate
-Prehn sign: relief with elevation of scrotum above pubic symphysis, not reliable

Differential: scrotal tumor (would be painless), testicular torsion, epididymis or appendage torsion

Workup
-CBC
-GC/chlamydia, followed by urethral swab with gram stain if negative
-UA will show pyuria, bacteriuria, and varying hematuria if agent is not an STI

Management
-Bed rest with scrotal elevation
-Treatment of pathogen: 10-21 days of abx if STI, 21-28 days of abx if not STI

Orchitis

-Usually viral: mumps, rubella, coxsackie, echovirus, parvovirus
-May be STI if sexually active

Signs & symptoms
-Scrotal swelling
-Pain and tenderness with erythema and shininess of the overlying scrotal skin
-May also have epididymis involvement with STI orchitis

Differential
-Epididymitis
-Testicular torsion: absent cremasteric reflex
-Appendix testis or appendix epididymis torsion
-Trauma
-Incarcerated inguinal hernia

Management
-NSAIDs
-ABs if suspecting STI cause
-Scrotal support
-Ice packs

Urethritis Differential

-With discharge → think gonorrhea or chlamydia first, others include *Mycoplasma, Ureaplasma, Trichomonas*
-Reactive arthritis with associated urethritis
-Urethral carcinoma
-Men: balanitis
-Women: candidiasis, cystitis

Prostatitis

Acute Bacterial Prostatitis

-Least common form of prostatitis
-Can be life-threatening

-Organisms: mostly gram negs, also gonorrhea or chlamydia in sexually active younger men

Etiologies
-STI
-Urine reflux seeding
-Spread from distant source or adjacent infection

Signs & sympoms
-Frequency, urgency, dysuria, nocturia, change in urine stream
-Low back pain
-Genital pain
-Abdominal pain
-Fever and chills
-Nausea/vomiting
-Hypotension
-Tender prostate on DRE, may be enlarged or fluctuant, be careful to not cause bacteremia

Differential: acute pyelonephritis, acute epididymitis, acute diverticulitis, BPH, malignancy

Workup
-UA with culture: should be +
-Urethral culture if discharge present
-GC/chlamydia

Management
-Send to ED for IV abx

Chronic Bacterial Prostatitis

Etiologies
-May evolve from untreated acute bacterial prostatitis

Signs & symptoms
-Wax and wane
-Irritative voiding symptoms
-Dull suprapubic or perineal discomfort
-Recurrent UTIs with same organism and no explanation
-DRE may appear normal

Workup
-UA using Meares-Stamey 4 glass method: 1st 10 mL is from the urethra, midstream void from the bladder, prostatic massage for 3rd sample (this should be +), next 10 mL will contain urethral and prostatic fluid

Management
-Prolonged course of FQ or Septra
-NSAIDs
-Sitz baths
-Consider suppressive abx treatment if 3+ recurrences per year

Chronic Nonbacterial Prostatitis and Chronic Pelvic Pain Syndrome

-Urinary or genital pain with no evidence of infection, with symptoms for 3 of the last 6 months
-Affects all ages of men
-The most common form of prostatitis

Etiologies
-Not well understood
-Nanobacteria?
-Voiding dysfunction
-Pelvic floor myalgia

Signs & symptoms
-Wax and wane

Differential: infection, GU cancer, urinary tract disease, urethral stricture, neurologic disease, emotional disorder

Workup
-May have WBCs in prostatic secretions

Management
-Refer for cystoscopy if at risk for bladder cancer
-NSAIDs for pain control
-Alpha blockers for urinary symptoms
-Muscle relaxants for painful ejaculations
-Finasteride to shrink prostate
-Sitz baths
-Pelvic floor PT

GENITOURINARY NEOPLASMS

Bladder Neoplasms

-More common in men than women
-Risk factors: smoking, exposure to dyes and solvents

Signs & symptoms
-Painless hematuria
-Urinary frequency or urgency
-May be asymptomatic

Workup
-Cystoscopy is initial test of choice
-Repeat urine cytologies (low sensitivity)
-CT to assess local extent of disease
-Staging based on biopsy results and imaging

Management
-Neoadjuvant chemo
-If superficial, resection (usually total cystectomy with urinary diversion) ± intravesicular chemo
-If advanced, combo chemo ± radiation

Prognosis
-Early disease has > 80% survival

Benign Bladder Neoplasms

Low-Grade Intraurothelial Neoplasia

Urothelial Papilloma
-Can have malignant potential

Inverted Papilloma
-Can have malignant potential

Malignant Bladder Neoplasms

Carcinoma In Situ

Squamous Cell Carcinoma
-More common in areas of the world with schistosomal infections
-Aggressive

Adenocarcinoma
-Aggressive

Small Cell Carcinoma
-Neuroendocrine in origin
-Aggressive clinical course with poor prognosis

Metastatic Disease
-Commonly from the colon or rectum, prostate, or cervix

Invasive Urothelial Cell Carcinoma
-AKA transitional cell carcinoma
-Most common form of bladder cancer in US

Prostate Cancer

-Usually adenocarcinoma
-The most commonly diagnosed male cancer and 2nd leading cause of male cancer deaths
-Risk factors: age, black, high fat diet, FH, obesity
-No association with smoking, sexual activity, prior infections, or BPH

Screening
-USPSTF grade I for men up to age 75 and grade D after 75
-If patient elects, DRE and PSA should be done every 2 years
-PSA will be elevated in cancer, inflammation, or BPH, and will naturally rise as men age

Signs & symptoms
-Asymptomatic early in disease
-Later disease: obstructive urinary symptoms, hematuria, hematospermia
-Bone pain with mets

Workup
-Prostate biopsy guided by transurethral US, with scoring by Gleason system
-MRI
-PET if suspected mets
-CXR, LFTs for mets

Management
-Treatment based on life expectancy, general health, tumor characteristics
-Treatment is controversial for localize disease
-Radical prostatectomy
-Radiation
-Hormone therapy for advanced or metastatic disease

Renal Neoplasms

-"Small renal mass" is often detected incidentally and defined as a contrast-enhancing mass < 4 cm; most are renal cell carcinomas

Signs & Symptoms
-Most are asymptomatic
-Hematuria
-Paraneoplastic syndrome
-Abdominal or flank mass
-Abdominal pain

Differential
-Polycystic kidney disease

Workup
-Imaging can't reliably differentiate a benign tumor from RCC
-Dedicated renal CT or MRI for incidental lesions
-Surgical resection for masses 1-4 cm
-Percutaneous biopsy for low malignancy suspicion or for nonsurgical candidates

Management
-Active surveillance if < 1 cm

Prognosis
-Neither tumor size at diagnosis nor growth rate are accurate predictor of malignancy status

Benign Renal Neoplasms

Simple Renal Cyst

Angiomyolipoma

Renal Oncocytoma

Metanephric Adenoma

Cystic Nephroma

Renal Medullary Fibroma

Malignant Renal Neoplasms

Renal Cell Carcinoma
-Accounts for 80% of renal cancers
-More common in men than women
-Risk factors: smoking, obesity, HTN, polycystic kidney disease, occupational exposures, prolonged NSAID use, chronic hep C, sickle cell disease
-Signs & symptoms: hematuria, flank pain or abdominal mass, cough, bone pain with mets, paraneoplastic syndromes
-Nephrectomy needed

Prognosis
-Good for cancers confined to renal capsule
-50-60% for tumors extending beyond capsule
-0-15% for node positive tumors

Testicular Cancer

-Risk factors: cryptorchidism, abnormalities in spermatogenesis, FH
-Most commonly germ cell tumor, but cana lso be stromal tumor

Screening
-USPSTF grade D in asymptomatic adolescents and adult males

Signs & symptoms
-Firm, painless mass arising from the testis
-Scrotal pain
-Affected area is usually unilateral
-Signs of mets: cough, GI, back pain, neuro signs, supraclavicular lymphadenopathy

Investigation
-Scrotal US: distinguishes benign vs malignant and intra vs extratesticular
-Excisional biopsy
-β-hCG levels: will be elevated in some carcinomas and seminomas
-AFP: elevation excludes diagnosis of seminoma
-Chest, abdomen, and pelvis CT

Management
-Inguinal orchiectomy with f/u of tumor markers
-May need chemo

Prognosis
-High survival rate if caught early

Wilms Tumor

-A renal cancer that is the 4th most common childhood cancer
-Most diagnosed before age 10

Signs & symptoms
-Abdominal mass or swelling
-Abdominal pain
-Hematuria
-HTN

Management
-Refer to surgery and pediatric cancer center
-Abdominal US or contrasted CT to differentiate from other masses

Prognosis
-Good with early disease
-Lung is most frequent first site of recurrence

RENAL DISEASES

Chronic Kidney Disease

-Defined as GFR < 60 for at least 3 months or presence of kidney damage irrespective of GFR
-Blacks have 3.8x greater risk, native Americans have 2x greater risk, and Latinos have 1.5x greater risk

Stages
-Stage I = GFR > 90 with kidney damage but asymptomatic
-Stage II = GFR 60-89 and kidney damage but asymptomatic
-Stage III = GFR 30-59, mild anemia, ↑ BUN and Cr, but asymptomatic
-Stage IV = GFR 15-29, symptoms of fatigue, electrolyte imbalance, acidosis, anemia
-Stage 5 = GFR < 15 or dialysis-dependent

Screening
-Regularly screen those with risk factors for DM, glomerular diseases, vascular diseases, cystic diseases, transplant complications, FH of severe kidney disease, CV disease, etc, as early diagnosis can add 2+ ESRD-free years to a patient's lifespan
-Use spot urine:creatinine test in diabetics
-Use spot test or urine dipstick in all other populations

Signs & symptoms
-Salt and water imbalance → fluid accumulation, HTN, peripheral edema, hypo or hypernatremia, neuro effects
-Cardiac conduction errors due to potassium imbalance
-Imbalance of Ca and P affects bone metabolism and cell membrane activity → loss of bone Ca with metastatic deposition, osteoporosis, fx
-Acid/base imbalance affects functioning of cells and enzymes
-Buildup of uremic toxins → nausea, anorexia, abnormal metallic taste in mouth, insomnia, seizures, coma, bleeding, immune dysfunction, arrhythmias, accelerated atherosclerosis, cardiomyopathy, pruritus
-No erythropoietin → anemia
-No activation of vit D → hyperparathyroidism, renal osteodystrophy, fx
-Heart disease: CAD lesions, CHF, acute MI (most patients will die of a cardiac-related cause before ESRD develops)

Management
-BP control using ACEIs
-Smoking cessation
-Lipid management
-Tight control of A1c in diabetics
-Avoid nephrotoxins: contrast, NSAIDs
-Monitor for anemia and treat with folate, B12, or Fe if needed, then consider EPO
-Check GFR 1-2 times per year
-Every 3 months monitor PTH, P, Ca, bicarb, and vitamin D if stage 3 or above
-Diet: good Ca intake, minimize P, low salt, fluid restriction to 2 L
-Phosphate binders if needed: Ca acetate, sevelamer, lanthanum
-Treat hyperkalemia: usually avoided as long as GFR > 10, prevent by avoiding K-sparing diuretics and cautious use of ACEI in ESRD
-Treat hypokalemia
-Treat vol overload: thiazides for stages 1-2, loop for stages 4-5 (greater diuresis)
-Daily baby aspirin
-Refer to nephrologist when GFR < 30

Hydronephrosis

-Distension and dilation of the renal pelvis and calyces
-Obstruction can occur anywhere along the urinary tract

Etiologies
-BPH
-Prostate cancer
-Nephrolithiasis
-Structural abnormalities

Signs & symptoms
-Depends on site of obstruction, degree, and rapidity of development
-Pain
-Change in urine output
-Hematuria
-Increasing serum creatinine
-HTN
-Distended abdomen or abdominal mass

Workup
-US is imaging of choice, will show dilation of collecting system

Management
-Relief of obstruction
-Bladder catheterization
-Nephrostomy tube

Prognosis
-Return of renal function depends on severity and duration of obstruction

Sequelae
-Risk of UTI, urosepsis or ESRD if untreated

Polycystic Kidney Disease

-Common after age 50

Etiologies
-Genetic form characterized by multiple cysts in both kidneys, with increased risk of UTIs, renal cell carcinoma, ESRD

Signs & symptoms
-Usually asymptomatic
-If inherited form, may have abdominal or flank pain, hematuria, history of UTIs, history of stones, HTN, abdominal mass

Workup
-UA for hematuria or proteinuria
-Renal US: diagnosis depends on age and # of cysts present

Management
-Usually requires kidney transplant

Acute Kidney Injury (Acute Renal Failure)

• Azotemia: abnormally high levels of nitrogen-containing compounds, such as urea, creatinine, various body waste compounds, and other nitrogen-rich compounds in the blood → can result from many disorders including renal failure • Acute renal failure: an abrupt decrease in GFR sufficient to result in azotemia and perturbation of ECF volume, electrolyte, and acid-base balance = kidney is not removing proteins that should normally be removed from the blood	**Signs & symptoms** -Symptoms of uremia: DOE, pericarditis, fatigue, loss of appetite, headache, nausea, vomiting, nocturia, AMS, SOB, pruritus, easy bruising, asterixis -Flank pain from stretching of fibrous capsule surrounding kidney during blockage -Irregular heartbeat from hyperkalemia -Dehydration -Decreased UOP → vol overload, peripheral edema, pulmonary edema, pulmonary rales, elevated JVP, cardiac tamponade, pulsus paradoxus (drop in BP during inspiration) -Seizures -Kussmaul respirations from acidosis	**Workup** -Definition of AKI: acute ↓ in renal function with Cr ↑ of 0.5 (or 50%) above baseline, or CrCl ↓ by 50% -Assess degree of renal dysfunction: estimate GFR -UA -Labs: urine Na, urine Cr, compare with serum Na, Cr, and BUN -Renal US to rule out postrenal causes

Prerenal AKI

-A result of interrupted blood flow to the kidneys → ischemia of the proximal tubular cells -Decreased renal perfusion means renin is released → maximal retention of Na and water in an effort to ↑ circulating vol **Etiologies** -Decreased effective circulating vol: HF, cirrhosis, nephrotic syndrome, excessive diuretic use, vomiting, NGT, diarrhea, burns, DKA, hypercalcemia, Addison's, shock, pancreatitis, 3rd spacing -Decreased renal perfusion: severe hypoalbuminemia, sepsis, psychotropic drug overdose, excessive antihypertensives, NSAIDs, renal artery stenosis or renal vein thrombosis -Cardiac arrest -Low BP: anorexia, GIB, cardiac surgery	**Signs & symptoms** -Hypotension -Decreased UOP with concentrated urine **Management** -IV NS -Treat underlying illness -Stop antihypertensives or diuretics -Octreotide in patients with cirrhosis (helps increase blood flow to kidneys when sick liver is shutting it down)

Intrinsic Renal AKI

Acute Tubular Necrosis	Acute Interstitial Nephritis	Glomerulonephritis
-The most common cause of AKI where there is abrupt and sustained decline in GFR occurring within minutes to days in response to an acute ischemic or nephrotoxic insult **Signs & symptoms** -Volume overload -Oliguria or anuria **Workup** -Rise in Cr and BUN in = proportions -Check urine sodium (should be > 40) to distinguish from volume depletion (Na < 20) -FENa increased at > 2-3% as either excess sodium is lost due to tubular damage, or the damaged glomeruli result in hypervolemia resulting in the normal response of sodium wasting -Will see metabolic acidosis with hyperkalemia -UA shows multiple granular and epithelial cell casts with free epithelial cells **Management** -Typically conservative -Consider dialysis if severe	-Results in abrupt deterioration in renal function with inflammation and edema of the renal interstitium **Etiologies** -Allergic: cephalosporins, diuretics, NSAIDs, penicillin, rifampin, sulfa -Infectious: hantavirus, HIV, Legionnaire's, leptospirosis, pyelonephritis -Autoimmune: cryoglobulinemia, Sjogren's, SLE -Infiltrative: amyloidosis, multiple myeloma, sarcoidosis **Workup** -UA showing proteinuria, pyuria, hematuria, renal tubular epithelial cells or casts, eosinophiluria -Increased BUN and Cr, hyper or hypokalemia -Hyperchloremic metabolic acidosis -Elevated LFTs	**Focal disease** -IgA nephropathy -Membranoproliferative glomerulonephropathy -SLE **Diffuse disease** -Goodpasture's disease -Churg-Strauss -Cryoglobulinemia -Membranoproliferative glomerulonephropathy -SLE -Vasculitis -Wegener's -Microscopic polyangiitis -Postinfectious glomerulonephritis: usually Strep but can also be TB, HIV, hepatitis, MRSA, or meningococcal infection-induced, with onset 1-3 weeks after infection **Workup** -UA showing red cell casts

Post-Renal AKI

-A result of obstruction of urine flow

Etiologies
-Enlarged prostate
-Kidney stones
-Bladder tumor or injury
-Obstructed urinary catheter
-GYN cancers
-Retroperitoneal fibrosis
-Neurogenic bladder or spinal cord injury
-Retention secondary to meds: anticholinergics, opioids, amphetamines

Working Up Cause of AKI

			Intrinsic Renal Disease		
	Azotemia	***Postrenal Azotemia***	***Acute Tubular Necrosis***	***Acute Interstitial Nephritis***	***Acute Glomerulonephritis***
Serum BUN:Cr Ratio	-BUN:Cr ratio > 20:1 -Rise in BUN out of proportion to rise in Cr because decreased flow to kidney means back-up of urea and Cr in the blood, and while urea is reabsorbed into the blood after entering the kidney (and has greater time to do so due to the reduced flow) Cr can't be and is excreted	-BUN:Cr ratio > 20:1 because back-up into the kidney allows for increased urea reabsorption	-BUN:Cr ratio < 10:1 because there is renal damage causing reduced reabsorption of BUN		-BUN:Cr ratio > 20:1 (may not be reliable)
Urinary Indices	-UA will be normal or near-normal				
Urine Na	< 20	Variable	> 20	Variable	< 20
FENa	-FENa will be < 1% (salt conserving, indicating a functioning kidney with normal physiologic response to volume depletion)	Variable, usually normal if injury is acute and there is still tubular functioning	> 1% if oliguric	< 1%	-Variable
Urine Osmolality	> 500	< 400	250-300	Variable	Variable
Urinary Sediment	-Benign or hyaline casts	-Usually normal -May see RBCs, WBCs, or crystals	-Muddy br own casts, renal tubular casts	-White cells, white cell casts, ± eosinophils	-Red cells, dysmorphic red cells and red cell casts

Nephrotic Syndrome

-Occurs when kidney damage causing pores in the podocytes allows large amounts of protein leakage, but not large enough for RBCs to pass

Etiologies
-DM
-Focal glomerulosclerosis
-Membranous nephropathy
-Amyloidosis
-Minimal change disease
-SLE
-HIV, hep B or C

Signs & symptoms
-Generalized edema: periorbital, pitting edema of the legs, pleural effusions, ascites
-Anemia due to transferrin loss
-Foamy urine

Workup
-Proteinuria ≥ 3.5 g/day
-Hypoalbuminemia
-Hyperlipidemia
-Granular or fatty urine casts with oval fat bodies and Maltese cross crystals
-Must get biopsy

Renal Vascular Disease

-S/s:
-Testing indicated in patients with clinical evidence of secondary hypertension without a cause such as primary kidney disease, primary aldosteronism, or pheochromocytoma
-Intervention is planned if a significant stenotic lesion is found

Hypertensive Nephrosclerosis	Renal Artery Stenosis
-Renal disorder associated with chronic HTN → presence of vascular, glomerular, and tubulointerstitial lesions -Risk factors: black, underlying CKD with slow deterioration of renal function, severe HTN -S/s: CKD, h/o HTN, LVH, proteinuria, hyperuricemia	-Narrowed renal arteries renal hypoperfusion → activation of RAAS → adequate renal perfusion but peripheral HTN -Disease is usually unilateral and may be due to atherosclerosis or fibromuscular dysplasia -S/s: refractory HTN, rapid deterioration of renal function -W/u: only test pts with high suspicion as procedures can be invasive (begin with US but may need arteriography) -Tx: ACEI or ARB (risk of long-term ischemic damage to stenotic kidney), revascularization procedures, considered to be a CAD equivalent = treat CV risk

FLUID AND ELECTROLYTE DISORDERS

Hypermagnesemia

-Normally 1.5-2.5

Causes
-TPN
-Renal failure
-Iatrogenic oversupplementation

Signs & Symptoms
-CNS depression
-↓ DTRs
-Respiratory failure

Management
-Similar to hyperkalemia treatment
-IV calcium gluconate
-Insulin + glucose
-Furosemide
-Dialysis

Hypomagnesemia

Causes
-TPN
-Hypocalcemia
-Gastric suctioning
-Aminoglycoside abx
-Renal failure
-Diarrhea
-Vomiting

Signs & Symptoms
-Asterixis, tremor, Chovstek's sign
-Ventricular ectopy and other dysrhythmias
-Vertigo
-↑ DTRs
-Tachycardia

Management
-Fix any hypokalemia first and Mg may correct itself!
-IV MgSO4
-PO Mg oxide if chronic

Hypophosphatemia

-Normally 2.5-4.5

Causes
-GI losses
-Meds
-Sepsis
-EtOH abuse
-Renal loss

Signs & Symptoms
-Weakness
-Cardiomyopathy
-Neurologic dysfunction
-Rhabdomyolysis
-Hemolysis
-Poor pressor response
-Respiratory failure if severe

Management
-Supplement with IV K3PO4 or Na3PO4

Hyperphosphatemia

Causes
-Renal failure
-Sepsis
-Chemotherapy
-Hyperthyroidism

Signs & Symptoms
-Ectopic calcification
-Heart block

Management
-AlOH3 to bind phosphate

Hyponatremia

Management of hyponatremia
-Treat to magic number 125
-Don't exceed replacement of sodium by more than 12 mEq/L due to risk of causing a demyelination syndrome → confusion, spastic quadriplegia, horizontal gaze paralysis
-Definitive treatment is based on underlying cause of impaired renal water excretion

Signs & symptoms
-Seizures, coma, lethargy
-Nausea/vomiting
-Weakness
-Ileus

Isotonic Hyponatremia Low serum Na Normal ECF osmolality (total solutes) and tonicity (no oncotic pressure generated)	**Hypertonic Hyponatremia** Low serum Na High ECF osmolality and tonicity	**Hypotonic Hyponatremia** Occurs when ↑ ADH → water and Na loss in urine → low serum osmolality True hyponatremia! Further characterized based on volume status		
-A kind of pseudohyponatremia -Probably due to high levels of TG or proteins (multiple myeloma) that push Na intracellularly to prevent increased serum osmolality -No treatment needed	-A kind of pseudohyponatremia -Caused by a highly osmotic molecule with glucose or mannitol in the ECF, which draws water out of cells and dilutes the Na concentration -Treat with NS until hemodynamically stable, then ½ NS	**Hypervolemic hypotonic hyponatremia**	**Euvolemic hypotonic hyponatremia**	**Hypovolemic hypotonic hyponatremia**
		-3rd spacing of fluids → reduced circulating vol (even though TBW will be hypervolemic) → activation of ADH → serum that is hypotonic because too much Na is retained in the urine -Occurs in CHF, liver failure, nephrotic syndrome, ESRD, and iatrogenic fluid overload (the most common cause of mild postop hyponatremia) -Management is water and Na restriction, diuretics, treat underlying cause of 3rd spacing	-Due to excessive ADH release -Occurs in SIADH (sodium is always down here) & paraneoplastic syndromes, postoperative hyponatremia, hypothyroidism, psychogenic polydipsia, excess beer drinking -Management is water restriction with hypertonic NaCl infusion	-Occurs when water loss causes salt loss -An extrarenal cause when kidneys are attempting to resuscitate volume by saving Na and water, such as dehydration, diarrhea, vomiting, burns, NGT suctioning, diaphoresis, or pancreatitis -A renal cause when kidneys allow high Na losses despite ↓ circulating vol, such as diuretics, ACEIs, nephropathies, or mineralocorticoid deficiencies -Manage with NS IV and correction of underlying cause

Hypernatremia

Signs & symptoms
-Lethargy
-Weakness from brain cell shrinkage
-Irritability
-Twitching
-Seizures
-Coma
-Focal intracerebral and subarachnoid hemorrhages
-Peripheral edema

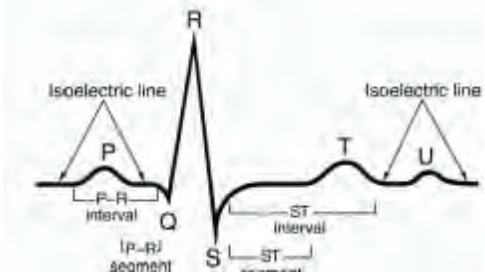
Hypovolemic Hypernatremia	**Euvolemic Hypernatremia**	**Hypervolemic Hypernatremia**	**Chronic Hypernatremia**
-Non-renal causes: excess water loss from skin or diarrhea or dehydration → concentrated urine with low Na -Renal causes: osmotic diuresis due to mannitol, glycosuria, or diuretics → salt as well as free water loss → high urine Na with normal urine osmolarity	-Non-renal causes: excessive sweating from skin or respiratory system water loss -Renal cause: due to diabetes insipidus (inadequate ADH or kidney nonresponse to ADH) -Management is to increase PO water or use IV D5W	-Non-renal causes: treatment of previous hypotonic fluid loss with higher sodium fluids, sea water ingestion, or overuse of NaHCO3 in CPR -Management is to give D5W to reduce hyperosmolality, may need dialysis if pt has renal failure	-Must correct especially slowly to prevent cerebral edema -Calculate water deficiency and accomplish correction over 36-72 hours (normal TBW = present TBW x (present serum Na/140)

Hypokalemia

Etiologies
-Decreased K+ intake
-Increased K+ entry into cells: alkaline pH, insulin, stress, epinephrine, β-agonists, ↑ RBC production, hypothermia, chloroquine toxicity
-Increase GI losses: vomiting, diarrhea, NGT drainage, laxative abuse, intestinal fistula
-Increased urinary losses: diuretics, mineralocorticoid excess, loss of gastric secretions, non-reabsorbable anions, alkalosis (causes intracellular K+ shift and increased urinary excretion of HCO3- coupled to K+), hypomagnesemia, amphotericin B, salt-wasting nephropathies, polyuria
-Increased sweat losses
-Dialysis
-Plasmapheresis
-Blood tube metabolism of K+
-Low Mg (inhibits K+ reabsorption from renal tubules)

Signs and symptoms
-Weakness
-Paresthesia
-Tetany
-Nausea/vomiting
-Ileus (very commonly seen in surgical pts)
-Exacerbation of digoxin toxicity

Workup
-EKG may show U wave, flattening of T waves, PACs, PVCs, afib



Management
-Mild hypokalemia can be treated orally with KCl supplements
-KCl IV supplementation if severe (prefer central line as K is corrosive to vasculature)
-K sparing diuretic if resistant

Hyperkalemia

-Normal K+ 3.5-5.0; critical value if > 6.5

Etiologies
-Renal insufficiency
-Meds: ACEI, ARBS, K-sparing diuretics, NSAIDS
-Mineralocorticoid deficiency
-Excessive release from cells: rhabdo, burns, tumor lysis, blood transfusion, hemolysis, acidosis, low insulin, β-blockers
-Excess intake: salt substitute, KCl infusion
-Lysis from cells after blood draw ("pseudohyperkalemia")

Signs & symptoms
-Malaise
-Palpitations & arrhythmias
-Muscle weakness
-↓DTRs
-Paresthesias
-Respiratory failure

Workup
-EKG showing peaked T waves, can go into vfib if severe

Management
-Depends on EKG findings; if there is a change treat it
-Ca gluconate or Ca chloride IV to stabilize the myocardium (give first!)
-Bicarb IV to cause alkalosis → intracellular K+ shift
-Insulin with glucose to increase K+ cellular uptake
-K+ binder (Kayexalate)
-Furosemide to renally excrete K+
-Dialysis if severe

Hypocalcemia

Etiologies
-If PTH is low → hypoparathyroidism
-If PTH is high → vit D deficiency, chronic renal failure, inadequate production of PTH or resistance, CKD, hepatic disease, osteoblastic mets, hypomagnesemia, large blood transfusion, acute pancreatitis, severe sepsis or illness, meds, pseudohypocalcemia (gadolinium interference with assay)
-Surgical: short bowel syndrome

Signs & symptoms
-Early: perioral and extremity paresthesias (areas with high innervation affected most quickly)
-Late: ↓ DTRs, AMS, hallucinations, psychosis, Chvostek's and Trousseau's signs (but don't do BP cuff in real life b/c it is extremely painful), laryngospasm, bronchospasm, prolonged QT, hypotension, HF, arrhythmia, papilledema

Workup
-Check Mg level, phosphate, vitamin D
-Need to correct Ca in face of low albumin or check ionized Ca

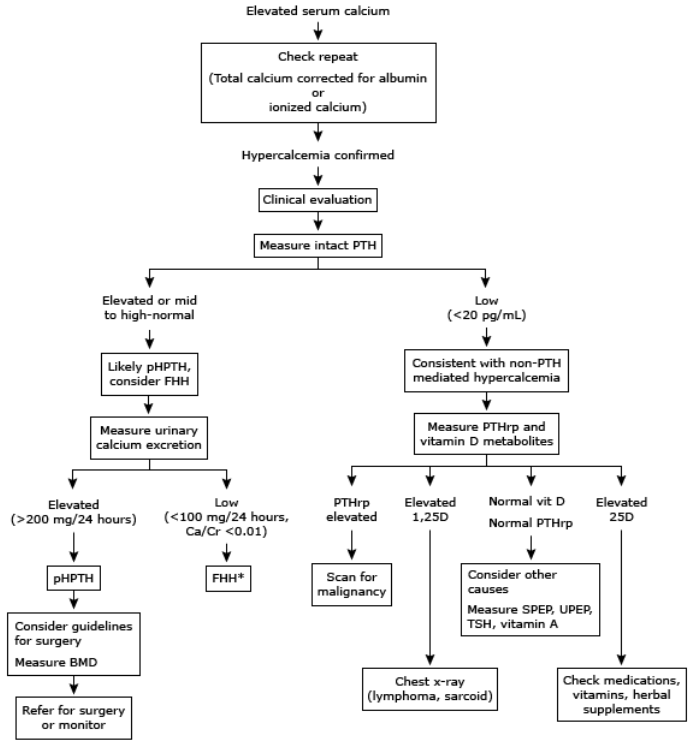
Management
-If severe or symptomatic, give Ca gluconate or CaCl3 (but beware tissue necrosis d/t Ca if IV infiltrates!), treat low Mg if present
-If asymptomatic, give oral Ca
-If due to vit D deficiency, give calcitriol (active vit D)
-Can add Ca to dialysis fluid if on HD

Hypercalcemia

Etiologies
-Increased bone resorption: malignancy/paraneoplastic effects, hyperparathyroidism, CKD, thyrotoxicosis, immobilization, Paget's disease of bone
-Increased Ca absorption: increased intake, lots of milk & Tums, elevated vit D, genetic, meds, pheochromocytoma, adrenal insufficiency, rhabdo, ARF

Signs & symptoms
-"Stones, bones, abdominal groans, and psychiatric overtones"
-Anxiety, depression, lethargy, confusion, psychosis, coma
-Constipation, anorexia, pancreatitis, PUD, abdominal pain, n/v
-EKG changes: prolonged PR and QRS, AV block, cardiac arrest
-Volume depletion, renal insufficiency, nephrolithiasis, nephrogenic diabetes insipidus, distal RTA → polydipsia and polyuria
-Muscle weakness, fatigue, bone pain, fractures

Management
-Treat if symptomatic or serum Ca is > 14
-IVF as pts tend to be volume depleted, followed by diuresis with furosemide
-Calcitonin if severe
-Bisphosphonates
-Steroids
-Dialysis is last resort



Hypomagnesemia

Etiologies
-GI loss: diarrhea, short gut syndrome, TPN
-Alcoholics
-Renal loss: diuretics, EtOH, nephrotoxic drugs, primary aldosteronism, post ATN
-Pancreatitis
-DM
-Hypercalcemia or hypophosphatemia
-Hungry bone syndrome

Signs & symptoms
-Secondary electrolyte disturbances like hypoK, hypoCa, vit D deficiency
-Weakness, anorexia, tetany, convulsions
-Widened QRS, peaked or inverted T waves, QT or PR prolongation, VT, torsades

Management
-Oral replacement with MgO2

ACID/BASE DISORDERS

Respiratory Acidosis

-Primary problem is that there is ↑ CO2 due to hypoventilation
-Kidneys respond by secreting H+, generating new bicarb and other H+ buffers, and excreting NH4+ → acidic urine, ↑serum bicarb

Respiratory Alkalosis

-Primary problem is that there is ↓ CO2 due to hyperventilation (lungs will override chemoreceptors in other parts of the body)
-Hypocapnea causes intracellar shift of K and PO4 → ↓K, ↓PO4
-Alkaline pH causes more binding of Ca to albumin → ↓Ca
-May also have hyponatremia and hypochloremia
-Kidneys compensate by increasing bicarb excretion in urine and decreased NH4+ excretion in urine → alkaline urine, ↓serum bicarb
-Seen in early asthma exacerbation, PE

Metabolic Alkalosis

-Primary problem is that there is too much bicarb (no H+ left to react with)
-Body will eventually compensate by hypoventilation to ↑ CO2, and kidneys can compensate by increasing bicarb excretion

Chloride-Responsive (urine Cl < 10 mEq/L)

Vomiting
-Occurs when vomiting/NGT → loss of HCl and vol depletion
-Vol depletion stimulates renin & aldosterone → ↑ Na reabsorption with loss of K+ and H+ (countertransporters) → low urine Na, serum hypokalemia, and alkalosis

Loop or thiazide diuretics
-Cause loss of Cl- with retention of bicarb

Chloride-Resistant (urine Cl > 20 mEq/L)

Hyperaldosteronism
-Occurs when adrenals make too much aldosterone → ↑↑ Na and bicarb reabsorption with urinary loss of K+/H+ (countertransporter pumps)

Other etiologies
-Bicarb retention
-Hypokalemia → H+ shift into cells
-Excess administration of antacids
-Weird syndromes

Metabolic Acidosis

-Initially kidney ramps up production of bicarb but then the primary problem is that bicarb is used up or there is not enough to offset the increased acid
-There is respiratory compensation by talking rapid, deep breaths (Kussmaul respirations); check to see if the pt is adequately compensating using Winter's formula

Etiologies
-Most common cause is decreased tissue perfusion/ischemia (exercise, sepsis, shock, seizures, neuroleptic malignant syndrome) → production of lactic acid from anaerobic metabolism
-Ketone bodies
-Decreased renal excretion of H^+
-Bicarb loss from kidney
-Diarrhea → loss of bicarb

Winter's formula: *Expected* $P_{CO2} = (1.5 \times bicarb) + 8 \pm 2$
-If pt's P_{CO2} corresponds, they are compensating adequately via respiration changes
-If measured P_{CO2} is higher than expected, there is also a primary respiratory acidosis
-If the measured P_{CO2} is lower than expected, there is also a primary respiratory alkalosis

Serum anion gap = diff bet + and – ions = $Na^+ - (Cl^- + bicarb)$
-Normally 8-12

Types of Metabolic Acidosis
1.) Normal anion gap (hyperchloremic anion gap)
-Occurs when Cl^- replaces lost bicarb as the H^+ buffer
-Causes (HARDUP): hyperalimentation or hyperventilation, acetazolamide, RTA (loss of bicarb from kidneys), diarrhea, ureterosigmoidostomy (loss of bicarb through colon), pancreatic fistula (loss of bicarb through colon)
2.) Increased anion gap
-Occurs when anion replacing bicarb is not one that is routinely measured, such as albumin, PO_4^{2-}, SO_4^{3-}, lactate
-Causes (MUDPILES): methanol, uremia, DKA, paraldehyde, iron ingestion/INH, lactic acidosis, ethanol (makes acetic acid), salicylates

Urine anion gap
-Only calculated in NAGMA
-Main anion is Cl-, unmeasured anions are bicarb, PO_4^{2-}, SO_4^{3-}, lactate
-Main cations are Na+ and K+, unmeasured cations are Li, Ca, Mg, and NH4+
-Normally urine is electrically neutral (UAG = 0) or slightly positive/acidic

$$UCl^- + UA = UNa^+ + UK^+ + + UC$$
$$(UA - UC) = UNa^+ + UK - UCl^-$$

-Used to estimate NH4+ levels if you don't have a direct test
-Decreased UAG (abnormally negative) when there is loss of bicarb via the bowel and kidneys respond by increasing excretion of NH4+ (= high amounts of an unmeasured anion), or after eating a protein-rich meal
-Increased UAG (higher than normal positive) when there is a renal cause blocking urinary acid excretion such as RTA because kidney is not getting rid of enough NH4+

Management
-Give $NaHCO3^-$
-Intubate for respiratory distress/can't keep up with breathing quickly

OTHER GENITOURINARY TOPICS

Enuresis

-Not clinically significant until child is > 5 years of age
-Contributing factors: nocturnal polyuria, detrusor overactivity, disturbed sleep, maturational delay, genetics, abnormal ADH secretion

Workup
-Voiding diary
-UA

Differential
-Kidney disease
-Daytime incontinence
-Constipation
-Pinworms
-Spinal dysraphism or abnormality
-Urologic anatomic abnormality

Management
-High rate of spontaneous resolution by 15 years of age
-Behavioral changes: regular voiding and emptying bladder before bedtime, no fluids after 6pm
-Rewards for voiding before bedtime, working up to rewards for staying dry overnight
-More active interventions needed as child gets older, social pressures increase, and self-esteem is affected
-Enuresis alarms for wetting > twice per week
-Desmopressin for children with nocturnal polyuria and normal bladder capacity who have failed alarm trials

Male Circumcision

-Currently promoted as the health benefits outweigh the risks: reduced UTIs, reduced STI transmission, reduced penile inflammatory and retractile disorders, easier hygiene
-Procedural risks are rare

-Not covered by Medicaid and typically costs $200 out of pocket
-Uncircumcised infants will need parent education on how to care for and clean the penis regularly to prevent phimosis

NEUROLOGIC SYSTEM

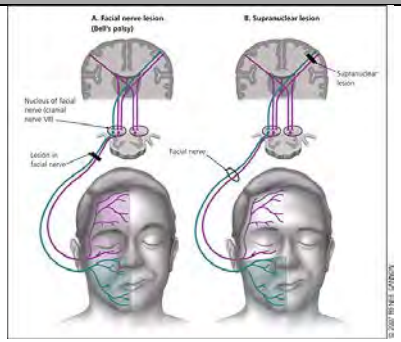
DISEASES OF PERIPHERAL NERVES

Bell's Palsy

Etiologies
-Usually idiopathic
-Virus
-Lyme
-Sarcoid
-HIV
-Tumors
-HSV reactivation

Signs & symptoms
-Abrupt onset that may progress over days
-Motor deficits: facial nerve paralysis, ptosis
-Sensory deficits: ear pain, taste differences, hyperacusis

Workup
-Must differentiate peripheral cause from central (stroke) → central cause will result in partial sparing of the frontalis muscle because there is bicortical input from the brain, so half the input is still functioning
-If uncertain refer for head CT to r/o stroke or TIA
-Lyme titer
-EMG/NCS will indicate severity but won't guide treatment



Management
-Controversial!
-Prednisone taper
-Artificial tears
-Acyclovir

Prognosis
-60% recover completely
-10% have permanent dysfunction

Complex Regional Pain Syndrome

-Pathophysiology not well understood

Reflex sympathetic dystrophy = old term for CRPS where there is no definable nerve lesion, now called CRPS type II

Causalgia = named peripheral nerve injury is present, now called CRPS

Etiologies
-Usually follows a minor or major injury such as soft tissue injury or fracture
-Post-surgical
-Post-stroke or MI
-May have no precipitating event
-Emotional stress can contribute

Prevention
-Early mobilization following stroke or fracture
-Supplemental vit C for wrist fractures

Signs & symptoms
-Severe burning or throbbing pain with low threshold or normal stimuli
-Swelling and limited ROM
-Vasomotor instability → altered skin temperature, can be warm or cold
-Diaphoresis
-Skin changes, thickening
-Loss of muscle strength
-Patchy bone demineralization
-Urinary problems

Differential
-Spinal nerve root impingement
-Pancoast syndrome
-Thoracic outlet syndrome
-Vasculitis
-RA
-Peripheral neuropathy
-Migratory osteolysis
-Venous thrombosis
-AV fistula
-Systemic sclerosis
-Diffuse atrophy
-Angioedema

Workup
-Diagnosis is clinical
-Radionuclide bone scan shows ↑ uptake
-MRI
-Sx will show good response to sympathetic nerve block

Management
-Stage 1 disease: topical capsaicin, TCA, calcitonin, NSAID daily; refractory pain → trigger point injections with local anesthetic and steroids
-Stage 2 disease: oral steroids
-Stage 3 disease → pain center referral

Peripheral Neuropathy

Common signs & symptoms
-Weakness, incoordination, ataxia, muscle wasting, numbness, tingling, loss of sensation, pain, ataxia, dizziness, loss of consciousness, exercise intolerance, difficulty digesting foods, constipation, urinary symptoms, sexual dysfunction, visual symptoms
-Axonal disease: sensory symptoms > motor symptoms, greater distal weakness, ↓ DTRs
-Demyelinating disease: motor symptoms > sensory symptoms, greater proximal weakness, ↓ DTRs

Workup
-Try to localize the lesion: unilateral extremity affected → brain or entire nerve plexus; symmetric disease → bicortical, brainstem, cord lesion, or peripheral neuropathy; portion of limb or trunk affected → brain, spinal cord, plexus, or peripheral nerve; dermatome or myotome affected → specific spinal cord segment
-EMG/NCS
-B12, CBC, glucose tolerance, RPR, CMP, serum protein electrophoresis, TSH
-Select patients: anti-Hu, ESR, ANA, RF, SS-A, SS-B, HMSN, HIV, Lyme, phytanic acid, 24 hour urine for heavy metals, referral for CSF sample or nerve biopsy

Management
-Acute focal neuro deficit → send to ED for neuro consult
-Subacute or chronic focal neuro deficit → refer for EMG/NCS (and neuro consult if at an unusual site)
-Multifocal deficit → refer to neuro for EMG/NCS
-Symmetric deficit → refer to neuro for EMG/NCS

Single Peripheral Neuropathies

Carpal tunnel: median nerve compression
+ Phalen's, + Tinel's
-Worse at night
-Late thenar atrophy
-Wrist splint

Ulnar neuropathy
-Worsened by elbow flexion or wrist extension
-Modify elbow or wrist activities
-Extensor splint at nighttime

Radial neuropathy
-Axilla crutch injury
-Saturday night palsy
-Humeral fx
-Motor deficits > sensory
-Splints, PT, OT

Meralgia paresthetica: compression of lateral femoral cutaneous nerve
-Risk: obesity, tight clothes, pregnancy, lumbar lordosis, DM
-No motor symptoms
-Pain and numbness on outer thigh

Femoral neuropathy
-Risk: lithotomy position, femoral artery cath, DM neuropathy
-Sensory deficits over thigh and leg to medial malleolus
-Weakness and atrophy of quads
-Knee buckling
-Depressed patellar DTRs

Sciatic nerve palsy/sciatica
-Risk: DJD, hip replacement, spinal stenosis, lumbar disc herniation
-Weakness with leg flexion, foot dorsiflexion, foot eversion
-Depressed ankle DTRs
-Sensory deficits over posterior thigh, leg, and foot
-EMG to distinguish from peroneal neuropathy
-X-rays

Multiple Peripheral Mononeuropathies

Discogenic neuropathies: impingement of spinal nerve by lateral disc protrusion or arthropathy
-Motor, sensory, and autonomic dysfunction
-MRI, CT myelogram
-EMG/NCS
-Rest, immobilization, PT, surgical decompression

Plexopathies: cervical, brachial, lumbar, sacral
-Risk: trauma, DM, radiation
-Motor, sensory, and autonomic dysfunction
-EMG/NCS

Mononeuritis multiplex

Peripheral Polyneuropathies

Hereditary
-Charcot-Marie-Tooth disease types

Endocrine
-Diabetic peripheral neuropathy and related Charcot arthropathy
-Uremic peripheral neuropathy
-Alcohol and nutrition deficiency peripheral neuropathy
-Infectious peripheral neuropathy
-HIV-related

Inflammatory

Toxic
-From exposure to neurotoxins like pesticides, heavy metals, mds

Metastatic polyneuropathies
-Invasion of plexus or peripheral nerves by malignant cells
-Treat with radiation

Paraneoplastic peripheral neuropathies
-Immune response to neoplasm

Peroneal (common fibular) nerve palsy	-Risk: leg crossing, trauma to knee, fx of fibula, tight casts, high boots -Weakness on dorsiflexion and foot eversion -Paresthesias over anterolateral calf and top of foot -EMG to distinguish from sciatic nerve palsy -Splints, PT		**Critical illness-related neuropathies**	-Associated with ICU admission, sepsis, multi-organ dysfunction, difficulty weaning from ventilator
Tibial neuropathy	-AKA tarsal tunnel syndrome -Rare			

HEADACHES

Pediatric Headaches

Etiologies	**Workup**	**Management of chronic headaches**	**When to refer**
-Acute & localized: URI, other viral infection, post-traumatic, dental abscess, TMJ dysfunction, brain abscess, first migraine -Acute & generalized: fever, systemic infection, CNS infection, HTN, CH, exertional, first migraine, trauma, toxins, meds -Acute & recurrent: migraine, cluster headache -Chronic & nonprogressive: tension headache, psychiatric issue, post-traumatic, postconcussive, medication overuse -Chronic & progressive: idiopathic intracranial HTN, space-occupying lesion, post-traumatic, postconcussive	-CT without contrast or MRI indicated for kids with headaches and neuro signs or symptoms suggestive of intracranial pathology = headache that awakens child during night or occurs upon waking, sudden severe headache, persistent nausea or vomiting, AMS, ataxia, headache worsened by cough, urination, or defecation, absence of aura, chronic and progressive headaches, change in headache quality, severity, or frequency, occipital headache, recurrent localized headache, lack of response to medical therapy, cranial bruits, growth abnormalities, papilledema or retinal hemorrhages, age < 3	-Provide realistic expectations for medical interventions -Plan for return to school -Avoid triggers: lack of sleep, dehydration -Address comorbid problems: insomnia, mood problems, anxiety	-Headaches associated with mood disturbance or anxiety -Uncertain diagnosis -Headaches refractory to primary care management -Chronic daily headaches

Pediatric Migraine Headaches

Signs & symptoms	**Workup**	**Management**
-May be shorter in kids, as short as 1 hour -Toddlers: pallor, decreased activity, vomiting, sensitivity to light and noise -Nausea, vomiting, abdominal pain, desire to sleep -Complicated migraine symptoms: hemiplegia, ophthalmoplegia, tinnitus, vertigo, ataxia, weakness, confusion, paresthesias	-Imaging to rule out more serious causes is indicated for complicated-type symptoms and for occipital location	-Initial abortive therapy with acetaminophen or ibuprofen -Antiemetic like promethazine for nausea & vomiting -2nd line is abortive therapy with triptans -Begin prophylactic treatment if > 4-5 migraines per week → cyproheptadine for kids under 6, propranolol for older kids, amitriptyline for concomitant depression -For menstrual migraines, naproxen BID just before beginning period has been shown to be beneficial

Pediatric Tension Headaches

Signs & symptoms	**Differential**	**Workup**	**Management**
-Bilateral pressing tightness -Non-throbbing -Lasts hours to days -May have sensitivity to light and noise -Daily activities typically not affected -Not aggravated by walking stairs or similar routine activity	-Migraine without aura -Increased ICP -Tumor -Infection	-Imaging has a low yield but may be needed to relieve parental apprehension or with sleep-related headache, no FH of migraine, presence of vomiting, absence of visual symptoms, headache of < 6 months duration, confusion, or abnormal neuro exam	-Minimize stress -Acetaminophen or NSAIDs for infrequent use -If prophylactic therapy is needed, can use amitriptyline -Psychotherapy, relaxation techniques, and biofeedback techniques about as successful as amitriptyline

HEADACHES

Tension Headache

-Most common type of headache

Signs & symptoms
-No aura, nausea, or vomiting
± Photophobia and phonophobia
-May have muscle tender points
-Bilateral head pain "like a tight hat"
-Steady, non-pulsating pain that is dull, tight, or vise-like
-Mild to moderate pain intensity
-Normal physical activity does not aggravate

Management
-DOC are OTC analgesics: ibuprofen, naproxen, aspirin, acetaminophen, Excedrin tension headache
-Consider prophylaxis if > 2 days/week with TCAs (amitriptyline or nortriptyline) or duloxetine if there is comorbid depression (at least 8 weeks)
-Muscle relaxants should be taken at first sign of headache: cyclobenzaprine, methocarbamol, tizanidine
-Trigger point Botox injections

With tension and migraine headaches, watch for signs of hemicrania continua (daily unilateral headache with miosis, ptosis, eyelid edema, lacrimation, nasal congestion, rhinorrhea), which can transform from migraine or tension headaches and is prompted by medication overuse; responds only to indomethacin!

Migraine Headache

-Highest prevalence in 25-45 year olds with decreased incidence during childbearing years
-May have genetic component incurring hypercoagulability
-High incidence of comorbid depression
-Precipitators: stress, hormones, hunger, sleep deprivation, odors, smoke, alcohol, meds, high tyramine foods
-High incidence of PFO with migraines with aura

Signs & symptoms
-May have prodrome of sensitivity to touch or combing hair
-May have aura up to 1 hour before, most commonly scintillating scotoma, followed up visual, sensorimotor, speech, or brainstem disturbances
-Nausea, vomiting, photo and phonophobia, unilateral pulsating frontotemporal pain
-May correlate with menstrual cycle

Management
-If related to menstrual cycle, prophylax with 2-7 days of NSAIDS prior to menses and continue through last day of flow, consider OCPs as long as there is no aura
-Exercise shown to be just as good as meds
-Refer when symptoms are refractory to treatment, worsening disability, comorbid conditions requiring polypharmacy, rebound headaches, symptoms no longer fitting diagnostic criteria

Pharmacologic Therapy

Abortive	**Non-opioids**: NSAIDs, acetaminophen, rectal indomethacin, IM ketorolac, Excedrin migraine **Triptans:** constrict intracranial bood vessels, interrupt pain transmission centrally -Never use during an aura due to risk of stroke -Sumatriptan, zolmitriptan (wafer avail), etc. -AEs: paresthesias, dizziness, flushing, somnolence, rebound HA with overuse	**Ergots**: direct smooth muscle vasoconstrictors, non-selective 5-HT1-R agonists -Ergotamine -Dihydroergotamine: available as injection, nasal, rectal, SL -AEs: fibrosis with long-term use, rebound headache, paresthesias
Prophylactic	-Consider with migraines > 2 per week, incomplete response to acute therapies, or patients with rebound headaches using acute treatments -Must give 6-8 week trial for each therapy -Prolonged headache-free intervals can signal time for dose reduction or d/c with slow taper **β-blockers** -Propranolol, timolol, metoprolol, nadolol, atenolol -Helpful in patients with comorbid anxiety, HTN, or angina **CCB:** prevent vascular spasm -Verapamil -Take up to 8 weeks to work -β-blockers work better **TCAs** -Amitriptyline (best evidence), imipramine, doxepin, nortriptyline -AEs: tremor, weight gain, anticholinergic	**SSRIs** -Fluoxetine, fluvoxamine, sertraline -Not as much data **Atypical antidepressants** -Bupropion -Venlafaxine -Not much data **Anticonvulsants** -Carbamazepine, gabapentin, tiagabine, topiramate, valproate, oxcarbazepine, lamotrigine, vigabatrin, zonisamide **Botox**

Cluster Headache

-More common in men

Signs & symptoms
-Unilateral, excruciating, steady pain in the eye, periorbital region, or temple
-Increased swelling on ipsilateral side of face and eyelid
-Ipsilateral miosis or ptosis
-Ipsilateral nasal congestion or rhinorrhea
-Lasts 15-180 minutes untreated
-Occurs in bouts up to every other day or up to 8 attacks daily for weeks at a time followed by remission for months or years
-Can be precipitated by sleep, occurring 90 minutes after falling asleep
-Patient may complain of "worst headache of life"

Management
-Abortive therapy: 100% O2 on a non-rebreather @ 6-12 L/min for 15 min, SQ or nasal sumatriptan, octreotide, nasal lidocaine
-Prophylaxis: DOC is verapamil (takes 8 weeks to work), Li, ergotamine, prednisone taper, nerve block

Thunderclap Headache

-Refers to sudden onset headache that is severe
-Patients may also complain of "worst headache of life"

-If features do not fit cluster headache, must send to ED to r/o ICH

INFECTIOUS DISORDERS

Encephalitis

-Represents an infection of the brain itself
-May be primary or postinfectious

Differential
-Meningitis
-Meningoencephalitis
-Stroke

Agents
-HSV
-Rabies virus
-WNV

Signs & Symptoms
-Fever
-Headache
-Lethargy, confusion, AMS (what usually differentiates it from meningitis)
-Seizures
-Can have focal neuro abnormalities
-No meningeal signs like photophobia or nuchal rigidity

Workup
-CT or MRI to investigate space-occupying lesions, brain abscess, demyelination, areas of abnormalities
-CSF shows lymphocytic predominance
-PCR for HSV
-Serum for WNV
-Test for other agents depending on travel or exposure history

Management
-Empiric treatment with acyclovir until results come back

Meningitis

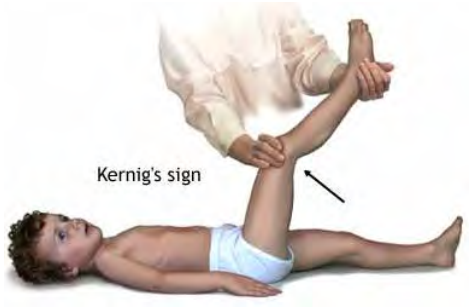
-Represents an infection of the arachnoid mater and CSF

Group	Agents	Empiric Drugs
Under 1 mo	GBS, *E. coli, Listeria, Klebsiella, Enterobacter*	-First 3-6 days of life: ampicillin (cover *Listeria*) + gentamicin (together cover enterococci)
1 mo to 50 years	-#1 cause is *Strep pneumo* -Greater prevalence of *Neisseria meningitidis* in ages 0-5 and 14-21	-Cefotaxime (cover normal *Strep pneumo, H.flu, M. cat, Neisseria*) + vanco (cover MSSA/MRSA, PRSP) -Can sub ceftriaxone for cefotaxime
Over 50	-*Strep pneumo* still #1 but also consider *Listeria*	-Cefotaxime (cover normal *Strep pneumo, H.flu, M. cat, Neisseria*) + vanco (cover MSSA/MRSA, PRSP) + ampicillin (cover *Listeria*) -Can sub ceftriaxone for cefotaxime
Impaired cellular immunity (lymphoma, cytotoxic chemo, steroids)		-Vanco + ampicillin + cefepime (to cover *Pseudomonas* and other gram negs) -Can sub meropenem for cefepime

Signs & Symptoms
-Bacterial: fever, nuchal rigidity, AMS, severe HA
-Listeria: more likely to have seizures and focal neuro deficits
-Viral: severe HA, fever, photophobia, no focal neurologic signs
-Meningococcal: petechial rash, palpable purpura, DIC, arthritis
-TB: gradual onset with listlessness and irritability, CN palsies
-Brudzinski's and Kernig's signs have low sensitivity but high spec

Workup
-CT before LP to assess for ↑ICP ONLY if immunocompromised, h/o CNS disease, new seizure, papilledema, altered LOC, focal neuro deficit

Any; typically younger	-Viral or "aseptic meningitis" -Usually enteroviruses -Also Coxsackie, ECHO, mumps, HSV, HIV -Rodent exposure: LCMV -Tick exposure: Lyme, RMSF, ehrlichia -Mosquitoes: WNV, St. Louis encephalitis -Sex: syphilis	Acyclovir or valacyclovir if suspecting HSV	-Otherwise don't delay LP -Gram stain of CSF -CBC shows leukocytosis or leukopenia, poss thrombocytopenia -Get 2 sets of blood cx before starting abx -HIV if suspecting viral cause -Remember that a patient who has recently received abx and has a bacterial meningitis may have milder lab findings more suggestive of an aseptic meningitis **Management** -Begin empiric abx or acyclovir after LP if suspecting bacterial meningitis or HSV meningitis -Give dexamethasone with abx if bacterial meningitis -Meningococcal contacts need prophylaxis -Tailor abx to results of gram stain and cultures
Any	-Subacute or chronic meningitis -Could be viral, bacterial, fungal, or parasitic -HIV, TB, syphilis, late stage Lyme, *Cryptococcus, Histoplasma, Coccidioides*, cysticercosis		



Cerebrospinal fluid analysis in central nervous system infection

	Glucose (mg/dL)		Protein (mg/dL)		Total white blood cell count (cells/microL)		
	<10*	10-45•	>250Δ	50-250◇	>1000	100-1000	5-100
More common	Bacterial meningitis	Bacterial meningitis	Bacterial meningitis	Viral meningitis Lyme disease Neurosyphilis	Bacterial meningitis	Bacterial or viral meningitis TB meningitis	Early bacterial meningitis Viral meningitis Neurosyphilis TB meningitis
Less common	TB meningitis Fungal meningitis	Neurosyphilis Some viral infections (such as mumps and LCMV)	TB meningitis		Some cases of mumps and LCMV	Encephalitis	Encephalitis

MOVEMENT DISORDERS

Benign Essential Tremor

-AKA familial tremor if there is a FH

Signs & symptoms
-Involvement of one or both hands, the head, or hands + head
-May affect manual skills or speech
-Unusual to have in legs
-May be enhancement with emotional stress
-Often relieved with alcohol

Workup
-No other abnormalities
-Refer to neurology to r/o other causes

Management
-Propranolol if symptoms are disabling
-Primidone is 2nd line therapy
-Other options: alprazolam, topiramate, gabapentin, mirtazapine
-Botox

Huntington's Disease

-Abnormal CAG repeats on chromosome 4 → loss of caudate and squared-off lateral ventricles
-Onset will occur between ages 20-40, which can be affected by anticipation

Signs and symptoms
-Usually begins with a psychiatric disorder
-Subcortical dementia, chorea, dystonia, motor impersistence, incoordination, gait instability, depression, anxiety, impulsivity, apathy, OCD, athetosis

Workup
-Diagnosis is clinical
-CT or MRI will show cerebral atrophy and loss of caudate

Management
-Symptomatic
-Give dopamine-R blockers for chorea (like a DA-excess state) such as haloperidol or risperidone
-SSRIs for depression and anxiety

Prognosis
-Death within 10-15 years of onset of symptoms

Parkinson's Disease

-Degeneration of CNS due to death of DA-generating cells in the substantia nigra and accumulation of Lewy bodies in neurons
-Risk factors: age, exposure to synthetic heroin byproduct MPTP, manganese exposure, flu epidemic

Etiology
-Most cases are idiopathic
-5% of cases are hereditary

Signs & symptoms
-Most symptoms are unilateral (if bilateral the cause is more like to be an exposure)
-Pill-rolling resting tremor, cogwheel rigidity, shuffling steps, difficulty initiating movements with festinating gait (unwanted acceleration after walking commences)
-Bradykinesia, fatigue, stooped posture, masked facies
-Memory loss
-Small, cramped handwriting

Differential
-Lewy body dementia and other neurodegenerative disorders
-Essential tremor
-Secondary parkinsonsism (usually a drug reaction)

Workup
-Diagnosis is usually clinical
-Head MRI to rule out brain lesions
-Genetic testing is available but should only be performed after careful consideration due to inconclusive testing results and lack of specific treatment for the disease

Management
-Treatment should begin when patient experiences functional impairment

Pharmacologic Therapy

Synthetic DA: DA precursor that can cross the BBB to replace deficit	**DA Agonists:** act directly on DA-R in the corpus striatum	**COMT Inhibitors:** prevent breakdown of levodopa by another pathway	**MAO-B Inhibitors:** inhibit metabolism of dopamine	**Others**
-Levodopa -Must also give carbidopa to prevent peripheral conversion -Can be given to help diagnose Parkinson's -First-line therapy in older patients (> 65) as effectiveness ↓ over time -Won't help with postural instability, dementia, autonomic dysfunction, or "freezing" -Won't stop disease progression -AEs: n/v, anorexia, postural hypotension, arrhythmias, mental disturbance, dyskinesias, overactivity, agitation	-Bromocriptine, pramipexole, ropinirole, apomorphine -First-line therapy for younger patients with milder disease -Can delay need for levodopa -Can be add-on to levodopa therapy -AEs: cardiac valve fibrosis, dizziness, HA, insomnia or somnolence, fonusion, hallucinations, hypotension, syncope, impulsivity	-Entacapone, tolcapone -Allows for decreased levodopa dosage -AEs: dyskinesias, nausea, dizziness, hallucinations, abd pain, diarrhea, orthostasis, somnolence, HA	-Selegiline, rasagiline -Can help delay need for levodopa -AEs: insomnia, nausea	-Amantadine: antiviral that increases DA release from nerve terminals -Anticholinergics: block Ach -Vit E supplementation not shown to be beneficial

VASCULAR DISORDERS

Cerebral Aneurysm

-Baseline prevalence of 0.2-6% in adults
-Generally low risk of rupture

Risk factors
-Ehlers Danlos
-Polycystic kidney disease
-Bicuspid aortic valve
-Aldosteronism

Screening
-Not indicated in the general population
-Consider screening of individuals with 2+ first-degree relatives with known cerebral aneurysm with MRI every 3-5 years

Management
-Treatment depends on patient age, severity and progression of symptoms, and available alternatives
-Endovascular techniques associated with lower mortality than surgical clipping
-Monitoring via CTA or MRA annually for 2-3 years, then every 2-5 years if stable
-Risk reduction: quit smoking, avoid heavy alcohol consumption, stimulants, illicit drugs, and heavy lifting and straining

Stroke

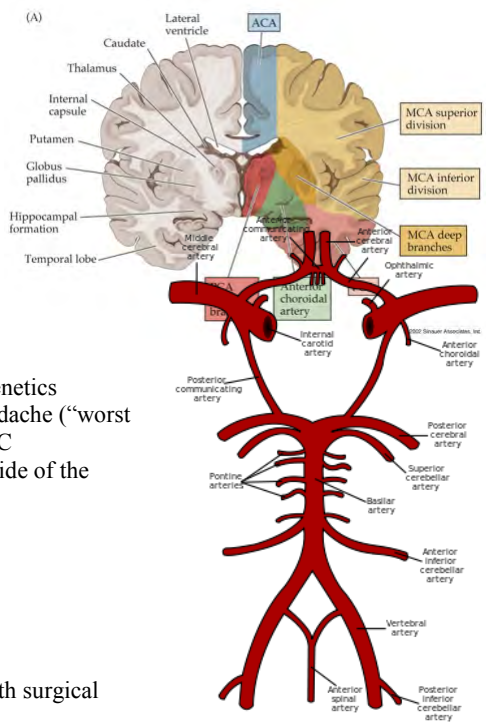
-An acute neurological deficit of vascular etiology with symptoms lasting > 24 hours
-More prevalent in the "stroke belt" in SE US

Differential: transverse myelitis, Bell's palsy, Gullain-Barre, myasthenia gravis, TIA

Hemorrhagic Stroke (Intracerebral or Intracranial Hemorrhage)

Accounts for 15-20% of strokes

Parenchymal ICH
-Bleeding within the brain itself
-Primary if due to spontaneous rupture of small vessels damaged by chronic HTN or amyloid angiopathy
-Secondary if due to trauma, vascular abnormalities, tumors, impaired coagulation, or vasculitis
-Presentation will be severe HTN, bad HA, n/v, focal neuro deficits
-If in thalamus or basal ganglia → contralateral motor and sensory deficit, aphasia, language or spatial neglect, depressed LOC due to mass effect, intraventricular extension → hydrocephalus
-If in the cerebellum → ipsilateral ataxia, depressed LOC
-If in the pons → vertigo, diplopia, crossed signs, depressed LOC



Subarachnoid hemorrhage
-Bleeding outside the brain
-Most common cause is a ruptured aneurysm, usually of the anterior communicating artery, but can also occur at the bifurcation of the carotid, PCCM, MCA, basilar tip artery, or PICA
-Less common causes are vasculitis, infection, neoplasms, or blood coagulopathies
-Risk factors: heavy alcohol, smoking, HTN, genetics
-Presentation will be with an abrupt, severe headache ("worst HA of life"), meningismus, may have rapid LOC
-Neuro exam will be nonfocal because it is outside of the brain

Workup
-CT will show white in area of bleed
-If SAH, LP will show xanthochromia

Management
-Parenchymal ICH is managed supportively, with surgical consult if there is mass effect
-Subarachnoid hemorrhage typically requires surgical intervention

Ischemic Stroke

-Accounts for 80-85% of strokes

Atheroembolic
-Occlusion of artery supplying the brain due to CAD, stenosis, or cholesterol embolus
-The most common kind of stroke
-There will be warning signs with a stepwise progression to full stroke, such as transient language disturbances and weakness
-Ophthalmic artery occlusion → amaurosis fugax
-ACA occlusion → weakness and sensory loss in contralateral leg, may have mild weakness of the arm
-MCA occlusion → contralateral hemiplegia, hemisensory loss, homonymous hemianopia with eyes deviating towards affected side, global aphasia if in dominant hemisphere,
-Lacunar stroke: a kind of atheroembolic stroke where one of the penetrating arteries providing blood to the brain's deep structures is occluded → can appear as pure motor strokes, pure sensory strokes, ataxic hemiparesis, or dysarthria + clumsy hand
-Dilated pupils with vertebrobasilar stroke

Cardioembolic
-Embolus thrown from the heart goes to the brain; can then break up into many clots and travel to multiple vascular territories
-Can also cause a hemorrhagic infarction as the ischemic blood vessels die and split open (will want to differentiate this from primary ICH, because if it's primary you can't give blood thinners or lytics ever again but if the hemorrhage is secondary to blood clot you want to put the pt on blood thinners to prevent future embolic strokes)
-Sources: afib, cardiomyopathy, acute MI, valvular heart disease
-Most commonly lodges in the middle cerebral artery

Workup
-Head CT: warning! will look normal until several hours into stroke

Management
-Start TPA if within 4.5 hours of symptom onset (this is the cutoff point for prevention of disability) after cleared by head CT
-TPA relative contraindications, within 3 hours of symptom onset: recent head trauma or stroke, prior ICH, recent arterial puncture, active bleeding or acute trauma, on oral anticoags with hi INR, low platelets, hypoglycemia, HTN > 185/110, CT with hypodensity in > 1/3 of cerebral hemisphere, rapidly improving symptoms, seizure with postictal impairment, recent MI, recent major surgery
-TPA further contraindications if > 3 hours after symptom onset: age over 80, oral anticoags, h/o prior stroke + DM
-Consider endovascular repair or clot removal if TPA is not an option, and give aspirin instead
-Avoid D5W as glucose crosses into the brain and is quickly metabolized → edema
-Only lower BP if > 220/120, and only by 15-20% the first day, otherwise avoid treating HTN for at least 2 weeks to avoid further cerebral ischemia
-Hypothermia induction therapy?
-Antithrombotic agents
-Later PT, OT, speech therapy

Prognosis
-Everyone improves to some degree after a stroke
-Fastest period of recovery is the first 30-60 days afterwards

Transient Ischemic Attack

-An acute focal neurologic deficit as a result of ischemia that resolves within 24 hours
-Can be caused by brain, spinal cord, or retinal ischemia

Differential
-Seizure
-Migraine with aura
-Syncope
-Hypoglycemia
-Encephalopathy
-Multiple sclerosis

Workup
-CBC, BMP to r/o metabolic causes
-Lipids
-Brain imaging to r/o hemorrhage or cerebral tumor
-Neurovascular imaging: carotid US, CTA if needed
-Cardiac eval: EKG, echo if there is suspected endocarditis

Management
-Risk reduction: smoking cessation, statins, BP control
-Antiplatelet therapy indicated for noncardioembolic attacks: aspirin or clopidogrel (only marginally better than ASA)
-Anticoagulant therapy indicated for pts with concurrent afib
-Carotid endarterectomy for select patients

Prognosis
-Incurs greater risk of stroke in the future, can use $ABCD^2$ criteria (age, BP, clinical features, duration of sx, DM) to estimate risk

Subdural Hematoma

-Usually due to tearing of bridging veins on brain surface to dorsal sinuses
-Can also occur from arterial rupture

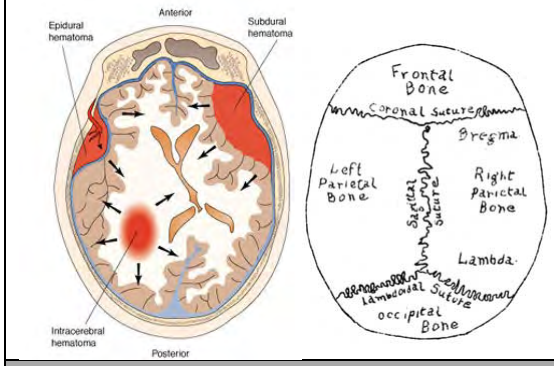
Signs & Symptoms
-HA, vomiting, dysphagia, anisocoria, CN palsies, nuchal rigidity, ataxia
-H/o acceleration/deceleration injury
-Usually acute but can become chronic
-Behavioral changes such as apathy, cognitive impairment with chronic SDH

Workup
-Noncontrast head CT will show crescent-shaped hematoma because it extends beyond the suture lines

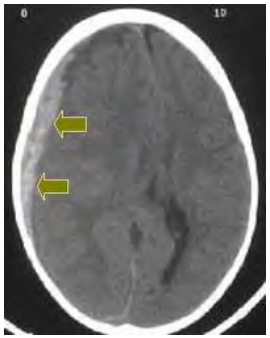
Management
-ABCs and resuscitation
-C-spine immobilization
-Short-term hypoventilation in order to ↓pCO2 → ↓ICP by cerebral vasoconstriction
-Mannitol or hypertonic saline to ↓ICP
-Seizure prophylaxis
-May require surgery
-ICU admission with serial head CTs

Prognosis
-More severe injury than epidural; mortality of those requiring surgery is 40-60%

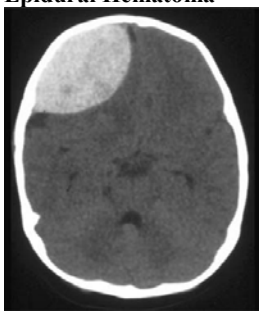
The textbook presentation of an **epidural hematoma** is that there will be a lucid interval followed by a loss of consciousness, however in reality EITHER a **subdural** or an **epidural** can have a lucid interval → LOC; the only difference is that the ***decompensation to coma may be slower in a subdural



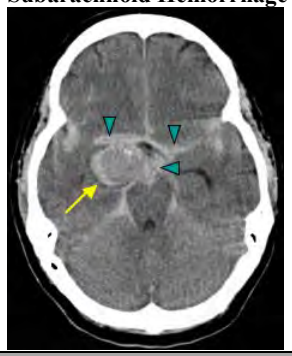
Subdural Hematoma



Epidural Hematoma



Subarachnoid Hemorrhage (Stroke)



Epidural Hematoma

-Due to tearing of middle meningeal artery
-Rarely seen in kids < 2 and in the elderly as the dura is firmly attached in these ages

Signs & Symptoms
-HA, vomiting, confusion/lethargy, aphasia, seizures, hemiparesis
-Unconsciousness, abnormal pupil reactions to light, or abnormal posturing due to compression of CN by hematoma
-Usually coexists with a skull fracture

Workup
-Noncontrast head CT shows hematoma that does not cross suture lines, brain parenchyma may be compressed to the midline

Management
-Usually requires craniotomy with evacuation of bleed

OTHER NEUROLOGIC DISORDERS

Cerebral Palsy

-A group of nonprogressive clinical syndromes characterized by motor and postural dysfunction

Etiologies
-Most cases are prenatal due to prematurity, intrauterine growth restriction, intrauterine infection, antepartum hemorrhage, placental pathology, or multiple pregnancy
-Perinatal hypoxia or ischemia
-Perinatal stroke
-Low birth weight

Presentations
-Spastic CP: an UMN syndrome with slow effortful voluntary movements, impaired fine-motor function, difficulty in isolating individual movements, and fatigability
-Dyskinetic CP: usually a result of severe perinatal asphyxia; encephalopathy characterized by lethargy, decreased spontaneous movement, hypotonia, suppressed primitive reflexes, later athetosis, chorea, and dystonia
-Ataxic CP: ataxic movements and speech, widespread disordered motor function; a diagnosis of exclusion
→ Frequently accompanied by other disorders of cerebral function such as intellectual disability or learning disability, behavioral and emotional disorders, seizures, impaired vision or speech
→ Also may have secondary consequences such as poor growth and nutrition, orthopedic problems, osteopenia, and urinary disorders

Signs & symptoms
-FH of the disease
-Loss of developmental milestones
-Ataxia, involuntary movements, oculomotor abnormalities, muscle atrophy, or sensory loss
-Hypotonia associated with weakness
-Rapid deterioration of neuro signs

Workup
-Glucose, ammonia, lactate, pyruvate, ABG, and other studies are needed to exclude a metabolic disorder
-Requires a constellation of findings including motor delay, neurologic signs, persistence of primitive reflexes, and abnormal postural reactions
-Diagnosis may require serial exams and is not possible until later infancy; CP is a diagnosis of exclusion
-Brain MRI to determine site of lesion

Management
-Multidisciplinary team needed
-Botox for joint contractures
-Regular x-ray screenings for hip dysplasia
-Physical therapy to reduce muscle tone
-May need gastrostomy tube

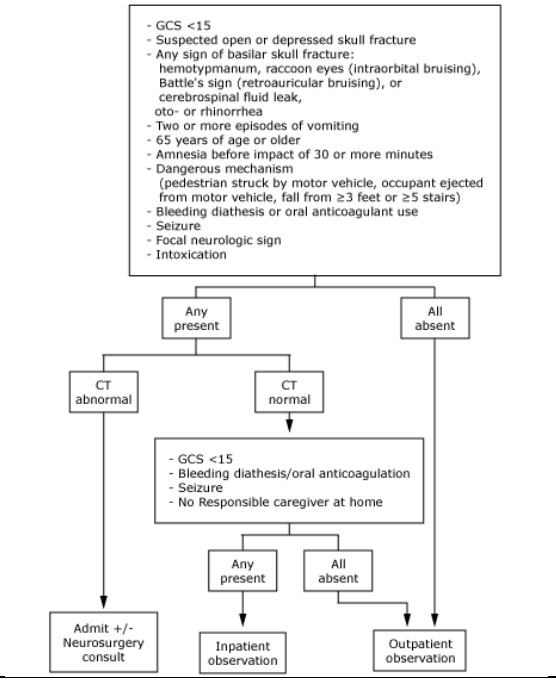
Prognosis
-CP lesion will be static but clinical signs may evolve as the nervous system matures

Concussion

-Considered to be a mild TBI as a result of blunt force or acceleration/deceleration injury

Signs & symptoms
-Often involves brief loss of consciousness
-Confusion or amnesia
-HA
-Dizziness
-N/v
-Seizures within first week after injury
-Hours to days after: mood and cognitive disturbances, photo and phonophobia, sleep disturbances

Acute evaluation and disposition of patients with mild TBI



Management
-Avoid further contact sports as repeat TBI soon after initial injury can lead to cerebral edema
-ED workup indicated for LOC > 1 min, concern for C spine injury, high risk for intracranial bleed, possible skull fracture, or worsening of patient's condition
-NSAID analgesics (don't want to affect cognition)

Sequelae
-Post-concussive syndrome: HA, dizziness, neuropsychiatric symptoms, cognitive impairment; generally resolve within a few weeks to months
-Epilepsy
-Vertigo

Dementia

-The most common cause of dementia is Alzheimer's disease
-Other major types are Lewy body dementia, frontotemporal dementia (more prominent personality and behavioral changes), vascular dementia, and Parkinson-associated dementia
-Dementia is characterized by memory impairment and impairment in one other cognitive domains: aphasia, apraxia (clock drawing), agnosia (inability to interpret sensations correctly), executive function (planning, organization, abstract thinking)

Screening
-USPSTF grade I

Signs & symptoms
-Insidious appearance and progression of cognitive deficits, usually beginning with language function and visuospatial skills
-Pt will not usually report memory loss but family members will
-Later deficits in executive function and behavior
-Noncognitive neurologic deficits in late-stage disease: pyramidal and extrapyramidal motor signs, myoclonus, and seizures
-Stepwise loss of function in vascular dementia as microinfarcts add up
-Atypical presentations: visual variant, primary progressive aphasia
-May have psychotic symptoms or paranoia
-Personality changes and mood swings
-Depression
-Anger

Differential
-Delirium
-Depression ("pseudodementia")
-Structural brain disease: Parkinson's, Huntington's, Down's syndrome, head trauma, brain tumor, normal pressure hydrocephalus, MS, subdural hematoma
-Metabolic: hypothyroid, hypoxia, B12 or folate or thiamine deficiency, Wilson's disease, lead toxicity
-Infectious: Lyme, HIV, Creutzfeldt-Jakob, neurosyphilis, meningitis, encephalitis
-Drugs
-Mild cognitive impairment: normal daily function with abnormal memory for age; most progress to dementia
-Normal age-related cognitive decline: mild changes in memory and rate of info processing that are NOT progressive and don't affect daily function

Workup
-15% of "demented" patients have a treatable and potentially reversible condition!
-Full neuro exam
-MMSE: scores < 24 suggestive of dementia or delirium
-Clock drawing test appears to correlate with MMSE
-Labs: B-12, TSH
-Depression screen
-Noncontrast head CT or MRI (will easily diagnose vascular dementia)
-DSM-IV criteria for dementia:
1. Memory impairment
2. At least one of the following: apraxia, aphasia, agnosia, or disturbance in executive functioning
3. Disturbance significantly interferes with work, social activities, or relationships
4. Disturbance does not occur exclusively during delirium

-Additional DSM-IV criteria for Alzheimer type dementia:
1. Gradual onset and continuing cognitive decline
2. Not caused by identifiable medical, psychiatric, or neurologic condition

-Additional DSM-IV criteria for vascular dementia:
1. Focal neuro signs or evidence of cerebrovascular condition

-Gross pathology will show diffuse atrophy with enlarged ventricles and flattened sulci
-Microscopic pathology (brain biopsy) will show senile plaques composed of amyloid, neurofibrillary tangles derived from Tau protein, and neuronal and synaptic loss (however senile plaques and neurofibrillary tangles are also seen in normal aging)

Management
-Cholinesterase inhibitor for mild to moderate dementia (MMSE 10-26): donepezil, rivastigmine, galantamine
-Vitamin E 1000 IU BID if no CV disease
-Add memantine for mod-advanced dementia (MMSE < 17)
-D/c treatment when MMSE < 10 unless pt worsens significantly when off of them
-For behavioral issues, first r/o superimposed delirium
-Agitation and aggression are best managed by behavioral intervention rather than antipsychotics like haloperidol (evidence for aromatherapy exists as well as exercise, music, pet therapy, and massage)
-Depression: SSRIs may help, better choice for the elderly is citalopram
-Sleep disorders: small quantities of trazodone if needed
-Wandering
-Sexually inappropriate behavior: antidepressants, antipsychotics, cholinesterase inhibitors, other agents
-Delusions and hallucinations: atypical antipsychotics incur increased risk of mortality but may be necessary

Prognosis
-Average life expectancy of 3-8 years

Tourette Syndrome

-A movement and neurobehavioral disorder in children characterized by multiple motor and vocal tics
-Possible genetic cause

Signs & symptoms
-Waxing and waning tics: eye blinking, facial grimacing, shoulder shrugging, head jerking
-Irresistible urge before a tic and sudden relief afterwards
-Complex sequences of coordinated movements: bizarre gait, kicking, jumping, gyrations, scratching, seductive or obscene gestures
-Utterances: simple noises, obscene words, echolalia, palilalia
-May have bizarre thoughts and ideas, fixations, compulsive ruminations, perverse sexual fantasies
-Sleep complaints: restlessness, insomnia, nightmares, enuresis, teeth grinding, sleepwalking
-Onset usually between ages 2-15
-Comorbid ADHD, OCD, conduct disorder

Management
-Pharmacotherapy indicated only when symptoms interfere with social interactions, school or job performance, or ADLs
-Bothersome tics: fluphenazine or Botox injections
-Behavioral therapy with habit reversal training
-Stimulants for comorbid ADHD
-Clonidine for impulse control and rage attacks

Delirium

-AKA acute dementia, acute confusional state, acute organic brain syndrome
-A severe neuropsychiatric syndrome with core features of acute onset and fluctuating course with attentional deficits and generalized disorganization of behavior
-Pathophysiology not well understood
-Occurs in 30% of hospitalized patients

Risk factors/possible etiologies

-Metabolic disorders
-Infections
-Anemia
-Decreased cardiac output
-Hypotension
-Greater than 3 new meds
-Hypo or hyperthermia
-Fecal impaction or urinary retention
-Foley catheter
-Untreated pain
-Withdrawal
-Electrolyte imbalances
-Advanced age
-Males
-Deprivation: sensory stimuli, sleep
-Immobilization
-Transfer to new environment
-Severe illness
-Dehydration

Signs & symptoms
-Decreased consciousness
-Reduced focus and attention
-Changed cognition or perception
-Agitation or lethargy
-Not explained by underlying dementia
-Rapid onset
-Fluctuating

Workup
-CMP
-Ca
-CBC
-UA
-Tox screen
-ABG

Management
-Thiamine supplementation
-Frequent reassurance, touch, and verbal orientation
-Cautious trial of haloperidol for severe agitation or psychosis

Prognosis
-May require weeks to months to resolve
-62% of patients will still have symptoms 6 months out from discharge

Guillain-Barre Syndrome

-Refers to the acute immune-mediated polyneuropathies, of which there are several variants
-Thought to be an immune response to a preceding infection that cross-reacts with peripheral nerve antigens

Differential
-Other polyneuropathy
-Spinal cord disease
-Brainstem stroke
-Wernicke's encephalopathy
-Brainstem encephalitis

Agents
-EBV
-HIV
-HSV
-CMV
-*Campylobacter*

Signs & Symptoms
-Symmetric motor and sensory polyneuropathy that begins in an ascending fashion, from legs to trunk to arms
-Progresses over a 2 week period
-Paresthesias
-Areflexia
-Recent viral illness
-Absent or depressed DTRs
-Dysautonomia: tachycardia, urinary retention, hypo/hypertension alternating, ileus, loss of sweating

Workup
-Initial test is CSF: increased protein, normal WBCs (but not present in all pts)
-EMG studies

Management
-Supportive: mechanical ventilation
-Plasma exchange
-IV Ig

Prognosis
-Gradual recovery of function 2-4 weeks after progression stops

Multiple Sclerosis

Etiology & natural history
-Likely multifactorial, with infectious agents, genetic predisposition, and environmental factors all playing a role in the abnormal immune response
-Inflammation → demyelination → axonal loss
-More prevalent in individuals living further from the equator
-Demyelination continues to occur during the clinically silent periods in between relapses
-As disease progresses, MRI lesion burden and disability increase as cognitive function decreases

Forms
1.) Relapsing-remitting: partial recovery for disability between relapses, accounts for most cases, all meds are for this variety!
2.) Secondary progressive: Increasing disability with distinct relapses
3.) Primary progressive: Nearly continuous worsening of disability

Signs & symptoms
-Optic neuritis
-Transverse myelitis
-Paresthesias
-Ataxias
-Weakness
-Incoordination
-Spasticity
-Cognitive impairment
-Episodes that come and go

Workup
-Can't diagnose MS from just one attack, need to have at least 2
-Refer for brain MRI: T1/gadolinium contrast imaging enhances active lesions while T2 imaging represents cumulative disease burden; white spots must be in characteristic MS locations

Management
-Refer to neurology for immunomodulators and immunosuppressants
-Large dose steroids for relapses or aggressive disease
-Vitamin D supplementation

Prognosis
-If untreated brain atrophy will occur and half of all MS patients will need an assistive device to walk within 5 years and relapsing MS will progress to progressive MS within 10 years

Myasthenia Gravis

Etiology
-Production of ABs against Ach receptors
-Many cases are associated with a thymoma

Signs & symptoms
-Ocular: Ptosis, blurred vision, diplopia
-Bulbar/facial: difficulty chewing or swallowing, dysarthria, tired facial appearance, difficulty smiling or whistling, difficulty keeping food in mouth
-Asymmetric proximal or distal extremity weakness
-SOB
-Muscle weakness with repetitive use

Differential
-Botulism
-ALS
-Dermatomyositis or polymyositis
-Lambert-Eaton myasthenic syndrome
-MS
-MI
-PE
-Sarcoidosis
-Neuropathy
-Thyroid disease

Workup
-Tensilon test: acts as an Ach converting enzyme inhibitor to temporarily overcome neuromuscular jcn Ach deficit
-Labs: anti-Ach-R AB, MuSK AB
-Repetitive nerve stimulation test
-Single fiber EMG is the most sensitive test for myasthenia gravis
-Chest CT to rule out thymoma

Management
-Thymectomy
-Meds: Ach converting enzyme inhibitors like pyridostigmine, immunosuppressants
-Intubation if needed (don't use depolarizing or non-depolarizing agents)
-Plasmapheresis to remove pathologic AB
-Exacerbation can be precipitated by infection, surgery, pregnancy, or certain meds = avoid aminoglycosides, azithromycin, quinolones, botox, CCB, Mg, IV contrast

Seizure Disorders

-Epilepsy = documented h/o at least 2 seizures not related to a metabolic or febrile cause
-Risk factors: head trauma, CNS infections, cerebrovascular disease, alcohol, drug overdose or withdrawal, metabolic disorders, genetics, malignancy
-Common provoking factors: sleep deprivation, excessive stimulants, withdrawal from sedatives or alcohol, substance abuse, high fever, hypoxia, hypoglycemia, electrolyte disturbance, estrogen (= more seizures during ovulation and menses)

Differential
-Hyperventilation
-Migraine
-Panic attack
-Pseudoseizure
-Syncope
-Transient global ischemia
-TIA
-Sleepwalking
-Meningitis

Workup
-EEG to determine seizure type
-Electrolytes, glucose, anticonvulsant levels, alcohol and tox screen, ABG
-LP to r/o meningitis
-Head CT or MRI

Management
-When to treat after a single seizure: patients with structural lesion, abnormal EEG, focal seizure
-When NOT to treat after a single seizure: EtOH, drug abuse, provoked seizure, head injury with no structural abnormality
-Drugs are selected based on type of seizure, AEs, toxicity, cost, and childbearing potential
-Begin with monotherapy
-Consider 2nd agent if inadequate trial of 2 different single agents

Prognosis
-Most epileptics who go into remission do so within 3 years after their first seizure
-Poor prognostic factors for remission: FH of epilepsy, psychiatric comorbidity, h/o febrile seizures, > 20 seizure history, adult age, failed monotherapy

Partial Seizures

Management
-1st line: carbamazepine, phenytoin, lamotrigine, valproate, or oxcarbazepine
-2nd line: gabapentin, topiramate, levetiracetam, zonisamide, tiagabine, phenobarbital, felbamate

Simple Partial Seizures

-No LOC
-Alternation contraction and relaxation of muscle groups
-Eye movements and turning of head to the same side
-Speech arrest or vocalization
-May see flashes of light or color or have hallucinations
-May hear humming, buzzing, or hissing
-May experience unpleasant tastes or odors
-Dizziness
-Autonomic symptoms: flushing, incontinence, nausea, vomiting, goose bumps, pupillary dilation, sweating, tachycardia
-Psychiatric symptoms: detachment, memory distortion, time distortion, unprovoked emotion
-Can turn into a complex partial seizure or manifest in a continuous form (epilepsia partialis continue)

Complex Partial Seizures

-The most common kind of seizure
-Can occur after head trauma and many of these pts will have abnormal tissue or lesions in their temporal lobe
-Involves alteration of consciousness
-Automatisms such as lip smacking, picking, patting, chewing, or swallowing
-Inability to carry out simple commands or execute willful movement
-Lack of awareness of surrounding and events
-Can become generalized tonic-clonic seizure

Generalized Seizures			
Absence (Petit Mal) Seizures	**Tonic-Clonic (Grand Mal) Seizures**	**Myoclonic Seizures**	**Other Seizures**
-5-10 recurrent episodes of staring -May have minor motor automatisms -Pts have no memory of incident but are completely normal afterwards -Can be triggered by hyperventilation **Workup** -EEG abnormality will be present even when not seizing **Management** -1st-line: valproate, ethosuximide -2nd line: lamotrigine, levetiracetam **Prognosis** -Most cases resolve spontaneously	-Tonic phase begins with LOC, tensing of muscles, and often a loud yell or moan -Clonic phase commences with convulsions, eyes rolling back, strong jaw contractions -May have aura -Lasts 5-20 minutes -May have incontinence -May have unconsciousness after seizure followed by post-ictal state **Management** -1st line: phenytoin, carbamazepine, valproate -2nd line: lamotrigine, levetiracetam, topiramate, phenobarbital, primidone, oxcarbazepine	-Caused by metabolic abnormalities such as hepatic or renal failure -Brief major motor seizure with quick, lightning-like jerking movements of the trunk or extremities -May occur throughout body or limited to certain muscle groups -Onset may be so sudden that pt falls to the ground but can also be so brief that consciousness is not lost **Management** -1st line: clonazepam, valproate -2nd: lamotrigine, levetiracetam, topiramate, felbamate, zonisamide	**Tonic seizures** -Relatively rare to occur alone -Involve stiffening of the body, upward deviation of the eyes, dilation of the pupils, and altered respiratory patterns **Atonic seizures** -Sudden loss of muscle tone that may cause a fall -Last 1-4 secondes but without LOC -May affect one part of body to all body tone **Management** -1st line: valproate -2nd line: lamotrigine, topiramate, zonisamide

Anticonvulsants		
Agent & MOA	**Info**	**Risks & AEs**
Carbamazepine: inhibits voltage-gated Na channels	-Also for treating bipolar disorder, trigeminal neuralgia, and glossopharyngeal neuralgia	-AEs: diplopia, dizziness, drowsiness, nausea, Stevens-Johnson (don't use in Asians), hypoCa, hypoNa, SIADH, hematologic, hepatitis → monitor CBC, LFTs, mental status, bone density, levels -Decreases effectiveness of OCPs and warfarin -Pregnancy D
Oxcarbazepine: blocks voltage-gated Na channels, modulates Ca channels, increases K conductance	-For partial seizures	-AEs: sedation, dizziness, ataxia, nausea, Stevens-Johnson, hypoNa → monitor Na -Decreases effectiveness of OCPs and phenytoin -Pregnancy C
Clonazepam: modulates GABA transmission in the brain	-Not a first-line choice -Frequently added as a 2nd agent with levetiracetam	
Ethosuximide: increases seizure threshold, depresses nerve transmission in the motor cortex	-For absence seizure	-AEs: ataxia, drowsiness, GI, unsteadiness, hiccups, Stevens-Johnson, hematologic, SLE -Interactions with carbamazepine and valoproate -Pregnancy C
Felbamate: glycine-R agonist	-For partial and generalized seizures	-AEs: anorexia, n.v, insomnia, HA, Stevens-Johnson, aplastic anemia, hepatic failure = weekly LFTs & last resort drug! -Must sign informed consent -Interacts with many other seizure meds -Pregnancy C
Gabapentin or **pregabalin:** modulate Ca channels	-Add-on therapy for seizures -Also for neuropathic pain	-Renal dosing needed -AEs: dizziness, fatigue, ataxia, nystagmus, tremor, HA, peripheral edema, Stevens-Johnson -Pregnancy C
Lamotrigine: blocks voltage-gated Na channels and inhibits glutamate release	-Also for bipolar disorder	-AEs: nausea, diplopia, dizziness, unsteadiness, HA, rash, Stevens-Johnson, hematologic, liver failure -Interaction with valproate -Pregnancy C

Levetiracetam: inhibits Ca channels, facilitates GABA, reduces K currents, modulates NT release	-For partial, tonic-clonic, and myoclonic seizure	-AEs: sedation, suicidal ideation, pancytopenia, liver failure -Pregnancy C
Phenobarbital: decreases post-synaptic excitation	-Indicated for seizure and sedation	-AEs: ataxia, hyperactivity, HA, unsteadiness, sedation, nausea, cognitive impairment, blood dyscrasia, S-J, hepatic injury, osteopenia -Many drug interactions -Pregnancy D
Phenytoin: stabilizes neuronal membranes by altering Na efflux	-May be given as fosphenytoin for faster effect -For generalized and complex partial seizures	-AEs: ataxia, nystagmus, behavior, dizziness, HA, sedation, lethargy, incoordination, blood dyscrasias, rash, hirsutism, peripheral neuropathy
Tiagabine: inhibits GABA reuptake	-Adjunct therapy for partial seizures	-Pregnancy C
Topiramate: modulates Na channels, enhances GABA, antagonizes glutamate-R	-For partial or generalized seizures -Also indicated for migraine prevention	-AEs: difficulty concentrating, psychomotor retardation ("dopamax"), speech or language problems, fatigue, HA, metabolic acidosis, kidney stones → monitor BMP -Interacts with OCPs -Pregnancy C
Valproate: increases GABA	-For absence, complex partial, or mixed-type seizures -Also for bipolar disorder or migraine prophylaxis	-AEs: GI upset, sedation, unsteadiness, tremor, thrombocytopenia, palpitations, immune hypersensitivity, ototoxicity → monitor CBC and LFTs -Many drug interactions -Pregnancy D
Vigabatrin: irreversibly inhibits GABA transaminase	-For refractory complex partial seizures or complex generalized seizures	-AEs: permanent visual loss, psychiatric disturbances = in a restricted dist program
Zonisamide: MOA unknown	-Adjunct for partial seizure	-AEs: sedation, dizziness, cognitive impairment, nausea, kidney stones, S-J, schizophreniform disorder -Pregnancy C

Status Epilepticus

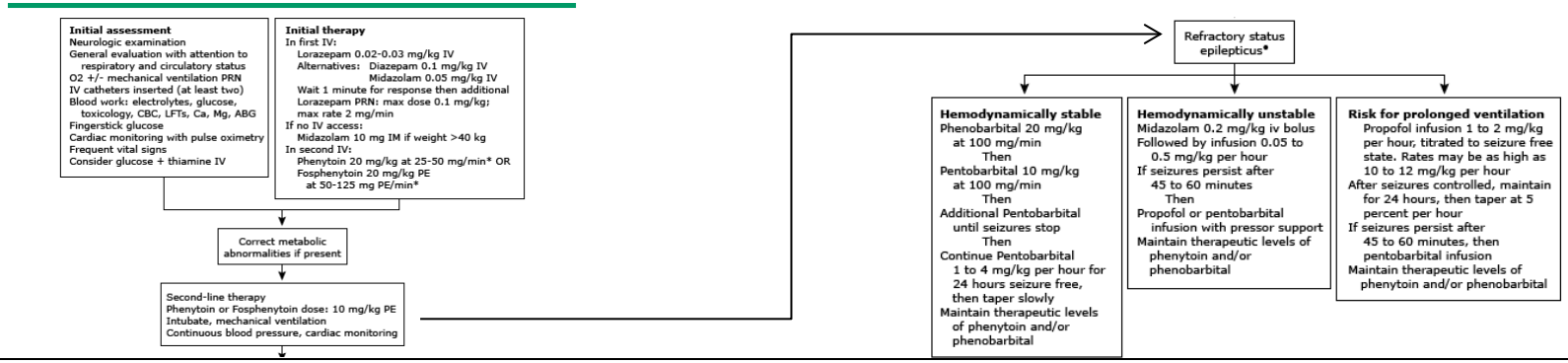
-Single unremitting seizure with duration > 5-10 minutes, or frequent seizures w/o interictal return to baseline clinical state

Causes
-Noncompliance with antiepileptic drug regimen
-Drug or EtOH withdrawal
-Acute brain injury or infection
-Metabolic disturbances

Workup
-Simultaneous assessment & treatment
-Careful neuro exam for any focal deficits
-EEG

Management
-Lorazepam, repeat as needed
-Loading dose of phenytoin of fosphenytoin for maintenance

Prognosis
-May have increased risk of mortality and neurologic sequelae



Febrile Seizures

-Typically occur in the setting of systemic bacterial or viral infection, but patient/family may not be aware of infection until sudden fever
-Genetic component

Type	Information	Workup	Management	Prognosis
Simple febrile seizures	-Less than 15 min (or total duration < 30 min if they occur in a series) -No focal features -Usually generalized tonic clonic seizures but may be atonic	-LP only indicated with meningeal signs or suspected intracranial infection, with infants 6-12 months not immunized with HIB and PCV, when patient is on antibiotics (masking of meningeal signs), and with seizures occurring after 2nd day of illness -Imaging for abnormal neuro exam	-Treat any febrile seizure longer than 5 minutes: lorazepam -Intubation if breathing becomes compromised -Electrolytes and glucose if > 5 minutes -Parents may be taught how to give rectal lorazepam once for recurrent febrile seizures -Generally preventative antiepileptic drug therapy is not indicated in this population	-Recurrence rate is 30% or more -Neurologic sequelae are rare -Preventative acetaminophen administered at the first sign of fever may or may not prevent a febrile seizure -Greater risk of later epilepsy, although prevention of febrile seizures using antiepileptics does not appear to reduce this risk
Complex febrile seizures	-Greater than 15 minutes (or total duration > 30 min if they occur in a series) -Focal features -Postictal paresis			
Febrile status epilepticus	-Lasts 30 minutes or longer -Unlikely to stop if not treated with antiepileptics	-May need LP	-Emergency management with antiepileptics and lorazepam to end seizure, cooling blanket, antipyretics	

Syncope

-True syncope is defined as a loss of consciousness followed by an immediate, spontaneous return to baseline without any new focal neurologic findings

Differential

Cardiac causes
-Arrhythmia (#1 cause!): slumping, brief loss of consciousness
-Aortic stenosis
-Valvular disease
-Acute MI or CAD: slumping
-Cardiac tamponade
-Pacemaker malfunction
-Obstructive cardiomyopathy: with exertion

Neurocardiac causes ("vasovagal syncope")
-Micturition or defection syncope
-Situational syncope
-Cough-mediated syncope
-Carotid sinus hypersensitivity: occurs with neck rotation or pressure
-Orthostatic syncope: usually a result of volume depletion or autonomic instability
-After prolonged standing
-Exertion in an athlete
→ Typically have a prodrome of nausea, chills, sweats

Blood loss
-Acute aortic dissection
-Trauma
-GIB
-Ruptured ovarian cyst or ectopic pregnancy
-Ruptured spleen

Neurologic causes
-Subarachnoid hemorrhage
-TIA
-Subclavian steal syndrome
-Complex migraine headache

Metabolic causes
-Hypoglycemia
-Hypoxia
-Hyperventilation

Other causes
-Intoxication
-Pulmonary embolism
-Cataplexy
-Drop attacks
-Meds: causing orthostasis or cardiotoxicity
-Epilepsy
-Psychogenic syncope
-Pulmonary HTN
-Seizure
-Neural cause: specific trigger, no cardiac history, after meals, loss of consciousness > 5 minutes

Workup

-EKG: check the PR and QT intervals
-BMP, BNP
-CXR
-Autonomic testing: carotid sinus massage
-Referral for electrophysiology studies
-Referral for upright tilt table test for suspected or to r/o vasovagal syncope

Management

-ER if a cardiac etiology is suspected or if there is recurrent syncope or if it related to exertion
-Admit for cardiac monitoring if syncope is due to cardiac cause or pt is high risk (abnormal EKG, h/o cardiac disease, hypotension, anemic, older age with comorbidities, FH of sudden cardiac death)

PSYCHIATRY

PSYCHIATRIC PHARMACOLOGY

Receptor type	Effects of psychiatric drugs	Receptor type
Dopamine (D_2)	Antagonists → antipsychotic effect, relief of + symptoms of schizophrenia, ↑extrapyramidal symptoms, increased prolactin levels	Serotonin 3 (5-HT_3)
Serotonin 1A (5-HT_{1A})	Agonists → antidepressant & anxiolytic effects	Alpha-1 adrenergic (α-1)
Serotonin 2A (5-HT_{2A})	Antagonists → improvement in neg symptoms of schizophrenia and improved cognition	Histamine (H_1)
Serotonin 2C (5-HT_{2C})	Antagonists → weight gain and associated risks	Muscarinic (m_1)

Class & MOA	Generic Agent	Brand	Info	
SSRIs: inhibit reuptake of serotonin as well as slight effects on histamine-R, α1-R, and muscarinic-R	Fluoxetine	Prozac	-Longest half-life = highest risk for serotonin syndrome -Many drug interactions -Most stimulating SSRI -Lowest weight gain = good for eating disorders	-AEs: GI, CNS, sexual, sedation, fatigue, dry mouth, hypotension, withdrawal if d/c abruptly, prolonged QT, rash, insomnia, asthenia, seizure, tremor, somnolence, mania, suicidal ideation, worsened depression -Risk of serotonin syndrome: shivering, hyperreflexia, myoclonus, ataxia, n/v/d
	Citalopram	Celexa	-Low risk of sexual AEs	
	Escitalopram	Lexapro		
	Fluvoxamine	Luvox		
	Sertraline	Zoloft	-Few drug interactions -Highest risk of GI problems	
	Paroxetine	Paxil	-Shortest half-life = highest risk of d/c symptoms -Most sedating SSRI and greatest weight gain and greatest sexual AEs -Greatest anticholinergic activity	
SNRIs: inhibits reuptake of both serotonin and norepinephrine	Venlafaxine (ER avail)	Effexor	-HTN -Sedating	-Equally effective as SSRIs for treating major depression -May be more effective in the setting of diabetic neuropathy, fibromyalgia, msk pain, stress incontinence, sedation, fatigue, and patients with comorbid anxiety -AEs: GI, HTN, CNS, permanent sexual?, diaphoresis, dizziness, fatigue, insomnia, blurred vision, suicidal ideation, dysuria, worsened depression -Fewer drug interactions
	Duloxetine	Cymbalta	-Less AEs than venlafaxine -Works well for fibromyalgia -Good for sleep and pain	
	Desvenlafaxine	Pristiq		
Atypical Antidepressants	Bupropion	Wellbutrin	-May increase sexual function -Has stimulant effects = good for comorbid ADHD or for helping quit smoking but don't use if comorbid anxiety or eating disorder -AEs: lower seizure threshold, insomnia, nervousness, agitation, anxiety, tremor, arrhythmias, HTN, tachycardia, S-J, weight loss, GI, arthralgia or myalgia, confusion, dizziness, HA, psychosis, suicidal ideation	
	Mirtazapine	Remeron	-Less nausea and sexual AEs -Overdose is generally safe -AEs: the most sedating antidepressant (= good for insomnia!), weight gain, orthostatic hypotension, dizziness, dry mouth	
	Nefazodone	Serzone		
	Trazodone	Oleptro	-AEs: arrhythmia, hyper or hypotension, diaphoresis, GI, hemolytic anemia, leukocytosis, dizziness, HA, insomnia, lethargy, memory impairment, seizure, somnolence, priapism, weight gain	

Class & MOA	Generic Agent	Brand	Info	Class & MOA
Tricyclic Antidepressants: inhibits reuptake of both serotonin and norepinephrine	Amitriptyline	Elavil	-Good for sleep, pain, and depression	-AEs: anticholinergic, CV, CNS, weight gain, sexual dysfunction, decreased seizure threshold -CV effects: orthostatic hypotension, conduction disturbance, cardiotoxicity → consider EKG prior to initiation -Overdose can be lethal
	Clomipramine	Anafranil		
	Desipramine	Norpramin	-Least sedating	
	Doxepin	Silenor		
	Imipramine	Tofranil		
	Nortriptyline	Pamelor		
MAOIs: block destruction of monoamines centrally and peripherally	Phenelzine	Nardil	-Irreversible	-MAO-A acts on norepinephrine and serotonin -MAO-B acts on phenylethylamine and DA -AEs: anticholinergic, lower seizure threshold, weight gain, rash, orthostasis, sexual dysfunction, insomnia or somnolence, HA, HTN crisis in presence of monoamines -Must be on tyramine-free diet = no wine, beer, cheese, aged food, or smoked meats -Overdose is lethal -2 week washout period of other antidepressants needed before starting in order to prevent serotonin syndrome
	Tranylcypromine	Parnate	-Irreversible	
	Selegiline	Emsam (transdermal)	-Reversible	
Mood Stabilizers	Carbamazepine	Tegretol	-MOA: antiepileptic; inhibits voltage-gated Na channels -AEs: diplopia, dizziness, drowsiness, nausea, Stevens-Johnson (don't use in Asians), hypoCa, hypoNa, SIADH, hematologic, hepatitis → monitor CBC, LFTs, mental status, bone density, levels -Contraindicated with bone marrow depression -Decreases effectiveness of OCPs and warfarin -Pregnancy D	
	Valproate	Depakene Depakote	-MOA: antiepileptic; increases GABA -AEs: GI upset, sedation, unsteadiness, tremor, thrombocytopenia, palpitations, immune hypersensitivity, ototoxicity → monitor CBC and LFTs and levels -Contraindicated with liver disease -Many drug interactions -Pregnancy D	
	Lamotrigine	Lamictal	-MOA: blocks voltage-gated Na channels and inhibits glutamate release -AEs: nausea, diplopia, dizziness, unsteadiness, HA, rash, Stevens-Johnson, hematologic, liver failure -Overdose can be fatal -Interaction with valproate -Pregnancy C	
	Lithium	Eskalith Lithobid	-Inhibits adenylate cyclase -AEs: diabetes insipidus, cognitive complaints, tremor, weight gain, sedation, diarrhea, nausea, hypothyroidism -Many drug interactions -Requires baseline BMP, TSH, EKG, Ca as well as monitoring of BMP and TSH q 6-12 mo -Monitoring for signs of toxicity: nausea, tremor, polyuria, thirst, weight gain, diarrhea, cognitive impairment -Need to monitor levels -Pregnancy D for neural tube defects	
	Gabapentin	Neurontin	-AEs: somnolence, dizziness, ataxia, fatigue, leukopenia, weight gain, Stevens-Johnson	

Class & MOA	Generic Agent	Brand	Info
Benzodiazepines: GABA-R agonists → CNS inhibition	Chlordiazepoxide	Librium	-Long-acting -Used often during EtOH withdrawal
	Clorazepate	Tranxene	-Long-acting
	Diazepam	Valium	-Long-acting
	Flurazepam	Dalmane	-Long-acting
	Alprazolam	Xanax	-Intermediate acting -Approved for panic disorder
	Clonazepam	Klonopin	-Intermediate acting -Approved for panic disorder
	Lorazepam	Ativan	-Intermediate acting
	Temazepam	Restoril	-Intermediate acting
	Oxazepam	Serax	-Short acting
	Triazolam	Halcion	-Short acting
Other Anxiolytics	Buspirone	BuSpar	-5-HT partial agonist -Gradual onset in 2 weeks -Does not potentiate effects of alcohol = useful in alcohols -Low addiction potential = good for pts who were addicted to benzos or other drugs -AEs: sexual, dizziness, nausea, HA -Drug interactions
Typical Antipsychotics: nonselective DA-R antagonists	Haloperidol (inj avail)	Haldol	-Good for acute agitation as onset is 30 min
	Fluphenazine	Prolixin	
	Perphenazine	Trilafon	
	Thioridazine	Mellaril	-AE: retinitis pigmentosa -Less risk of EPSEs
	Chlorpromazine	Thorazine	-Less risk of EPSEs
Atypical Antipsychotics: block postsynaptic DA-R, block serotonin-R, variable effect on histaminic and cholinergic-R	Aripiprazole	Abilify	
	Asenapine (SL tablet avail)	Saphris	-Costs $$$
	Olanzapine (inj avail)	Zyprexa Zyprexa Relprevv (inj)	-High risk of weight gain and metabolic syndrome -Injectable can cause post-injection delirium → must give at healthcare facility and monitor for 3 hours
	Quetiapine	Seroquel	-Need q 6 month eye exams due to risk of cataracts
	Risperidone	Risperdal Consta (inj)	-Least amount of AEs -Highest risk of hyperprolactinemia
	Ziprasidone	Geodon	-AE: dose-related QT prolongation -Less wt gain
	Clozapine	Clozaril	-The only atypical antipsychotic proven effective in treatment of schizophrenia -Use limited by AEs: high risk of weight gain and metabolic syndrome, seizures, agranulocytosis, myocarditis, lens opacities → need to monitor WBC and ANC frequently
	Iloperidone	Fanapt	-Costs $$$ -Not proven better than other atypical antipsychotics
	Lurasidone	Latuda	-Best choice for reversing metabolic effects
	Paliperidone (inj avail)	Invega Invega Sustenna (inj)	

Management of Psychiatric Drug Adverse Effects

Dystonias
-Benztropine
-Biperiden
-Diphenhydramine
-Trihexyphenidyl

Akathisias = restlessness
-Propranolol
-Benzos

Parkinsonianism
-Amantadine
-Levodopa

Extrapyramidal Symptoms
-Parkinsonian syndrome, acute dystonias, akathisia
-Benztropine
-Benadryl

ANXIETY DISORDERS

Generalized Anxiety Disorder

-An inappropriate response to a perceived threat or no adequate source for fear
-More common in women; 30% lifetime risk
-More common in high SES

Etiology
-Likely a combination of genetic factors and environment

Screening
-GAD-7 has been validated as a primary care tool

Signs & symptoms
-Palpitations
-Diaphoresis
-Dizziness
-Trembling
-SOB or choking sensation
-Tingling of extremities
-Somatic complaints such as muscle tension
-Common comorbidity is major depression

Workup
-DSM-IV criteria:
1. Excessive anxiety and worry about lots of things out of proportion to the likelihood or impact of feared events, occurring more days than not for at least 6 mo
2. Worry is pervasive and difficult to control
3. Anxiety and worry are associated with 3+ of these sx: restlessness or on edge, easily fatigued, difficulty concentrating, irritability, muscle tension, sleep disturbance
4. Anxiety, worry, or physical symptoms cause significant stress or impairment socially, at work, or in other areas of life

-Labs to consider: CBC, BMP, TSH, UA with tox screen, EKG in pts over 40 with associated chest pain or palps

Differential
-*Adjustment disorder with anxious mood*: fits pts who have had sx < 6 mo that are associated with a particular stressor or event within 3 months of onset of sxs
-CV problem
-Excess caffeine or MSG intake or other stimulants
-Vitamin deficiency
-Anemia, adrenal disease, or secreting tumor
-Hyperthyroidism
-B12 deficiency
-Hypoxia
-Drugs: antidepressants, penicillin, amphetamines
-EtOH withdrawal

Management
-Combination of psychotherapy and pharmacotherapy is most effective
-First-line is CBT or an SSRI or SNRI with a 6-8 week trial, treat for at least a year
-Second-line:
- buspirone: reduced abuse potential, less weight gain and sexual AEs (should NOT be taken PRN)
- benzos
- TCA such as imipramine: limited use due to AEs

-Third-line:
- hydroxyzine: use limited by sedation and lack of efficacy in comorbid diseases
- pregabalin
- quetiapine

-For remaining insomnia, add on a non-benzo hypnotic, benzo, trazodone, mirtazapine, or sedating hypnotic
-For inadequate response, switch antidepressants or add atypical antipsychotic, benzo, antihistamine, or buspirone

Prognosis
-Half will fully recover within a few years of therapy while half will continue to struggle with lifelong fluctuating symptoms

Panic Disorder

-Periods of intense fear or apprehension that are of sudden onset and relatively brief duration
-Further specified as being with or without agoraphobia

Etiologies
-Caused by an overreaction to stimulation of the amygdala and adrenal gland
-Common inciting factors: hyperventilation, breathing in and out of a paper bag, caffeine, nicotine

Differential
-SVT
-Angina pectoris
-CHF
-Hyperthyroid
-Pheochromocytoma
-Caffeine, nicotine, cocaine, amphetamines, pseudoephedrine
-Other anxiety disorder: OCD, PTSD
-Somatization

Signs & Symptoms
-May not have inciting factor
-Shaking and trembling
-Choking sensation
-SOB
-Diaphoresis, hot flashes, or chills
-Derealization and depersonalization
-Chest pain and palpitations
-Persistent concern about having another attack
-Fear of dying or losing control
-Abdominal pain
-Paresthesias
-Can occur up to several times per week, with waxing and waning of frequency
-Medical comorbidities often present: HTN, asthma, mitral valve prolapse, IBS, interstitial cystitis, migraines

Workup
-DSM-IV criteria for *panic attack*: 4+ symptoms that develop abruptly and reach a peak within 10 minutes
-DSM-IV criteria for *panic disorder*: h/o panic attack + intense fear of having another
-BMP
-Cardiac enzymes for suspected ACS
-TSH
-CBC
-Urine tox screen
-D-dimer
-ABG or VBG shows primary respiratory alkalosis from hyperventilation
-EKG

Management
-Acute: benzodiazepines
-CBT ± pharmacotherapy
-1st line long-term management SSRI or SNRI, 60-80% of pts will be panic free after > 4 weeks
-2nd line therapy with benzodiazepines, studies show low risk of misuse in panic disorder pts, alprazolam has the most data with 55-75% of pts panic free after 1 week

Prognosis
-Untreated attack usually subsides in 25 minutes

Posttraumatic Stress Disorder

-A response to a catastrophic life experience in which the pt re-experiences the trauma, avoids reminders of the event, and experiences emotional numbing or hyperarousal

Causes
-Military combat
-Sexual or physical assault
-Disasters
-Childhood sexual abuse
-Severe physical injury
-ICU hospitalization

Signs & Symptoms
-Marked cognitive, affective, and behavioral responses to stimuli reminding them of the experienced trauma
-Avoidance, emotional numbing, and diminished interest in people and activities
-Comorbid depression, substance use, and somatization

Differential
-*Acute stress disorder*: meets criteria for PTSD but has had symptoms < 1 month

Workup
-Diagnosed clinically by presence of criteria at least one month after traumatic event has passed
-DSM-IV criteria:
1. Experienced a traumatic event that was potentially harmful or fatal and initial reaction was intense fear or horror
2. Persistent re-experiencing of the event through dreams, flashbacks, or recollections
3. Avoidance of stimuli associated with the trauma
4. Numbing of responsiveness: limited affect, feelings of detachment or estrangement from others
5. Persistent symptoms of increased arousal: difficulty sleeping, anger outbursts, exaggerated startle response, difficulty concentrating

Management
-CBT is first-line
-SSRI or SNRI 2nd line
-Atypical antipsychotics for refractory symptoms
-Prazosin for sleep disruption or nightmares

Prognosis
-Half of pts will remain symptom-free after 3 months of treatment

Obsessive-Compulsive Disorder

-Obsession = recurrent and intrusive thought, feeling, or idea
-Compulsion = a conscious repetitive behavior linked to an obsession that functions to relieve anxiety caused by the obsession
-Onset is usually in adulthood

Causes
-Genetic predisposition
-Stressful life event triggers many cases
-Abnormal serotonin levels

Signs & Symptoms
-Common obsessions: contamination, doubt/repeated checking of objects for safety, symmetry, intrusive sexual or violent thoughts
-Common psych comorbidities include MDD, eating disorders, other anxiety disorders, and obsessive-compulsive personality disorder

Workup
-DSM-IV criteria:
1. Obsessions and/or compulsions
2. Person is aware that the obsessions and compulsions are unreasonable and excessive
3. Obsessions cause marked distress, are time-consuming, or significantly interfere with daily functioning

Management
-1st line: CBT using exposure and response prevention and/or SSRI (may need HIGH doses!)
-Augment nonresponders with an antipsychotic or TCA
-Deep brain stimulation for refractory cases is showing promising results

Prognosis
-20-40% of pts will remain significantly impaired from their symptoms

Phobias

-The most common mental disorders in the US
-An irrational fear that leads to avoidance of feared object or situation
-Specific phobia = fear of specific object or situation
-Social phobia (social anxiety disorder) = fear of social situations in which embarrassment can occur

Causes
-Likely a combination of genetic, behavioral, and neurochemical factors

Workup
-DMS-IV criteria for phobia:
1. Persistent, excessive fear brought on by a specific situation or object
2. Exposure to the situation brings about an immediate anxiety response
3. Pt recognizes that this fear is excessive
4. Situation is avoided when possible or tolerate diwth intense anxiety
5. Duration must be > 6 mos of < 18 years old

Management
-Pharmacologic treatment of specific phobias is not effective
-Systemic desensitization ± benzos during session
-Supportive psychotherapy
-Paroxetine effective for social anxiety disorder
-β-blockers for performance anxiety

EATING DISORDERS

-Anorexia and bulimia are more common in middle and upper-class families

Etiology: combination of psychological, social, and biologic factors
-Psych: perfectionism, high expectations, need for control, people pleasing, hypersensitivity to real or perceived rejection
-Social: over-valuing thinness, sexualization of women, restricted expression of emotion, familial emphasis on weight control, high amount of life stressors
-Biologic: genetic influences, serotonin imbalance in bulimia, comorbid major depression or bipolar disorder in bulimia

Screening
-SCOFF screen useful in primary care, considered to be 100% sensitive: sick, control, "one stone", fat, food → 2+ points suggest eating disorder

Workup
-EKG
-BMP, TSH, vitamin levels
-DEXA

Management
-Intervention designed to decrease shame, validate patient feelings, assess social supports, encouragement of patient honesty and openness, inform about available resources, and affirm provider willingness to provide ongoing support
-Psychotherapy: individual and family
-Regular medical visits
-Admit for: weight loss > 20% ideal, unresponsiveness to outpatient therapy, rapid weight loss, hypovolemia, electrolyte abnormalities, malnutrition, severe depression or suicidality

Prognosis
-Complication of refeeding syndrome, when shift from fat to CHO metabolism causes ↓ P → depletion of intracellular ATP and tissue hypoxia → impairment of myocardial contractility → CV collapse, seizures, delirium, or rhabdomyolysis
-Complication of Wernicke's encephalopathy, prevented with thiamine supplementation

Anorexia Nervosa

-Far more common in women, in developed countries, and in professions requiring a thin physique

Restrictive type anorexia nervosa
-Eat very little
-Exercise vigorously
-More often withdrawn with obsessive-compulsive traits

Binge eating & purging type anorexia nervosa
-Eat in binges followed by purging, laxatives, excessive exercise, or diuretics
-Melanosis coli: darkening of colon secondary to laxative abuse
-Comorbid major depression or substance abuse

Other signs & symptoms
-Low body weight
-Preoccupation with food
-Social withdrawal
-Frequent weighing
-Fatigue
-Hair loss
-Cessation of menses
-Sensitivity to cold
-Serious: arrhythmia, dehydration, malnutrition, hypotension, bradycardia, reduced bone density, heart failure, dental problems, hypothermia, fainting, lanugo

Differential
-Malignancy or other medical condition causing cachexia
-Major depression
-Bulimia
-Somatization disorder
-Schizophrenia

Workup
-DSM-IV criteria:
1. Body weight at least 15% below normal
2. Intense fear of gaining weight or becoming fat
3. Disturbed body image
4. Amenorrhea

Management
-Inpatient needed if < 20 below ideal body weight
-Refer for psychotherapy: family therapy
-Meds only after weight is restored: atypical antipsychotics, tricyclics, SSRIs, Li, anxiolytics before eating

Prognosis
-Mortality of 5-20%
-50% will have good results, 25% intermediate, 25% poor
-Average duration of illness is 5.9 years

Bulimia Nervosa

Purging type bulimia nervosa
-Use vomiting, laxatives, or diuretics to counteract binge eating

Nonpurging type bulimia nervosa
-Use excessive exercise or fasting to counteract binge eating

Signs & symptoms
-Normal weight or overweight
-Patients are embarrassed by bingeing
-Over-concern with body weight
-Esophagitis
-Dental erosion
-Callused knuckles
-Salivary gland hypertrophy
-Comorbid mood disorder, impulse control disorder, or alcohol abuse

Workup
-DSM-IV criteria:
1. Recurrent episodes of binge eating
2. Recurrent, inappropriate attempts to compensate for overeating and prevent weight gain
3. Binge eating and compensatory behaviors occur at least twice a week for 3 months
4. Perception of self worth is excessively influenced by body weight and shape

-Chem panel shows hypochloremic hypokalemic alkalosis

Management
-Psychotherapy
-SSRIs or TCAs

Prognosis
-Better than anorexia nervosa
-May have relapse in times of stress
-Half will recover fully and half will be chronic with fluctuating symptoms

Binge-Eating Disorder

Binge-eating = eating an excessive amount of food in a 2 hour period with associated lack of emotional control

Signs & symptoms
-Emotional distress associated with overeating
-No use of laxatives or vomiting
-Often obese
-Comorbid mood or anxiety disorder

Workup
-DSM-IV criteria:
1. Recurrent episodes of binge eating
2. Severe distress over binge eating
3. Bingeing occurs at least 2 days a week for 6 months and is not associated with compensatory behaviors
4. 3+ of the following are present: eating very rapidly, eating until uncomfortably full, eating large amounts when not hungry, eating alone due to embarrassment over eating habits, feeling disgusted, depressed, or guilty after overeating

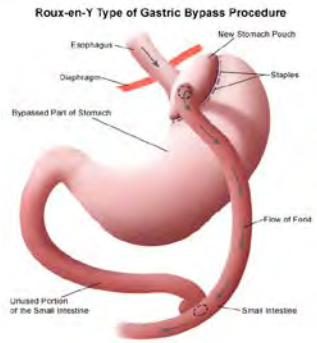
Management
-Psychotherapy
-Strict diet and exercise program
-Pharmacotherapy to support weight loss: stimulants, orlistat, or sibutramine (Meridia)

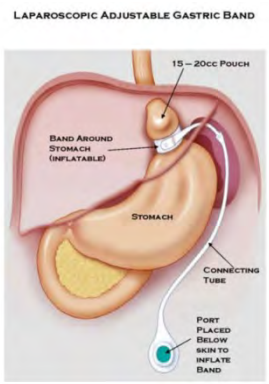
Obesity

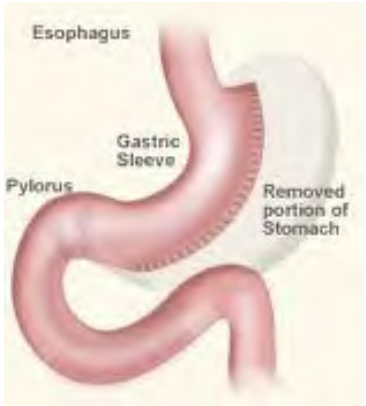
-Overweight = BMI 25-29.9
-Obesity = BMI > 30 → greater risk of DM, stroke, CAD, early death

Pharmacologic options
-Catecholaminergics (phentermine, diethylpropion, mazindol): short-term use only
-Orlistat: inhibits lipase

Bariatric surgery
-NIH recommends limiting to patients with BMI > 40, or > 35 if obesity complications are present
-Results in significant reduction in deaths from obesity
-Options: adjustable "lap band", sleeve gastrectomy, Roux-en-Y bypass







MOOD DISORDERS

-Mood disorders can be further classified with specifiers
-"Psychotic features" = delusions or hallucinations
-"Catatonic features" = prominent psychomotor disturbances
-"Postpartum onset" = onset of mood episodes within 4 weeks of childbirth

Major Depressive Disorder

-"Episodes" are classified as mild, moderate, or severe and are defined by presence of symptoms for at least 2 weeks
-A pt has a "disorder" when there is a pattern of episodes
-Half of cases are missed by PCPs
-First episode most common in ages 30-40, with smaller peak at 50-60

Subtypes
-Melancholic depression: loss of pleasure in most activities, nonreaction to pleasurable stimuli, worsening of sx in early morning hours, psychomotor retardation, excessive weight loss or guilt
-Atypical depression: mood reactivity, weight gain, excessive sleep, heavy sensation, significant social impairment
-Catatonic depression: rare form involving disturbances of motor behavior
-Postpartum depression: intense, sustained depression experienced within 1 month of giving birth
-Seasonal affective disorder: resolves in spring
-Major depression with psychotic features: delusions or hallucinations are present

Etiology
-Multifactorial, involving biologic, psychologic, and social factors

Screening
-USPSTF grade B for nonpregnant adults as long as there are supports in place for effective treatment
-PHQ-2 or PHQ-9
-Geriatric depression scale

Differential
-Dysthymia
-Adjustment disorder with depressed mood
-Minor depressive disorder
-Bipolar disorder
-Dementia
-Normal grief response
-Anxiety disorder
-Domestic violence
-Hypothyroidism
-Parkinsonism
-Stroke
-Drug-induced depression
-Cushing's syndrome
-Borderline personality disorder

Workup
-MMSE
-TSH, CMP and Ca to r/o metabolic disturbance, RPR, CBC to r/o systemic infection, folate, vit D, vit B12, drug screen
-DSMI-IV diagnostic criteria for *major depressive episode* is 5+ of the following symptoms present nearly every day for a minimum of 2 weeks (at least 1 symptom must be depressed mood or loss of interest or pleasure)
- Depressed mood
- Loss of interest or pleasure in most or all activities
- Insomnia or hypersomnia
- Change in appetite or weight gain
- Psychomotor retardation or agitation
- Low energy
- Poor concentration
- Thoughts of worthlessness or guilt
- Recurrent thoughts about death or suicide

-DSM-IV criteria for *MDD*:
- At least 1 major depressive episode
- No h/o mania or hypomania

Management
-Referral to psychiatry indicated for pts with severe depression, depression unresponsive to initial treatment, psychotic depression, depression with other psychiatric diagnosis
-Psychotherapy recommended in combination with pharmacotherapy for patients with severe chronic or recurrent depression
-Exercise
-Melancholic subtype responds best to TCAs or MAOIs
-Atypical subtype responds best to SSRIs or SNRIs
-For refractory depression, confirm dx and medication adherence and r/o organic causes of depression, switch to another antidepressant or augment, and consider less common regimens such as tranylcypromine or venlafaxine + mirtazapine
-Electroconvulsive therapy is last resort

Prognosis
-15% will attempt suicide

Related Disorders

Dysthymia
-A chronic, milder mood disturbance that has been present for at least 2 years

Minor depressive disorder
-Pt meets criteria for 2-4 of MDD criteria

Adjustment disorder with depressed mood
-Occurs when identifiable psychosocial stressor(s) that has occurred within the last 3 months can be attributed to depressed mood
-Depressed mood resolves within 6 months after the stressor has ended
-However, if pt meets criteria for MDD this diagnosis will supersede dx of adjustment disorder

Pharmacologic Management

-Effectiveness of SSRIs, SNRIs, bupropion, TCAs, and MAOIs is generally comparable
-Begin dose at lowest effective dose and increase incrementally until patient achieves remission
-Switch after 8 weeks of therapy if no response
-If partial response at 8 weeks, can switch or augment therapy with bupropion or buspirone
-May need to use doses higher than what is FDA approved
-Duration of treatment should be continued until 4-9 months after remission of symptoms for 1st episode, an additional year after that for 2nd episode, and possibly continue treatment indefinitely for 3rd episode or more

Postpartum Depression

-Can occur in women or men
-Mood changes will develop in 40-80% of women postpartum and are normal as long as duration is < 2 weeks

Risk Factors
-Formula feeding
-H/o depression
-Cigarette smoking
-Childcare stress or low social support
-Infant colic
-Low SES
-Unplanned pregnancy

Screening
-Edinburgh Postnatal Depression Scale

Signs & Symptoms
-Sadness and crying episodes
-Fatigue
-Changes in sleeping and eating habits
-Reduced libido
-Irritability
-Feelings of hopelessness and low self-esteem
-Guilt
-Feeling overwhelmed and inadequate in caring for infant
-Inability to be comforted
-Anhedonia and social withdrawal
-Anxiety and panic attacks
-Anger spells

Workup
-DSM-IV criteria is depression symptoms > 2 weeks with onset within 4 weeks of childbirth

Management
-Attention to infant by other family members or friends
-Support groups or counseling, home visits
-Psychotherapy
-Healthy diet and sleep patterns
-Meds recommended only if support and adequate rest fail to improve symptoms: sertraline or paroxetine

Prognosis
-Can last several months to a year if untreated

Bipolar Disorder

-Affects men and women equally
-Mean age of onset is 19
-Illness can be further specified as "rapid cycling" (4+ episodes per year, separated in time by remission or switching to opposite pole) or "seasonal pattern"

Causes
-Genetic predisposition

Signs & Symptoms
-Buying sprees or sexual indiscretions
-Symptoms in younger children or adolescents will be much more vague and shorter-lived = harder to diagnose in this age group and requires lots of experience!
-Most bipolar disorder pts will have at least 1 comorbid psychiatric illness
-Neurocognitive deficits in attention, verbal memory, executive function, and info processing speed
-Suicide attempt
-Aggressive behavior

Differential
-Major depressive disorder
-ADHD
-Schizophrenia
-Hyperthyroidism
-Substance abuse
-Meds: steroids, thyroxine
-CNS disease: tumor, neurosyphilis
-Borderline or narcissistic personality disorder

Hypomania vs mania?
-Mania frequently includes psychotic features and leads to hospitalization while hypomania does not
-Psychosocial functioning in hypomania is either mildly impaired or improved while it is markedly impaired in mania

Workup
-DSM IV-TR criteria for *mania* are 3 (with elated mood-predominant) or 4 (with irritable mood-predominant) of the following for at least 1 week:
- Inflated self-esteem or grandiosity
- Decreased need for sleep
- ↑ Talkative or pressure speech
- Flight of ideas or racing thoughts
- Distractibility
- ↑ Goal-directed activity or psychomotor agitation
- Excessive involvement in pleasurable activities without regard to negative consequences

-DSM IV-TR criteria for *hypomania* are the same as above but last at least 4 days and are less severe than mania

Prognosis
-Rapid cycling subtype associated with poorer long-term course of illness
-10-15% death rate by suicide
-Untreated manic episode usually lasts 3 months and will relapse more frequently as the disease progresses

Types of Bipolar Disorder

Bipolar I Disorder	Bipolar II Disorder	Cyclothymic Disorder	Bipolar Disorder NOS
-1+ manic or mixed episodes -May also have MDD but not required -Episodes not better accounted for by schizophrenia, delusional disorder, or psychotic disorder NOS	-1+ major depressive episode AND 1+ hypomanic episode -No manic or mixed episodes -Episodes not better accounted for by schizophrenia, delusional disorder, or psychotic disorder NOS	-Numerous periods of: • hypomanic episodes for at least 2 years • depressive sx that don't meet criteria for MDD → "bipolar II disorder lite"	-Includes any other pt that does not meet criteria for bipolar I or II or cyclothymic disorder

Management of Bipolar Disorder

-Regimen is selected according to phase of the illness pt is currently experiencing (acute depression, acute mania or hypomania, mixed episodes, or maintenance therapy)
-Pts need to be aware of teratogenicity of drug regimens (ordered from greatest to least teratogenic): valproate > carbamazepine > Li > lamotrigine > antipsychotics > antidepressants

Antidepressants
-Efficacy of antidepressants for bipolar disorder is uncertain
-No evidence for use as a maintenance therapy
-Certain classes may incite switch to mania
-Should never be used as monotherapy, but in conjunction with antimanic or 2nd gen antipsychotic

Maintenance therapy
-Goals are to reduce symptoms, delay or prevent new episodes, reduce risk of suicide, and promote psychosocial functioning
-Li monotherapy is DOC: rapid absorption, steady state within 3-5 days after dose changes, however only 50-60% of pts will respond
-2nd line is anticonvulsants or antipsychotics: lamotrigine, risperidone, aripiprazole, divalproex, quetiapine, or olanzapine
-Refractory or inadequate response → add a 2nd med
-Psychotherapy can also help prevent recurrences and hospitalizations
→ Most pts will require maintenance therapy for years, and some require it for life
→ Li use requires baseline BMP, TSH, EKG, Ca as well as monitoring of BMP and TSH q 6-12 mo; monitoring for signs of toxicity (nausea, tremor, polyuria, thirst, weight gain, diarrhea, cognitive impairment)

Acute mania, mixed episodes, or hypomania
-D/c any antidepressants
-Severe manic or severe mixed episode → (Li or valproate) + antipsychotic is treatment of choice; switch antimanic if not working; ECT is last resort after 4-6 failed drug therapy trials
-Hypomania, mild-mod mania, or mixed episode → risperidone or olanzapine monotherapy
-ECT an option

Acute depression
-1st line: quetiapine, Li, lamotrigine, or valproate
-Dietary supplementation with omega 3 FAs has been shown to be effective in improving depressive symptoms

PERSONALITY DISORDERS

-Involve deeply ingrained, inflexible patterns of relating to others that are maladaptive and cause significant impairment in social or occupational functioning
-Pts lack insight into their problems
-Genetic predisposition
-These are Axis II diagnoses

Cluster A Personality Disorders

-Pts are eccentric, peculiar, withdrawn
-FH of psychotic disorders

Schizoid Personality Disorder

Signs & symptoms
-Lifelong pattern of social withdrawal
-Reclusive
-Quiet and unsociable
-Restricted affect
-No desire for close relationships
-Prefer to be alone

Differential
-Paranoid schizophrenia: will have fixed delusions
-Schizotypal personality disorder: will have eccentric behavior or magical thinking

Workup
-DSM-IV criteria: pattern of voluntary social withdrawal and restricted range of emotion beginning by early adulthood and present in a variety of contexts; with at least 4 of the following present:

- Neither enjoying nor desiring close relationships
- Generally choosing solitary activities
- Little interest in sexual activity with another person
- Taking pleasure in few activities
- Few close friends and confidants
- Indifference to praise or criticism
- Emotional coldness, detachment, or flattened affect

Management
-Psychotherapy is treatment of choice
-Low-dose antipsychotics if transiently psychotic
-Antidepressants if comorbid major depression is diagnosed

Prognosis
-Usually chronic

Paranoid Personality Disorder

Signs & symptoms
-Pervasive distrust and suspiciousness of others
-Usually interpret motives as malevolent
-Blame own problems on others
-Angry and hostile

Differential
-Paranoid schizophrenia: will have fixed delusions and are psychotic while paranoid personality disorder may only have transient psychosis

Workup
-DSM-IV criteria: must begin by early adulthood and be present in a variety of contexts; with at least 4 of the following present:

- Suspicion without evidence that others are exploiting or deceiving pt
- Preoccupation with doubts of loyalty or trustworthiness of acquaintances
- Reluctance to confide in others
- Interpretation of benign remarks as threatening or demeaning
- Persistence of grudges
- Perception of attacks on pt character that are not apparent to others; quick to counterattack
- Recurrence of suspicions regarding fidelity of spouse or lover

Management
-Psychotherapy is treatment of choice
-Anxiolytics
-Short course of antipsychotics

Prognosis
-May eventually become schizophrenic
-Chronic, lifelong job and marital issues

Schizotypal Personality Disorder

Signs & symptoms
-Eccentric behavior
-Peculiar thought patterns

Differential
-Paranoid schizophrenia: will have psychotic features but schizotypal won't unless under high stress (will be transient)
-Schizoid personality disorder: won't have eccentric behavior like schizotypal

Workup
-DSM-IV criteria: a pattern of social deficits marked by eccentric behavior, cognitive or perceptual distortions, and discomfort with close relationships, beginning by early adulthood and present in a variety of contexts; 5+ of the following must be present:

- Ideas of reference (but not delusions of reference)
- Odd beliefs or magical thinking inconsistent with cultural norms (clairvoyance, telepathy, bizarre fantasies, superstitious beliefs)
- Unusual perceptual experiences
- Suspiciousness
- Inappropriate or restricted affect
- Odd or eccentric appearance or behavior (cults, strange religious practices)
- Few close friends or confidants
- Odd thinking or speech
- Excessive social anxiety

Management
-Psychotherapy is treatment of choice
-Short course of low-dose antipsychotics for transient psychosis

Prognosis
-Course is chronic
-Pts may eventually develop schizophrenia

Cluster B Personality Disorders

-Pts are emotional, dramatic, or impulsive
-FH of mood disorders
-Often utilize splitting people/things into good & bad as a coping mechanism

Borderline Personality Disorder

Signs & symptoms
-Pattern of instability of interpersonal relationships
-Poor self-image
-Unstable affect
-Impulsivity
-Common comorbidities are mood, anxiety, and substance use disorders
-May have suicide attempts

Differential
-Mood disorder
-PTSD
-Dissociative identity disorder
-Other personality disorders
-Schizophrenia: will have frank psychosis whereas BPD may decompensate into transient psychosis during stress

Workup
-DSM-IV criteria: 5+ of the following present in a variety of contexts by early adulthood:

- Frantic efforts to avoid real or imagined abandonment
- A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes and idealization and devaluation
- Markedly and persistently unstable self-image or sense of self
- Impulsivity in at least 2 areas that are potentially self-damaging
- Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior
- Marked reactivity of mood
- Chronic feelings of emptiness
- Inappropriate, intense anger or difficulty controlling anger
- Transient, stress-related paranoid ideation or severe dissociative symptoms

Management
-If comorbid Axis I disorder is present, BPD should be treated first as remission of both disorders often depends upon successful treatment of BPD
-Psychotherapy for at least 20 weeks
-Consider mood stabilizer for lability, inappropriate anger, impulsivity, or aggression
-Consider antipsychotic for perceptual disturbances
-Consider treating comorbid depression, PTSD, or panic disorder with antidepressants

Prognosis
-Rate of remission is 50% after 4 years and 90% after 10 years
-Remission associated with eventual improvement in psychosocial functioning

Antisocial Personality Disorder

-Higher incidence in poor urban areas and in prisoners
-Genetic component

Signs & symptoms
-Refuse to conform to social norms
-Lack remorse for actions
-Impulsive and deceitful
-Run-ins with the law
-Can appear charming and normal to new acquaintances
-Comorbid substance abuse or depression
-May have somatic complaints

Differential
-Drug abuse

Workup
-DSM-IV criteria: pattern of disregard for others and violation of rights of others since age 15; must be at least 18 years old; history of behavior as adolescent must be consistent with conduct disorder; additionally 3+ of the following are present:

- Failure to conform to social norms by committing unlawful acts
- Deceitfulness or repeated lying or manipulation of others for personal gain
- Impulsivity or failure to plan ahead
- Irritability and aggressiveness, repeated fights or assaults
- Recklessness and disregard for safety of self or others
- Irresponsibility or failure to sustain work or honor financial obligations
- Lack of remorse for actions

Management
-Psychotherapy is treatment of choice
-Caution use of pharmacotherapy for anxiety or depression due to high addiction potential for these patients

Prognosis
-Usually chronic
-May have improvement of symptoms with aging

Histrionic Personality Disorder

-More common in women

Signs & symptoms
-Attention-seeking behavior
-Excessive emotionality
-Dramatic, flamboyant, and extroverted
-Unable to form long-lasting, meaningful relationships
-Sexually inappropriate and provocative

Differential
-Borderline personality disorder: more likely to suffer from depression and suicide attempts, where histrionic personality disorders are generally more functional

Workup
-DSM-IV criteria: pattern of excessive emotionality and attention seeking, present by early adulthood and in a variety of contests, with at least 5 of the following present:

- Uncomfortable when not the center of attention
- Inappropriate seductive or provocative behavior
- Uses physical appearance to draw attention to self
- Has speech that is impressionistic and lacking in detail
- Theatrical and exaggerated expression of emotion
- Easily influenced by others or situations
- Perceives relationships as being more intimate than they really are

Management
-Psychotherapy is treatment of choice

Prognosis
-Chronic course
-May improve with age

Narcissistic Personality Disorder

Signs & symptoms
-Sense of superiority
-Need for admiration
-Lack of empathy
-Consider themselves to be "special"
-Will exploit others for own gain
-Have an underlying fragile self-esteem
-Become depressed when they don't get the recognition they feel they deserve

Differential
-Antisocial personality disorder: desire material gain or subjugation of others while NPD desires status and recognition

Workup
-DSM-IV criteria: pattern of grandiosity, need for admiration, and lack of empathy begging by early adulthood and present in a variety of contexts with 5+ of the following present:

- Exaggerated sense of self-importance
- Preoccupied with fantasies of unlimited money, success, brilliance, etc.
- Believes that they are "special" or unique and can only associated with other high-status individuals
- Needs excessive admiration
- Has a sense of entitlement
- Takes advantage of others for self-gain
- Lacks empathy
- Envious of others or believes others are envious of them
- Arrogant or haughty

Management
-Psychotherapy is treatment of choice
-Antidepressants or lithium may be used for comorbid mood disorder

Prognosis
-Chronic
-Higher incidence of depression and midlife crises

Cluster C Personality Disorders

Avoidant Personality Disorder

Signs & symptoms
-Intense fear of rejection
-Avoid situations where rejection may occur
-Avoidance of social interactions
-Seek jobs with little interpersonal contact
-Desire companionship but are extremely shy and easily injured
-Comorbid anxiety and depressive disorders

Differential
-Schizoid personality disorder: have no desire for companionship while APD desires being with others but are too scared
-Social phobia (social anxiety disorder): not an integral part of the individual's personality and involves fear of embarrassment in a specific kind of situation whereas APD is an overall fear of rejection
-Dependent personality disorder: aggressively seeks relationships whereas APD are slow to get involved; both types can be clingy

Workup
-DSM-IV criteria: a pattern of social inhibition, hypersensitivity, and feelings of inadequacy since early adulthood, with at least 4 of the following:
- Avoids occupation that involves interpersonal contact due to a fear of criticism and rejection
- Unwilling to interact unless certain of being liked
- Cautious of interpersonal relationships
- Preoccupied with being criticized or rejected in social situations
- Inhibited in new social situations because they feel inadequate
- Believes they are socially inept and inferior
- Reluctant to engage in new activities for fear of embarrassment

Management
-Psychotherapy with assertiveness training
-β-blockers for autonomic symptoms of anxiety
-SSRIs for comorbid depression

Prognosis
-Usually chronic
-Particularly difficult during adolescence

Dependent Personality Disorder

-More common in women

Signs & symptoms
-Poor self-confidence
-Fear of separation
-Excessive need to be taken care of
-Allows others to make decisions for them
-Feelings of helplessness when left alone
-Prone to depression, particularly after loss of person on whom they are dependent

Differential
-Avoidant personality disorder
-Borderline personality disorder and histrionic personality disorder: inability to form long-lasting relationships while DPD usually have a long-lasting relationship with one individual

Workup
-DSM-IV criteria: a pattern of submissive and clinging behavior due to excessive need to be taken care of, with at least 5 of the following present:
- Difficulty making everyday decisions without reassurance from others
- Needs others to assume responsibilities for most areas of their life
- Can't express disagreement because of fear of loss of approval
- Difficulty initiating projects because of lack of self-confidence
- Goes to excessive lengths to obtain support from others
- Feels helpless when alone
- Urgently seeks another relationship when one ends
- Preoccupied with fears of being left to take care of self

Management
-Psychotherapy
-Treatment of comorbid anxiety or depression

Prognosis
-Usually chronic
-Symptoms may improve with age

Obsessive-Compulsive Personality Disorder

-More common in men and in oldest children
-Familial predisposition

Signs & symptoms
-Pattern of perfectionism, inflexibility, and orderliness
-Preoccupation with unimportant details causes inability to complete simple tasks in a timely fashion
-Appear serious, stiff, and formal
-Constricted affect
-Successful professionally but with poor interpersonal skills

Differential
-OCD: have recurrent obsessions and compulsions while OCPD don't; OCD is ego-dystonic while OCPD is ego-syntonic (aren't aware of their problem)
-Narcissistic personality disorder: achievements are motivated by status whereas OCPD patients are motivated by the work itself

Workup
-DSM-IV criteria: pattern of preoccupation with orderliness, control, and perfectionism at the expense of efficiency, present by early adulthood and in a variety of contests, with at least 4 of the following present:
- Preoccupation with details, rules, lists, and organization such that the major point of the activity is lost
- Perfectionism that is detrimental to completion of task
- Excessive devotion to work
- Excessive conscientiousness and scrupulousness about morals and ethics
- Will not delegate tasks
- Unable to discard worthless objects
- Miserly
- Rigid and stubborn

Management
-Psychotherapy

Prognosis
-Course is unpredictable
-Some will develop OCD, schizophrenia, or major depressive disorder

Personality Disorder NOS

-Reserved for personality disorders that don't fit into cluster A, B, or C

Other subtypes
-Depressive personality disorder
-Sadomasochistic personality disorder
-Sadistic personality disorder

Passive-aggressive personality disorder
-Stubborn, inefficient procrastinators
-Alternation between compliance and defiance
-Passively resist fulfillment of tasks
-Frequently make excuses for self
-Lack assertiveness
-Attempt to manipulate others to do chores and errands
-Frequently complain about own misfortunes
-Treated with psychotherapy

PSYCHOTIC DISORDERS

-Psychosis involves a break from reality and involves delusions, perceptual disturbances, and/or disordered thinking

Differential
-Psychosis secondary to general medical condition: CNS disease, endocrinopathies, nutritional deficiency, connective tissue disease, porphyria
-Substance-induced psychosis
-Delirium
-Dementia
-Bipolar disorder
-Major depression with psychotic features
-Schizophrenia, schizophreniform disorder, or schizoaffective disorder
-Delusional disorder

Psychotic S/S	Examples	Definition
Disturbed thought content	*Delusions*	-Fixed, bizarre, unrealistic beliefs not subject to rational argument and not accounted for by accepted cultural or religious beliefs -May be delusions of paranoia, grandeur, religion, nihilism, guilt, or somatic delusions -Includes ideas of reference (a belief or perception that irrelevant, unrelated or innocuous phenomena in the world refer to a person directly or have special personal significance) and thought broadcasting (the belief that one's thoughts can be heard by others)
	Paranoid thoughts	-General mistrust or suspicion -Beliefs are plausible but false -Elaborate delusional systems
	Loss of ego boundaries	-Perceived loss of boundaries between self and the environment
	Suicidal or homicidal thoughts	
	Phobias	
	Obsessions	-Commonly about contamination, losing control, harm, unwanted sexual thoughts, religious, or perfectionism
	Compulsions	-Washing and cleaning, checking, repeating, mental review of events o prevent harm, counting, cancelling or undoing, collecting, arranging
Disturbed thought processes	*Tangentiality*	-Mental condition in which one tends to digress from the topic under discussion, especially by word association
	Loosening of associations	-A disorder of thinking in which associations of ideas become so shortened, fragmented, and disturbed as to lack logical relationship
	Poverty of thought	-A global reduction in the quantity of thought and thought perseveration where a person keeps returning to the same limited set of ideas
	Thought blocking	-When a person's speech is suddenly interrupted by silences that may last a few seconds to a minute or longer
Abnormal speech	*Poverty of speech*	-A general lack of additional, unprompted content seen in normal speech
	Verbigeration	-An obsessive repetition of meaningless words and phrases
	Mutism	-Unwillingness or refusal to speak
	Neologisms	-Making up words

	Clang associations	-A mode of speech characterized by association of words based upon sound rather than concepts
Perceptual disturbances	*Illusions*	-Stimulus is real but is misinterpreted
	Hallucinations	-Manufacturing a stimulus that is not really present -May be auditory (common with schizophrenia), visual or tactile (EtOH and opiate withdrawal), olfactory, or gustatory

Postpartum Psychosis

-Rare
-Thought to be a manifestation of bipolar I disorder

Causes
-Genetic predisposition
-Recent d/c of Li or other mood stabilizer

Signs & Symptoms
-Hallucinations, delusions, mania, and/or depression
-Waxing and waning delirium
-Usually within 2 weeks of childbirth

Differential
-Postpartum depression with psychotic features
-Schizoaffective disorder
-Schizophrenia

Management
-Increased risk of suicide and infanticide during psychosis = hospitalization with separation from infant required
-1st line therapy is atypical antipsychotic
-Adjunct therapy with benzos
-Treat underlying disorder, may need antidepressants or mood stabilizers

Schizophrenia

-Rarely presents before 15 or after 45
-Associated with lower SES

Causes
-Strong genetic predisposition
-Neurotransmitter abnormalities
-Seasonal theory: people born in winter and early spring have higher incidence

Phases
1.) Prodromal phase: functional decline that precedes the first psychotic episode, where pt may become socially withdrawn, irritable, have physical complaints, or newfound interest in religion or the occult
2.) Psychotic phase: perceptual disturbances, delusions, disordered thought
3.) Residual phase: occurs between episodes of psychosis; marked by flat affect, social withdrawal, and odd thinking or behavior

Signs & Symptoms
-Positive symptoms: hallucinations, delusions, bizarre behavior, disordered thought
-Negative symptoms: blunted affect, anhedonia, apathy, inattentiveness
-Disheveled appearance
-Intact memory and orientation
-Concrete understanding of similarities or proverbs
-Lacks insight into disease
-Delusions may be bizarre or non-bizarre and can be grandiose, paranoid, nihilistic, or erotomatic
-Often has comorbid substance abuse

Differential
-Major depressive or manic episode with psychotic features
-Drug or substance abuse or withdrawal
-Dementia
-Delirium
-CNS disease
-Nutritional deficiency
-Heavy metal poisoning
-Endocrinopathy
-Other psychotic disorder: schizoaffective, schizophreniform, delusional disorder, brief psychotic disorder
-Personality disorder

Workup
-DSM-IV criteria: illness causes significant social or occupational functional deterioration, duration of illness for at least 6 months, and 2+ of the following for at least 1 month:
- Delusions
- Hallucinations
- Disorganized speech
- Grossly disorganized or catatonic behavior
- Negative symptoms

Management
-Antipsychotic therapy is 1st line for acute and maintenance therapy
-Avoid clozapine and olanzapine unless symptoms are refractory due to adverse effects
-Can augment antipsychotic treatment with CBT, social skills training, supported employment, and assertive community treatment

Prognosis
-Disease is usually chronic and debilitating
-40-50% remain significantly impaired after diagnosis
-Worse prognostic indicators: early onset, poor social support, negative symptoms, FH, gradual onset, male sex, many relapses, poor premorbid functioning

Paranoid Type Schizophrenia	Disorganized Type Schizophrenia	Catatonic Type Schizophrenia	Undifferentiated Type Schizophrenia	Residual Type Schizophrenia
-Highest functioning -Older age of onset -Preoccupation with 1+ delusions or frequent auditory hallucinations -No predominance of disorganized speech, disorganized or catatonic behavior, or inappropriate affect	-Poor functioning -Early onset -Disorganized speech and behavior -Flat or inappropriate affect	-Rare -Motor immobility or excessive purposeless motor activity -Extreme negativism or mutism -Peculiar voluntary movements or posturing -Echolalia or echopraxia	-Has features of more than one schizophrenia type or none of the types	-Prominent negative symptoms -Minimal evidence of positive symptoms

Related Disorders

Schizophreniform disorder
-Same criteria as schizophrenia but symptom duration is < 6 months
-Manage with hospitalization, 3-6 mos of antipsychotics, supportive psychotherapy

Schizoaffective disorder
-DSM-IV criteria:
- Meets criteria for MDD or manic episode or mixed episode
- Additionally has had delusions or hallucinations for at least 2 weeks in absence of mood disorder symptoms (= period of time where psychotic symptoms don't coexist with mood symptoms; otherwise this would be mood disorder with psychotic features)
- However, mood symptoms are present for substantial portion of psychotic illness
- Symptoms not attributable to general medical condition or drugs

-Manage with hospitalization, supportive psychotherapy, short-term antipsychotics, maintenance therapy with mood stabilizers or antidepressants

Brief psychotic disorder
-Rare
-Same criteria for schizophrenia but duration is 1 day to 1 month

Delusional disorder
-Delusions may be erotomanic, grandiose, somatic, persecutory, jealous, or mixed
-DSM-IV criteria:
- Nonbizarre, fixed delusions for at least 1 month (schizophrenia may be bizarre or nonbizarre)
- Does not meet criteria for schizophrenia

Functioning in life not significantly impaired

SOMATOFORM DISORDERS

-Not all patients with somatization have a true disorder, but it is more likely to be a psychiatric illness if there are many organ systems involved in symptoms, if symptoms fluctuate, if there is comorbid anxiety or depression, if symptoms lead to psychological or emotional gain, if symptoms are chronic, or if there is idiosyncratic response to meds

Differential
-Factitious disorder = intentionally faking physical or mental in order to assume the sick role, without gain of external incentives, frequently present with wound healing problems, excoriations, infection, bleeding, hypoglycemia, and GI ailments, often have health care work experience or h/o abuse or neglect
-Munchausen syndrome: a factitious disorder with predominantly physical complaints, often demand specific meds, highly skilled at feigning symptoms necessitating hospitalization
-Munchausen by proxy: intentionally producing symptoms in someone else who is under one's care in order to assume to sick role by proxy
-Malingering = intentionally faking or grossly exaggerating symptoms for an obvious incentive such as avoiding work or criminal prosecution, obtaining financial compensation, room and board, or obtaining medications
-Depression
-Panic disorder
-Substance abuse

Management of somatoform disorders
-Investigate all symptoms
-Don't try to reason away symptoms as they are not a conscious process
-Focus on care rather than cure: provide reassurance and schedule brief, regular PCP visits that don't coincide with symptoms
-Insight-directed psychotherapy
-Hypnosis
-Relaxation therapy
-Treat comorbid psychiatric conditions
-Minimize polypharmacy and secondary gain

Conversion Disorder

-The most common somatoform disorder
-Increased incidence in women and low SES

Signs & symptoms
-Voluntary motor or sensory deficits that suggest neurologic condition but are medically unexplainable
-Preceded by psychological distress, can have relapses
-Typically there is onset of a dramatic but physiologically impossible condition such as shifting paralysis, aphonia, blindness, deafness, feeling a lump in the throat, or pseudoseizures
-Pt is surprisingly calm and unconcerned when describing dramatic symptoms ("la belle indifference")
-Focus is on one symptom rather than multiple in somatization disorder
-Comorbid schizophrenia, major depression, or anxiety disorders

Differential
-Underlying medical cause (50% will eventually receive)

Workup
-DSM-IV criteria:
1. At least 1 neuro symptom
2. Psychological factors are associated with initiation or exacerbation of symptom
3. Symptom not intentionally produced
4. Can't be explained by medical condition or substance use
5. Causes significant distress or impairment in social or occupational functioning
6. Not accounted for by somatization disorder or other mental disorder
7. Not limited to pain or sexual symptoms

Prognosis
-Symptoms usually resolve within 1 month, 25% will have relapses

Somatization Disorder	Hypochondriasis	Other Somatoform Disorders
-More common in females -Greater prevalence with low SES -Genetic and familial predisposition **Signs & symptoms** -Multiple vague complaints involving many organ systems -Long history of numerous office visits **Workup** -DSM-IV criteria: 1. At least 2 GI symptoms 2. At least 1 sexual symptom 3. At least 1 neuro symptom 4. At least 4 pain symptoms 5. Onset before age 30 6. Can't be explained by general medical condition or substance use	**Signs & symptoms** -Prolonged, exaggerated concern about healthy and possible illness -Pt fear disease or are convinced one is present -Misinterpret normal bodily symptoms as indicative of disease -Waxing and waning of symptoms, with exacerbation when under stress -Comorbid MDD or anxiety disorder **Differential** -Somatization disorder: focus is on symptoms, whereas hypochondriacs focus on disease **Workup** -DSM-IV criteria: 1. Fear of having a serious medical condition based on misinterpretation of normal bodily symptoms 2. Fears persist despite appropriate medical evaluation 3. Fears present for at least 6 months	**Body dysmorphic disorder** -Preoccupation with an imagined or exaggerated defect in physical appearance -Spend significant time trying to correct perceived flaws with makeup, derm procedures, or plastic surgery -Comorbid depression, anxiety, or psychotic disorder -SSRIs may reduce symptoms **Somatoform pain disorder** -Pain in one or more sites associated with psychological factors that have an important role in the onset, severity, exacerbation, or maintenance of the pain -Pain not due to medical disorder -Symptom is not intentionally produced or feigned -SSRIs may be useful **Undifferentiated somatoform disorder** -"Somatoform light" -For patients not fitting existing category criteria -Diagnostic criteria: 1+ medically unexplained physical complaints persisting for more than 6 months

SUBSTANCE USE DISORDERS

Substance Abuse	Substance Dependence
-DSM-IV criteria: pattern of substance use leading to impairment or distress for at least 1 year with 1+ of the following manifestations: • Failure to fulfill obligations at work, school, or home • Use in dangerous situations • Recurrent substance-related legal problems • Continued use despite social or interpersonal problems due to the substance use	-AKA "addiction" although this is not considered a scientific term -DSM-IV criteria: substance use leading to impairment or distress manifested by at least 3 in a 12-month period: • Tolerance: the need for increased amounts of substance to achieve the desired effect or diminished effect if using the same amount of substance • Withdrawal: the development of a substance-specific syndrome due to cessation of use that has been heavy and prolonged • Using substance more than originally intended • Persistent desire or unsuccessful efforts to cut down on use • Significant time spent in getting, using, or recovering from substance • Decreased social, occupational, or recreational activities because of use • Continued use despite subsequent physical or psychological problems

Cocaine Abuse & Dependence

-Cocaine blocks DA reuptake from the synaptic cleft → stimulant effect

Signs & symptoms of abuse
-Needle or track marks
-Nasal septal perforation

Signs & symptoms of intoxication
-Euphoria
-HTN or hypotension
-Tachycardia or bradycardia
-Dilated pupils
-Weight loss
-Psychomotor agitation or depression
-Chills
-Diaphoresis
-Respiratory depression
-Seizures
-Arrhythmias
-Tactile hallucinations
-MI or CVA from vasoconstriction

Withdrawal
-Cocaine withdrawal symptoms last 1-2 weeks and are predominately psychosocial
-Malaise, fatigue, depression, hunger, constricted pupils, vivid dreams, psychomotor agitation or retardation

Differential
-Amphetamine or PCP intoxication
-Sedative withdrawal

Workup
-Cocaine can be detected in urine for 2-4 days, up to 14 for heavy users (perpetuated by alcohol use as well)

Management of acute intoxication
-Inpatient
-Benzos for mild agitation
-Haloperidol for severe agitation
-Control HTN and arrhythmias

Management of dependence
-Cocaine withdrawal can be managed outpatient
-Tell patient to sleep off the crash
-Psychotherapy
-TCAs
-DA agonists: bromocriptine, amantadine

Amphetamine Abuse & Dependence

-Classic amphetamines: dextroamphetamine (Dexedrine), methylphenidate (Ritalin), methamphetamine (crystal meth, speed) → cause release of DA from nerve endings → stimulant effects
-Designer amphetamines: MDMA (ecstasy), MDEA (Eve) → release DA and serotonin from nerve endings → stimulant effects as well as hallucinogenic

Signs & symptoms of intoxication
-Similar to cocaine intoxication
-Psychosis

Withdrawal
-Similar to cocaine

Differential
-Cocaine or PCP intoxication

Workup
-UDS will be + for 1-2 days although most are not very sensitive

Management of acute intoxication
-Treat with lorazepam, activated charcoal, NS IVF

Management of dependence
-Similar to cocaine

Sedative or Hypnotic Abuse & Intoxication

-Benzodiazepines, barbiturates (phenobarbital), and GHB (date rape drug)
-Potentiate effects of GABA

Signs & symptoms of intoxication
-Drowsiness
-Slurred speech
-Incoordination and ataxia
-Mood lability
-Impaired judgment
-Nystagmus
-Respiratory depression
-Coma

Withdrawal
-Autonomic hyperactivity: tachycardia, sweating, insomnia, anxiety, tremor, n/v, delirium, hallucinations
-Seizures

Differential
-Alcohol intoxication
-Delirium

Workup
-UDS will be + for 1 week

Management of acute intoxication
-Activated charcoal
-Flumazenil for benzo overdose
-Alkalinize urine with sodium bicarb for barbiturate overdose to facilitate renal clearance
-Support respiratory status and BP

Management of dependence
-Can't abruptly DC or risk death!
-Tapered dose of long-acting benzo
-Tegretol or valproate for seizures control

Phencyclidine (PCP) Abuse & Dependence

-AKA "angel dust"
-NMDA gluatamate-R antagonist
-Dopaminergic neuron activator
-Similar to the anesthetic ketamine

Signs & symptoms of intoxication
-Recklessness
-Impulsiveness
-Impaired judgment
-Assaultiveness
-Rotatory nystagmus
-Ataxia
-HTN → ICH, MI, or aortic dissection
-Tachycardia
-Muscle rigidity
-High pain tolerance
-Seizures or coma with overdose
-Hyperthermia
-Hyponatremia
-DIC
-Rhabdomyolysis
-Serotonin syndrome

Withdrawal
-No withdrawal syndrome is observed
-May have "flashbacks"

Differential
-Acute psychosis
-Schizophrenia

Workup
-UDS will be + for > 1 week
-Elevated CPK and AST

Management of acute intoxication
-Monitor BP, temp, electrolytes
-Acidify urine with ammonium chloride and vitamin C to facilitate clearance
-Benzos to control agitation, muscle spasms, and seizures
-Haloperidol for severe agitation or psychosis

Opiate and Opioid Abuse & Dependence

-Opiates are naturally occurring chemical compounds include opium, morphine, and codeine
-Opioids are synthetic chemicals that bind to these same receptors (heroin, hydrocodone, hydromorphone, oxycodone, oxymorphone, buprenorphine, fentanyl, methadone, tramadol, dextromethorphan, meperidine (Demerol))
-We all have a small amount of endogenous opiates (endorphins) that normally act on these receptors

Signs & symptoms of intoxication
-Drowsiness
-N/v
-Constipation
-Slurred speech
-Constricted pupils (except for meperidine which dilates!)
-Seizures
-Respiratory depression

Withdrawal
-Begins in 8 hours and lasts up to 3 days: anxiety, insomnia, yawning, stomach cramps, lacrimation, rhinorrhea, diaphoresis, vomiting, diarrhea, fever, chills, tremor, tachycardia, piloerection, HTN, seizures

Differential
-Sedative or hypnotic intoxication
-Severe EtOH intoxication

Workup
-Opioid metabolites can be detected within 4 days of last use or longer in chronic users (false + in rifampin, quinolone, or poppy seed ingestion)

Management of acute intoxication
-Naloxone
-May need respiratory support

Management of dependence
-Withdrawal is NOT life-threatening
-Withdrawal can be treated with clonidine and/or buprenorphine for moderate symptoms or with methadone for severe symptoms

Hallucinogen Abuse

-Includes psilocybin (shrooms), mescaline (peyote), and lysergic acid diethylamide (LSD)
-Dependence and withdrawal do not occur with hallucinogens

Signs & symptoms of intoxication
-Perceptual changes
-Pupillary dilation
-Tachycardia
-Tremors
-Incoordination
-Sweating
-Palpitations
-LSD intoxication will have awake pt who is aware symptoms are drug-induced

Workup
-Hallucinogens are not detected on urine drug screen

Management of acute intoxication
-Guidance and reassurance
-Pts can be put in a quiet room until symptoms abate, PRN lorazepam

Sequelae
-Pts can experience "flashbacks" and symptom recurrence later in life due to reabsorption from lipid stores

Marijuana (Ab)use

-Active component is THC which acts on cannabinoid receptors in the brain
-No dependence or true withdrawal syndrome

Medicinal uses
-Depression
-Antiemetic in cancer patients
-Appetite stimulant in AIDS pts

Signs & symptoms of intoxication
-Euphoria
-Impaired coordination
-Mild tachycardia
-Conjunctival injection
-Dry mouth
-Increased appetite

Discontinuation effects
-Lasts 7-14 days or up to several weeks and has symptoms of sleep disturbance, irritability, and physical tension

Workup
-Cannabinoids can be detected in urine for 7-10 days in a casual user, 2-4 weeks in a heavy user, and months in a chronic heavy user

Inhalant Abuse

-Includes solvents, glue, paint thinners, fuels, and isobutyl nitrates → act as CNS depressants
-No dependence or withdrawal syndrome

Signs & symptoms of intoxication
-Impaired judgment
-Belligerence
-Impulsivity
-Perceptual disturbances
-Lethargy
-Dizziness
-Nystagmus
-Slurred speech
-Euphoria
-Stupor or coma
-Respiratory depression: can be fatal
-Damage to CNS, PNS, liver, kidneys, and muscle with long-term use

Discontinuation effects
-Irritability and hallucinations
-N/v
-Tachycardia

Workup
-Serum drug screen is + for 4-10 hours

Management of acute intoxication
-ABCs

Management of abuse
-Psychotherapy

Tobacco Dependence

Screening
-Readiness assessment: 5 A's (ask about smoking status, give clear personalized advice, assess readiness to quit and barriers, assist with quit resources, arrange for quit date and f/u

Withdrawal symptoms
-Onset of symptoms 2-3 hours after last tobacco use with peak in 2-3 days, resolution in 1 month after quitting
-Increased symptoms if > 25 cigs per day, first cig within 30 min of waking, discomfort if forced to refrain from smoking

Nonpharmacologic management
-STAR: set quit date 2 weeks out, tell family and friends, anticipate challenges, remove tobacco products from environment
-Assess dependence level and suggest dosing using Fagerstrom questionnaire
-Nonpharmacologic methods: cold turkey, unassisted tapering, assisted tapering (QuitKey), formal cessation programs, aversion therapy, acupuncture, hypnotherapy, massage therapy
-Electronic cigarette: recent safety issues with battery igniting, not FDA approved

Prognosis
-Tobacco smoke increases risk of certain cancers: lung, laryngeal, oral cavity, esophagus, bladder, kidney, pancreas, uterus, and cervix

Pharmacologic Management: Nicotine Replacement Therapy

-Good combination therapy: nicotine patch + lozenge or gum, nicotine patch + nicotine inhaler, nicotine patch + bupropion SR
-Increases likelihood of successful quit by 2-3x
-Low abuse potential
-Pt must not use any tobacco products while using
-Most formulations are available OTC although minors need a Rx
-Cautions: underlying CV disease (as nicotine causes ↑HR and BP), recent MI, serious arrhythmias, serious or worsening angina, pregnancy, lactation

Transdermal patch	Nicotine gum	Lozenge	Inhaler	Nasal spray
-Nicoderm -Avoids first pass metabolism -Can't cut patches as this causes nicotine evaporation and ↓ effectiveness -Must remove patches before MRI to avoid burns -Not for relief of acute cravings -AEs: skin irritation, insomnia, nightmares or vivid dreams	-Nicorette, generics -Need to chew n' tuck -Can't eat or drink for 15 before and after -Helps with acute ravings -AEs: n/v, abd pain, hiccups, mouth irritation, sore jaw, unpleasant taste -Contraindicated with TMJ, poor dentition, dentures	-Commit, Nicorette -Can't chew, need to let large lozenge dissolve over 30 min (although new mini lozenge will dissolve faster) -Dose based on time to first cigarette -Helps with acute craving relief -AEs: mouth irritation or ulcers, abd pain, n/v/d, HA, palps	-Nicotrol -Rx only -Benefit of hand-to-mouth behavior -Not meant to be inhaled all the way into lungs -Open cartridge only potent for 24 hours -AEs: mouth and throat irritation, cough -Caution with severe reactive airway disease	-Nicotrol NS -Rx only -Benefit of nicotine bolus that mimics burst from cigs → fast reduction of cravings but ↑ abuse potential -AEs: local nasopharyngeal irritation, runny nose, sneezing, cough, throat and eye irritation, HA -Caution with severe reactive airway disease

Other Pharmacologic Management

Bupropion
-1-2 daily doses, starting 1 week prior to quit date to allow time for accumulation of norepinephrine and DA in the body
-Try at least 7 weeks before d/c
-Best option for patients with CV disease
-AEs: insomnia, dry mouth, suicidality
-Contraindications: pt with seizure disorder, pt with h/o anorexia or bulimia, pts undergoing abrupt d/c of ethanol or sedatives

Varenicline (Chantix)
-Blocks nicotine from cigs from binding
-Begin 7 days before quit date, with differing doses throughout treatment
-AEs: insomnia, nausea, abnormal dreams, impaired driving or operating machinery, suicide risk (accounts for most cases of suicide attempt while undergoing smoking cessation), CV risk

Other 2nd line therapies:
-Nortriptyline
-Clonidine

Alcohol Abuse & Dependence

-The most commonly abused substance in the US
-Strong genetic risk factors, with heritability similar to DM or HTN
-Associated health risks include HTN, a-fib, cardiomyopathy, esophagitis, gastritis, upper GIB, pancreatitis, hepatitis, cirrhosis
-Related illnesses include pneumonia, TB, and cancers of the breast, liver, throat, and esophagus
-Alcohol activates GABA-R and serotonin-R and inhibits glutamate-R

Screening
-CAGE (2+ is positive):
1. Wanted to cut back on drinking
2. Annoyed by criticism of drinking
3. Guilty about drinking
4. Needed an eye opener

Signs & symptoms
-Displays at risk drinking habits, defined as >14 drinks per week for men or > 7 for women
-Wernicke-Korsakoff syndrome from untreated Wernicke's encephalopathy: impaired recent memory, anterograde amnesia, confabulation

Differential for intoxication
-Hypoglycemia
-Hypoxia
-Drug overdose
-Ethylene glycol or methanol poisoning
-Hepatic encephalopathy
-Psychosis
-Psychomotor seizures

Withdrawal
-Triggered by abrupt cessation or reduction of intake in dependent individuals
-Caused by new homeostatic set point of increased inhibitory tone (activates GABA-R) as well as inhibition of excitatory tone → alcohol tolerance where individuals retain arousal at concentrations of GABA that would produce lethargy or coma in a normal adult
-Onset in 12-24 hours after last drink, with peak intensity at 24-48 hours
-Lasts 4-7 days
-Most serious form is delirium tremens; 5% of those hospitalized for EtOH withdrawal will develop
-Mortality with treated DT is ~5%; untreated 15-20%

Minor withdrawal	Tremulousness, mild anxiety, headache, diaphoresis, palpitations, anorexia, GI upset; Normal mental status	6 to 36 hours
Seizures	Single or brief flurry of generalized, tonic-clonic seizures, short post-ictal period; Status epilepticus rare	6 to 48 hours
Alcoholic hallucinosis	Visual, auditory, and/or tactile hallucinations with intact orientation and normal vital signs	12 to 48 hours
Delirium tremens	Delirium, agitation, tachycardia, hypertension, fever, diaphoresis	48 to 96 hours

Outpatient management
-Brief intervention for nondependent alcohol abusers
-Alcoholics Anonymous
-Disulfiram (Antabuse)
-Psychotherapy
-SSRIs
-Can consider outpatient management of dependence if there are no risk factors

Inpatient management
-Needed for possible withdrawal if there is h/o seizures, delirium, mental instability, suicidal or homicidal ideation, psychosis, unstable environment, or no support or transportation available
-Thiamine (given to prevent Wernicke's encephalopathy, especially before giving glucose), folate, and B6 supplementation
-Supplement Mg (alcoholics chronically deficient, also an essential cofactor in thiamine metabolism)
-IV benzodiazepines every 10-15 minutes PRN per CIWA protocol
-Phenobarbital or propofol for refractory DTs

Prognosis
-Wernicke-Korsakoff syndrome may be irreversible

PERVASIVE DEVELOPMENTAL DISORDERS

-Includes autism, Asperger's syndrome, Rett's syndrome, and childhood disintegrative disorder
-Now considered to be a biologic rather than psychologic disorder, more related to mental retardation
-Highly genetic basis with possible environmental factors

Autism

Differential (many of these can co-exist with autism)
-Rett syndrome (almost exclusively in girls): genetic mutation of MECP2 on X chromosome with initial normal development, then onset of loss of hand skills and social development, decreasing rate of growth of head circumference, problems with gait or trunk movements, severely impaired language and psychomotor development, seizures, and cyanotic spells
-Fragile X syndrome
-Angelman syndrome
-Turner syndrome
-William syndrome
-Asperger's syndrome: will have normal language and cognitive development
-Pervasive developmental disorder NOS: don't meet criteria for autism or are still very young for diagnosis
-Childhood disintegrative disorder: normal development for first 2 years of life, then loss of previously acquired skills in language, social skills, bowel or bladder control, play, or motor skills

Screening
-MCHAT administered between 16-30 months (usually at 18 mo well child check)

Signs & symptoms
-Markedly impaired eye contact (red flag: lack of joint attention)
-Failure to develop peer relationships
-Not seeking to share enjoyment or interests (red flag: doesn't look up for approval by 2-3 years)
-Lack of social or emotional reciprocity
-Delayed or absent spoken language without attempt to compensate with gestures or mime (red flags: no words by 18 mo, no strings of words by 2 years)
-Repetitive language
-Inability to initiate and sustain conversation
-Lack of spontaneous make-believe play appropriate for developmental level
-Repetitive motor mannerisms (rocking, spinning)
-Preoccupation with parts of objects
-Strong fixations to objects or restricted interests ("little professor")
-Inflexible adherence to rigid routines
-May also exhibit sensory seeking or avoidant behavior
-Tantrums set off by noise or changes in routine
-Comorbid mental retardation or seizure disorder

Workup
-Diagnosis is clinical
-Send for comprehensive medical evaluation after failed MCHAT or parental concern: basic language and developmental testing via a developmental pediatrician, psychologist, and speech therapist, audiology screen, genetic microarray testing

Management
-Goals are to maximize functioning, move child towards independence, and improve quality of life
-Applied behavioral analysis is the best tested method of autism treatment
-Language therapy: focuses on pictures and visual communication
-Social skills groups
-Occupational therapy to aid stimuli sensitivity
-Gluten and casein-free diet
-Consider meds to target specific symptoms: methylphenidate for inattention or hyperactivity, risperidone for aggression and self-injury, fluoxetine for repetitive behaviors or anxiety or depression, atypical antipsychotic or SSR for dysregulated mood, melatonin for sleep disturbance

Diagnostic criteria for Asperger disorder

A. Qualitative impairment in social interaction, as manifested by at least two of the following:
1. Marked impairments in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction
2. Failure to develop peer relationships appropriate to developmental level
3. A lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (eg, by a lack of showing, bringing, or pointing out objects of interest to other people)
4. Lack of social or emotional reciprocity
B. Restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:
1. Encompassing preoccupation with one or more stereotyped and restricted patterns of interest that are abnormal either in intensity or focus
2. Apparently inflexible adherence to specific, nonfunctional routines or rituals
3. Stereotyped and repetitive motor mannerisms (eg, hand or finger flapping or twisting, or complex whole-body movements)
4. Persistent preoccupation with parts of objects
C. The disturbance causes clinically significant impairment in social, occupational, or other important areas of functioning
D. There is no clinically significant general delay in language (eg, single words used by age two years, communicative phrases used by age three years)
E. There is no clinically significant delay in cognitive development or in the development of age-appropriate self-help skills, adaptive behavior (other than social interaction), and curiosity about the environment in childhood
F. Criteria are not met for another specific pervasive developmental disorder or schizophrenia

Prognosis
-Early detection and treatment can affect course of disease

Diagnostic criteria for autistic disorder

A. A total of six or more items from 1., 2., and 3., with at least two from 1., and one each from 2. and 3.:
1. Qualitative impairment in social interaction, as manifested by at least two of the following:
a. Marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body posture, and gestures to regulate social interaction
b. Failure to develop peer relationships appropriate to developmental level
c. A lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (eg, by a lack of showing, bringing, or pointing out objects of interest)
d. Lack of social or emotional reciprocity
2. Qualitative impairments in communication as manifested by at least one of the following:
a. Delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime)
b. In individuals with adequate speech, marked impairment in the ability to initiate or sustain conversation with others
c. Stereotyped and repetitive use of language or idiosyncratic language
d. Lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level
3. Restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:
a. Encompassing preoccupation with one or more stereotyped and restricted patterns of interest that are abnormal either in intensity or focus
b. Apparently inflexible adherence to specific, nonfunctional routines or rituals
c. Stereotyped and repetitive motor mannerisms (eg, hand or finger flapping or twisting, or complex whole-body movements)
d. Persistent preoccupation with parts of objects
B. Delays or abnormal functioning in at least one of the following areas, with onset prior to age three years: 1. social interaction, 2. language as used in social communication, or 3. symbolic or imaginative play
C. The disturbance is not better accounted for by Rett disorder or childhood disintegrative disorder (CDD)

SEXUAL DISORDERS

Paraphilia

-Sexual disorders characterized by engagement in unusual sexual activities or preoccupation with unusual sexual urges or fantasies for at least 6 months that cause impairment in daily functioning

Signs & Symptoms
-Most common paraphilias are pedophilia, voyeurism, and exhibitionism

Management
-Insight-oriented psychotherapy, behavior therapy
-Antiandrogens in men

Prognosis
-Poor prognosis with early age of onset, comorbid substance abuse, high frequency of behavior, and related arrest causing presentation for treatment
-Good prognosis with self-referral for treatment, sense of guilt associated with behavior, and history of otherwise normal sexual activity in addition to the paraphilia

Sexual Dysfunction

Differential
-Atherosclerosis
-DM
-Pelvic adhesions → dyspareunia
-Low estrogen or testosterone or increased progesterone
-Med AEs: antihypertensives, anticholinergics, antidepressants, antipsychotics
-Substance abuse
-Depression
-True sexual disorder: hypoactive sexual desire disorder, sexual aversion disorder, male erectile disorder, female sexual arousal disorder, orgasm disorder, dyspareunia, vaginismus

Workup
-Ask males about morning erections

Management
-True sexual disorder: dual sex therapy, behavior therapy, hypnosis
-Treat low testosterone
-Erectile disorder: yohimbine, sildenafil, vacuum pumps, constrictive rings, prosthetic surgery
-Dyspareunia: gradual desensitization, muscle relaxation techniques
-Vaginismus: vaginal dilators

INTELLECTUAL DISABILITY (MENTAL RETARDATION)

-More common in males
-85% of cases are mild

Types
-Profound MR: IQ <25
-Severe MR: IQ 25-40
-Moderate MR: IQ 40-50
-Mild MR: IQ 50-70

Causes
-Most MR has no identifiable cause
-Genetic disorders: Down's syndrome, fragile X syndrome
-Prenatal infections and toxins: toxoplasmosis, syphilis, AIDs, alcohol or drug exposure, rubella, CMV, HSV
-Perinatal complications: anoxia, prematurity, birth trauma
-Postnatal causes: hypothyroidism, malnutrition, toxin exposure, trauma, encephalitis or meningitis

Differential
-Learning disorder

Workup
-DSM-IV criteria:
1. Significantly subaverage intellectual functioning with IQ ≤ 70
2. Deficits in adaptive skills appropriate for age group
3. Onset before age 18

Learning Disorders

Causes
-Usually due to deficits in cognitive processing (abnormal attention, memory, visual perception, etc.)
-Genetic factors
-Abnormal development
-Perinatal injury
-Neurologic or medical condition

Types
-Reading disorder: occurs in 4% of kids
-Mathematics disorder: occurs in 5% of kids
-Disorders of written expression: occurs in 3-10% of kids

Differential
-Hearing or vision deficit

Workup
-DSM-IV criteria:
1. Achievement in reading, math, or written expression that is significantly lower than expected for chronological age, level of education, and level of intelligence
2. Affects academic achievement or daily activities and can't be explained by sensory deficits, poor teaching, or cultural factors

Management
-Remediation tailored to child's specific needs

OTHER BEHAVIORAL & EMOTIONAL DISORDERS

Child and Elder Abuse

Differential	Signs & Symptoms	Workup	Management
-Child: coagulopathy, salicylate ingestion, vasculitis, Mongolian spots, complimentary or alternative medicine treatments, hereditary sensory autonomic neuropathies	-Inconsistent history for mechanism of injury and injuries observed -Vague history or no history offered -Story changing -Injury attributed to actions of siblings -History inconsistent with child's developmental stage or elder's cognitive status -Implausible history -Evidence of poor caretaking -Child who is too compliant with painful or disturbing examination of injuries -Child who is pseudomature or withdrawn, passive, depressed, violent or hyperactive -Sudden onset AMS not attributed to medical illness or poisoning -Older adults: pressure ulcers, dehydration, malnutrition, failure to get needed meds	-"Skeletal survey" radiographs mandatory in children under 2 in whom abuse is suspected -Noncontrast CT for suspected inflicted head trauma -Consult social worker and abuse specialist if available -Photographs of injuries (may require parental permission in some states)	-Mandatory reporting of suspected child abuse and neglect to law enforcement and DSS -Parents must be made aware of claim and rationale and what will happen next -Community-dwelling older adults covered in most states by Adult Protective Services

Sexual Assault

Management
-Evaluation and physical examination of victim by trained personnel
-Evidence collection kit
-Empiric STD treatment
-Acute crisis counseling
-Follow-up STD and pregnancy testing

Intimate Partner Violence (Domestic Violence)

	Signs & Symptoms	Management
-Refers to actual or threatened psychological, physical, or sexual harm by a current or former partner or spouse -May begin or escalate during pregnancy	-Inconsistent explanation of injuries -Delay in seeking treatment or missed appointments -Frequent ED visits -Late prenatal care -Inappropriate affect -Overly attentive partners -Reluctance to be examined -Somatization	-Provider expression of empathy and continued ability to support and assist patient -Consult domestic violence advocate to explore resources -Caution with providing written materials that may be seen by perpetrator -Don't confront perpetrator -Restraining orders have inconsistent effectiveness

Adjustment Disorder

-An excessive, prolonged reaction to a stressful event or situation or combination of situations serious enough to impair social and occupational functioning
-More common in females and adolescents
-Further coded based on predominance of depressed mood, anxiety, disturbance of conduct (aggression), or a combination of these

Causes
-Relationship problems, financial difficulties, family conflict, school or work changes, major life changes, health problems, divorce, death, moving, sexuality issues

Signs & symptoms
-Common comorbidities: depression, anxiety, disturbance of conduct, eating disorder

Differential
-PTSD: occurs in face of life-threatening event while adjustment disorder is a result of non life-threatening event

Workup
-DSM-IV criteria:
1. Development of emotional or behavioral symptoms within 3 mos after a stressful event that produce either severe distress in excess of what would be expected or significant impairment in daily functioning
2. Symptoms are not those of bereavement.
3. Symptoms resolve within 6 months after stressor has terminated.

Management
-1st line is psychotherapy
-Treat associated symptoms: insomnia, anxiety, depression

Grief

Normal grief
-Feelings of guilt and sadness
-Mild sleep disturbance and weight loss
-Illusions of the deceased (visual or auditory)
-Attempts to resume daily activities and work
-Symptoms resolve within a year

Abnormal grief
-Feelings of severe guilt and worthlessness
-Significant sleep disturbance and weight loss
-Hallucinations or delusions
-No attempt to resume activities
-Suicidal ideation
-Symptoms persist > 1 year

Conduct Disorder

Causes
-Genetic and psychosocial factors

Signs & symptoms
-Comorbid ADD or learning disorders

Workup
-DSM-IV criteria: a pattern of behavior that involves violation of the basic rights of others or of social norms and rules, with at least 3 acts within the following categories in the past year:

A. Aggression towards people and animals
B. Destruction of property
C. Deceitfulness
D. Serious violations of others

Management
-Consistent reinforcement of firm rules
-Psychotherapy focused on behavior modification and problem-solving skills
-Antipsychotics or Li for aggression
-SSRIs for impulsivity, irritability, and mood lability

Prognosis
-40% risk of developing antisocial personality disorder in adulthood

Oppositional Defiant Disorder

-Prevalence is ~20% in kids over 6

Signs & Symptoms
-Comorbid substance abuse, mood disorders, and ADD

Differential
-Conduct disorder: involves violation of basic rights of others while ODD does not

Workup
-DSM-IV criteria: at least 6 months of negativistic, hostile, and defiant behavior during which at least 4+ of the following are present:

- Frequent loss of temper
- Arguments with adults
- Defying adults' rules
- Deliberately annoying people
- Easily annoyed
- Anger and resentment
- Spitefulness
- Blaming others for mistakes or misbehaviors

Management
-Psychotherapy with behavior modification and problem-solving skills
-Parenting skills training

Prognosis
-Remits in 25% of children
-May progress to conduct disorder

Suicide

-In adolescents there are 50-100 attempts for every suicide

Risk factors for ideation
-Psych disorder
-Hopelessness
-Prior suicide attempt or threat
-High impulsivity
-Substance abuse

Workup
-Evaluate intent and lethality of plan

Management
-Ensure immediate safety
-Address precipitating events and life circumstances
-Counseling
-Involvement of support systems

ADHD

Etiology
-Genetic factors
-Prenatal trauma or toxin exposure
-Neurochemical and neurophysiologic factors
-Psychosocial factors

Subtypes
-Predominantly inattentive type
-Predominantly hyperactive-impulsive type
-Combined type

Signs & symptoms
-Peak severity at 7-8 years
-Hyperactivity: excessive fidgetiness, talking, difficulty remaining seated, difficulty playing quietly, frequent restlessness
-Impulsivity: difficulty waiting turns, blurting out answers, disruptive classroom behavior, intruding or interrupting other's activities, peer rejection, unintentional injury
-Inattention: forgetfulness, easily distracted, losing or misplacing things, disorganization, academic underachievement, poor follow-through with assignments or tasks, poor concentration, poor attention to details
-Teacher-reported symptoms should have a duration of at least 4-6 months!
-Comorbid mood disorders, personality disorders, conduct disorder, or ODD

Differential
-Learning disability
-Language or communication disorder
-Autism spectrum disorder
-Anxiety disorder
-Mood disorder
-Oppositional defiant disorder
-Conduct disorder
-OCD
-PTSD
-Adjustment disorder
-Stressful home environment
-Inappropriate educational setting
-Hearing or vision impairment

Workup
-Schools are federally mandated to perform appropriate evaluations at no cost to the family if a child is suspected of having a disability that impairs functioning, but the waiting period can be months
-Primary care toolkit available online via the NICHQ
-Psychometric testing is not necessary for routine evaluation for ADHD and does not distinguish children with ADHD from those without ADHD but can be valuable in excluding other disorders and pinpointing specific ADHD problem areas
-Specialist evaluation indicated for suspected intellectual disability, developmental disorder, learning disability, hearing or vision impairment, history of abuse, severe aggression, seizure disorder, continued dysfunction despite treatment
-Additional evaluations in speech and language, occupational therapy, and mental health as needed
-DSM-IV criteria
1. At least 6 symptoms involving inattentiveness, hyperactivity, or both that have persisted for at least 6 months
    a. Inattention: problems listening, concentrating, paying attention to details, or organizing tasks; easily distracted, often forgetful
    b. Hyperactivity-impulsivity: blurting out, interrupting, fidgeting, leaving seat, talking excessively
2. Onset before age 7
3. Behavior inconsistent with age and development

Diagnostic criteria
-Symptoms must be present and impair function in more than one setting (school, home, work)
-Symptoms must persist for at least 6 months
-Symptoms must present before the age of 7
-Symptoms must be excessive for the developmental level of the child (ie beyond normal hyperactivity for a child's age)

Management
-Methylphenidate (Ritalin) is first-line therapy
-Other CNS stimulants: dextroamphetamine, pemoline
-Individual psychotherapy
-Parental counseling
-Group therapy to improve social skills
-Reevaluation whenever symptoms change or worsen
-Treat comorbid anxiety, depression, and learning disorders

Prognosis
-Most cases remit in adolescence
-20% will have symptoms into adulthood

DERMATOLOGIC SYSTEM

ECZEMATOUS ERUPTIONS

Dermatitis

-Substances are either irritants (= not immunologically mediated) or allergens (type IV hypersensitivity)
-May require sunlight acting on substance to cause the dermatitis
-Inflammation may be acute, subacute, or chronic
-Distinguish irritant from allergic dermatitis by provocation testing: apply substance to AC fossa twice daily for a week; contact urticaria 15-30 min after application suggest allergic etiology
-Patch testing is only indicated when dermatitis is chronic, recurrent, or deters work or life activities (this tests for type IV hypersensitivities rather than type I, which is what skin scratch tests check)
-Does NOT include latex hypersensitivity as this is a type I reaction

Management
-Trigger avoidance
-Topical or systemic steroids
-Emollients or other barriers
-Oral antihistamines

Allergic Contact Dermatitis

-Causes a killer T-cell response
-Common allergens: metallic salts, plants (poison ivy), fragrances, nickel, preservatives, formaldehyde, propylene glycol, oxybenzone, bacitracin, neomycin, bleached rubber, chrome, sorbic acid

Signs & symptoms
-Acute with macules, papules, vesicles, and bullae
-Chronic with lichenification, scaling, fissures
-Uncommon on scalp, palms, soles, or other thick-skinned areas that allergens can't get through

Irritant Contact Dermatitis

-Accounts for most cases of dermatitis
-Common irritants: water, soaps, detergents, wet work, solvents, greases, acids, alkalis, fiberglass, dusts, humidity, chrome, lip licking or other trauma

Signs & symptoms
-Acute with bullae, erythema, and sharp borders
-Chronic with poorly-demarcated erythema, scales, and pruritus
-Fissured, thickened, dry skin
-Usually palmar

Workup
-Negative patch test
-Healing proceeds without plateau on removal of the offending agent

Atopic Dermatitis (Eczema)

-Inflammatory, acute or chronically relapsing, not contagious

Etiology
-Genetic predisposition
-Defects in skin barrier function
-Immune dysregulation

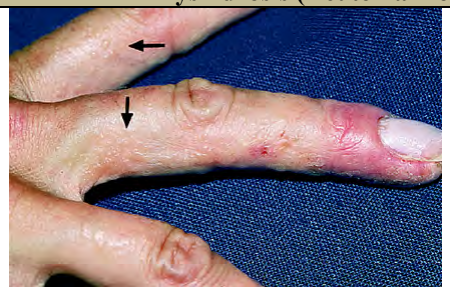
Signs & symptoms
-May have concomitant food allergy, asthma, or allergic rhinitis
-Infantile phase: affects cheeks, forehead, scalp, and extensor surfaces of limbs; lesions are vesicular, edematous, weepy, and crusty
-Childhood phase: affected areas are less vesicular, more papules and plaques that become lichenified
-Post-pubertal phase: skin becomes thickened, dry, and lichenified, may affect dorsal surfaces as well as flexural skin, dyshidrotic changes maybe present on the palms and soles

Workup
-Patch test to look for pustular reactions

Dyshidrosis (Acute Palmoplantar Eczema or Pompholyx)

-Recurrent, pruritic vesicular eruption affecting the palms, soles, or both

Signs & symptoms
-Acute eruption of intensely pruritic vesicles or bullae that persist for weeks, desiccate, and resolve with desquamation
-Episodes recur at intervals of 3-4 weeks for months or years



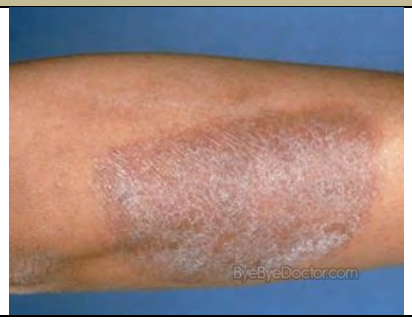
Differential
-Allergic contact dermatitis
-Bullous tinea: usually on the feet, unilateral, + KOH prep
-Irritant contact dermatitis
-Atopic dermatitis
-Herpetic infection: painful
-Autoimmune bullous disease

Workup
-Diagnosis is clinical
-Can consider patch testing to check for component of allergic contact dermatitis

Management
-Avoidance of irritants or exacerbating factors
-Super high potency or high potency topical steroids BID for 2 weeks
-Oral steroids for severe cases (prednisone with taper for 3 weeks)
-Refractory cases: phototherapy, topical tacrolimus

Lichen Simplex Chronicus (Neurodermatitis)

-Thick, leathery, brownish skin due to chronic itching and scratching



Signs & symptoms
-May begin with something that rubs or irritates the skin, or an insect bite
-Cycle of scratching, itching, and thickening skin
-Commonly found on the scalp, nape of neck, extensor forearms and elbows, vulva, scrotum, upper medial thighs, knees, lower legs, or ankles
-Pruritus worse when sitting still or quiet and may be nonexistent when patients are active
-Affected area may spread rapidly throughout rest of body
-May have associated atopic dermatitis or psoriasis
-Comorbid anxiety or depression

Workup
-KOH scraping to exclude fungal cause

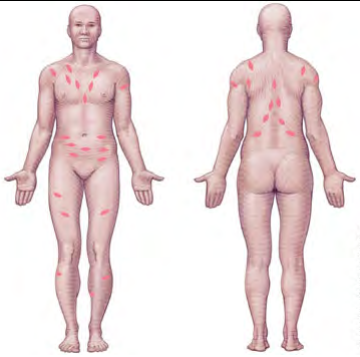
Management
-Steroid cream
-Antihistamines: Benadryl, hydroxyzine
-Anxiolytics: doxepin, clonazepam

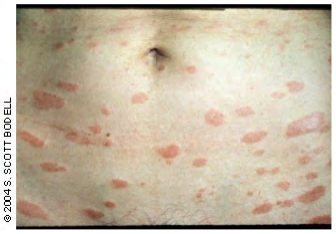
PAPULOSQUAMOUS AND DESQUAMATION DISEASES

Pityriasis Rosea

-A common, benign self-limited dermatosis
-May have viral origins: HSV-7 or 8

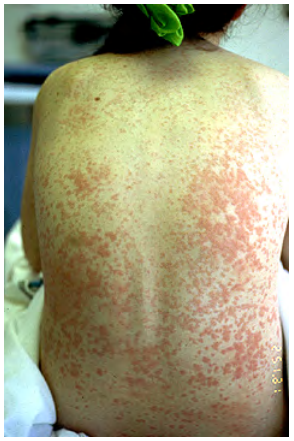
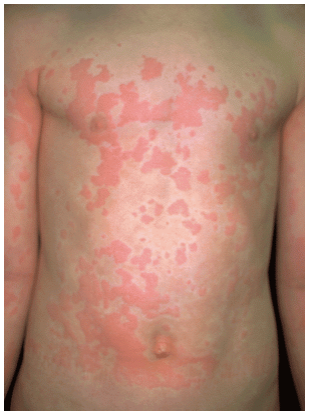
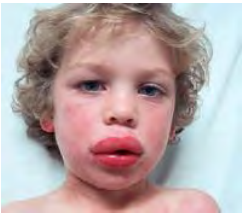
Signs & symptoms
-Typically asymptomatic other than skin presentation
-May have recent h/o infection with fatigue, HA, sore throat, lymphadenitis, fever
-Initially just one herald lesion that is raised with fine scale ("collarette scale")
-7-14 days later there are diffuse eruptions on trunk dermatomes that are salmon in white pts and hyperpigmented in black patients
-Lesions may be pruritic at night or with heat

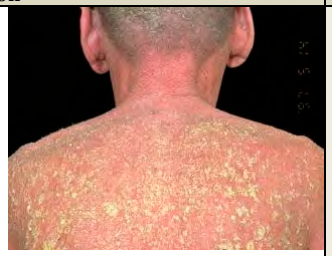
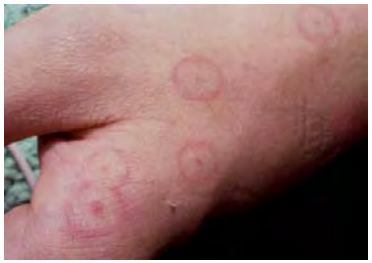
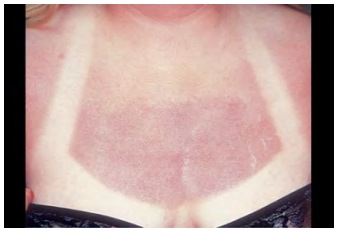
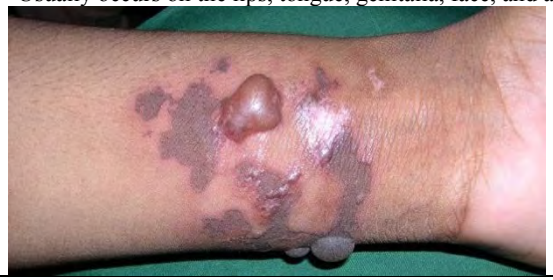
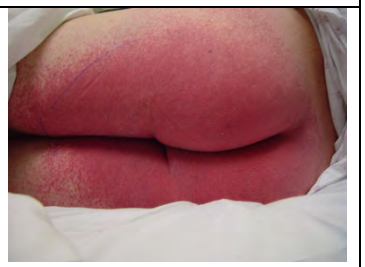




Differential
-Secondary syphilis

Management
-Oral antihistamines and topical steroids
-UVB phototherapy

Classic Drug Eruptions			
Type	**Information**	**Type**	**Information**
Drug-Induced Exanthem	-The most common drug eruption -Thought to be a delayed type IV hypersensitivity reaction -Common offenders are antibiotics, especially sulfa -Described as morbilliform, macular, or papular eruptions -Eruptions begin in dependent areas (= lower than the heart) and then generalize -Onset is usually within 2-3 weeks of beginning new drug or within days if there was prior exposure -May have mucous membrane erythema, pruritus, low grade fever -Can progress to more serious reactions such as S-J, toxic epidermal necrolysis, hypersensitivity syndrome, or serum sickness -D/c of offending drug usually results in resolution of rash in 7-14 days -Drugs causing a morbilliform eruption should only be continued when there is no alternative therapy available 	**Urticaria & Angioedema**	Mechanism can be type I hypersensitivity, or via a non-IgE mechanism → = can be immediate, accelerated (hours later), or delayed (days later) -Urticaria is mediated by mast cells in the superficial dermis -Angioedema is swelling of the deeper dermis and subcutaneous tissues that can be mediated by mast cells (although not always) and can coexist with urticaria -Common offenders causing type I hypersensitivity are antibiotics, especially penicillins, cephalosporins, and sulfonamides -Common offenders causing a non-IgE type hypersensitivity are morphine and codeine ("red man syndrome") -Described as intensely pruritic , circumscribed, raised, and erythematous eruption with central pallor -Lesions may coalesce -Typically disappear over a few hours, although urticaria can manifest in a chronic form (present > 6 weeks) which is usually due to autoimmunity or chronic disease -Can be treated with H1 or H2 blockers, doxepin, glucocorticoids if acute, epinephrine, or around-the-clock antihistamines if chronic  
Anaphylaxis	-A type I hypersensitivity that affects multiple organs, including pruritus, urticaria, angioedema, laryngeal edema, wheezing, nausea, vomiting, tachycardia, sense of impending doom, and occasionally shock 	**Hypersensitivity Vasculitis**	-AKA drug-induced vasculitis, leukocytoclastic vasculitis, serum sickness, serum sickness-like reaction, or allergic vasculitis -Symptoms usually begin 7-10 days after exposure -ACR criteria: age > 16, use of a possible offending drug in temporal relation to the symptoms, palpable purpura, maculopapular rash, skin biopsy showing neutrophils around an arteriole or venule -Other S/S include fever, urticaria, arthralgias, lymphadenopathy, low serum complement levels, and elevated ESR -Common offenders include penicillins, cephalosporins, sulfonamides (including loop and thiazide diuretics), phenytoin, and allopurinol -Resolves after days to weeks, NSAIDS or steroids may be needed for more severe cases

Type	Information	Type	Information
Exfoliative Dermatitis (Erythroderma)	-Begins with generalized eczema or morbilliform erythema that progresses into chronic erythema and scale involving > 50% of the body surface area -Can be caused by drugs as well as atopic dermatitis, malignancy, or psoriasis -Common offenders include penicillins, barbiturates, gold salts, arsenic, and mercury 	**Stevens-Johnsons Syndrome & Toxic Epidermal Necrolysis**	-Severe mucocutaneous eruptions characterized by epidermal necrosis and sloughing of mucous membranes and skin -Whether it is S-J or TEN depends on % of body surface involved, if > 10% it is S-J, if > 30% it is TEN 
Erythema Multiforme	-A different condition than S-J -An acute eruption with distinctive target skin lesions that tend to affect the distal extremities including the palms and soles -Often caused by infections such as HSV or Mycoplasma pneumoniae, but it has had some reports with medication use 	**Photosensitivity**	-Phototoxic variety: result of direct tissue or cellular damage following UV irradiation of a phototoxic agent that has been ingested or applied to the skin (tetracyclines, thiazides, sulfonamides, fluoroquinolones, NSAIDs, phenothiazines, griseofulvin, voriconazole, retinoids, St. John's wort)  -Photoallergenic variety: a delayed-type hypersensitivity reaction to an allergen whose antigenicity has changed after UV exposure (sunscreens, antimicrobials, NSAIDs, fragrances, griseofulvin, quinolones, sulfonamides, ketoprofen, piroxicam)
Fixed Drug Eruption	-A distinctive reaction characterized acutely by erythematous and edematous plaques with a grayish center or bullae, dark postinflammatory pigmentation -Usually occurs on the lips, tongue, genitalia, face, and acral areas  -Common offenders are laxatives, tetracyclines, barbiturates, sulfonamides, NSAIDs, and salicylates	**Symmetrical Drug-Related Intertriginous and Flexural Exanthem**	-AKA baboon syndrome -Rare -Occurs hours to days after administration of offending drug -Sharply demarcated V-shaped erythema in the gluteal, perianal, inguinal, or perigenital areas, often with involvement of at least one other flexural or intertriginous fold  -Common offenders are amoxicillin, ceftriaxone, penicillin, clindamycin, erythromycin, iodinate contrast, pseudoephedrine, valacyclovir -Treat with topical or systemic steroids

Hypersensitivity Syndrome	-AKA drug reaction with eosinophilia and systemic symptoms (DRESS) or drug-induced hypersensitivity syndrome (DIHS) -Infection with HHV-6 may play a role -Reaction typically occurs 2-6 weeks after drug is first used -Usually begins with morbilliform or erythrodermic eruption with possible erythematous follicular papules, pustules, bullae, or purpura, as well as fever, hepatitis, arthralgias, lymphadenopathy, hematologic abnormalities, pneumonitis, renal failure, myocarditis, thyroiditis, and neurologic symptoms -Severity is dependent on length of time drug is continued -Can be life-threatening no some cases -Common offenders include phenytoin, carbamazepine, phenobarbital, sulfonamides, lamotrigine, valproate, allopurinol, minocycline, antidepressants, NSAIDs, ACEIs, beta blockers	CLINICAL FEATURES OF ANTICONVULSANT HYPERSENSITIVITY SYNDROME [4] FINDING INCEDENCE % Fever 90 Skin Rash 90 Lymphadenopathy 70 Hepatitis 50 Hematologic Abnormalities (eosinophilia, atypical lymphocytosis) 50 Facial Edema 25

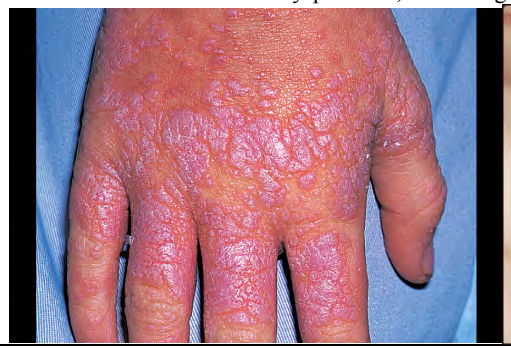
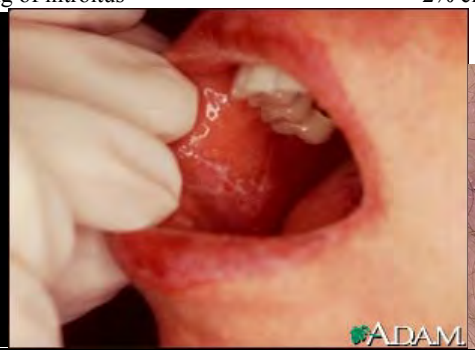
Lichen Planus

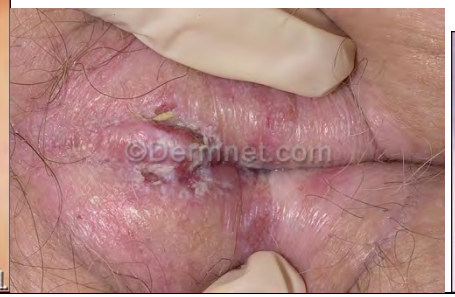
-Chronic mucocutaneous disease of uncertain etiology
-May involve CD8 activation against keratinocytes
-May be related to hepatitis C
-Can be drug-induced: ACEI, thiazide, antimalarial, β-blocker
-Mostly affects middle-aged adults

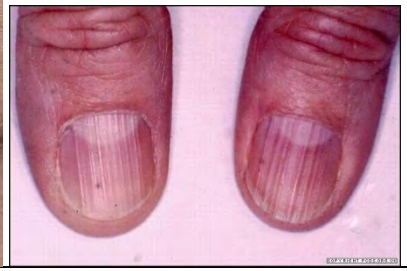
Signs & symptoms
-Can affect skin, nails, mucous membranes
-Lichen planus of the scalp (lichen planopilaris) can cause alopecia
-Cutaneous presentation: pruritic violaceous papules or plaques with overlying white or lacey pattern
-May also have hypertrophic or vesicobullous lesions
-Oral presentation: papular, atrophic, or erosive lesions
-Vulvar presentation: vaginal discharge, pruritus, burning, dyspareunia, narrowing of introitus

Management
-Topical steroids
-May need intralesional steroid injections for scalp or hypertrophic forms
-Systemic steroids for systemic disease
-Phototherapy
-Refractory disease: tetracycline, hydroxychloroquine, or mycophenolate mofetil
-Vulvar lichen planus: DOC is hydrocortisone suppositories with 2% clotrimazole for candidiasis prophylaxis

Prognosis
-Disease may remit in 1-2 years or may be chronic
-Oral lichen planus is risk factor for SCC





Psoriasis

-A hyperproliferation of the epidermis with altered differentiation → inflammation of the epidermis and dermis with accumulation of T-cells and cytokines
-Can be flared by strep infections, injury, trauma, drugs, low humidity, emotional stress, and overtreatment

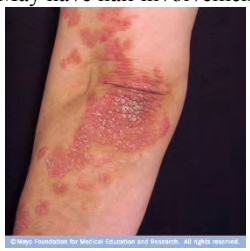
Signs & symptoms
-Red scaling papules that coalesce into round-oval plaques with a silvery white adherent scale
-Pustules may border lesions
-Lesions are most commonly on the scalp, elbows, legs, knees, arms, trunk, lower body, palms, and soles, and occur at sites of trauma
-Variable pruritus
-Extracutaneous manifestations: onycholysis, geographic tongue, destructive polyarthritis, ankylosing spondylitis, DIP arthritis, CV disease, depression, lymphoma

Differential
-Atopic dermatitis (eczema)
-Contact dermatitis
-Nummular eczema
-Tinea
-Candidiasis
-Intertrigo
-Seborrheic dermatitis
-Pityriasis rosea
-Secondary syphilis
-Onychomycosis
-Cutaneous features of reactive arthritis
-Cutaneous T-cell lymphoma
-Lichen simplex chronocus

Management
-Initial treatment with topical corticosteroids and emollients for mild to moderate plaque psoriasis
-Alternatives: tar, topical retinoids, topical vitamin D, topical tacrolimus or pimecrolimus
-Localized phototherapy
-Systemic therapy for refractory cases: methotrexate, cyclosporine, biologics
-Treating coexisting depression can also help the psoriasis

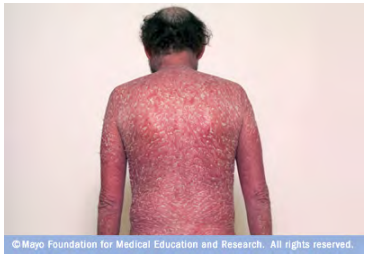
Chronic Plaque Psoriasis

-Sharply defined erythematous scaling plaques in symmetric distribution
-The most common type of psoriasis
-Lasts months to years
-May have nail involvement



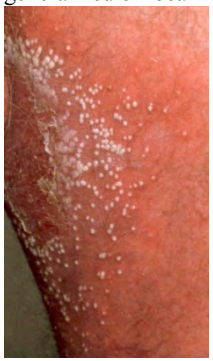
Erythrodermic Psoriasis

-Generalized erythema with scaling and exfoliation
-Accounts for 10% of cases
-Patients are very sick, with hypo or hyperthermia, protein loss, dehydration, renal and cardiac failure
-May need a punch biopsy to differentiate from contact dermatitis



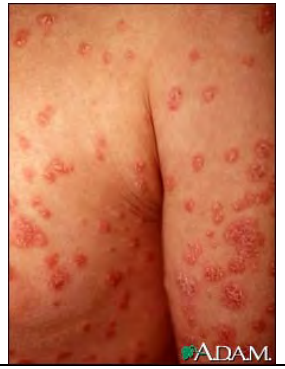
Pustular Psoriasis

-Individual or coalescing non-infectious pustules 1-10 mm that are generalized or localized



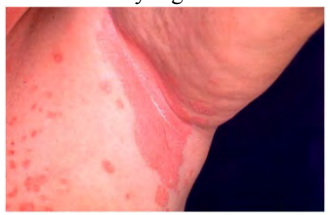
Guttate Psoriasis

-Multiple small papules of short duration
-Associated with recent strep infection
-Can also see typical plaque lesions on the knees and elbows



Intertriginous (Inverse) Psoriasis

-Presentation involves the inguinal, perineal, genital, intergluteal, axillary, and inframammary regions

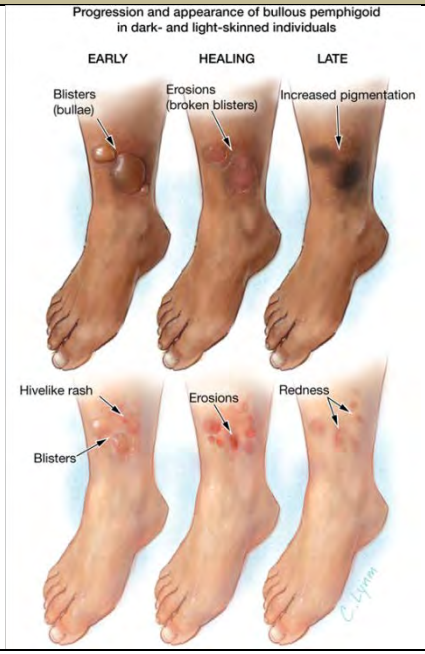


VESICULAR BULLAE

Bullous Pemphigoid

-Uncommon autoimmune subepithelial blistering disease
-On the same spectrum as linear IgA disease, pemphigus, bullous lupus erythematosus, dermatitis herpetiformis, and epidermolysis bullosa acquisita
-May be a result of genetic factors as well as environmental exposures
-Most common in older adults

Signs & symptoms
-Prodromal phase with pruritic inflammatory plaques resembling eczema or urticaria
-Followed by development of multiple tense 1-3 cm bullae with erosions and crusts
-Heal without scarring



Differential
-Broad!
-Aphthous stomatitis
-Contact dermatitis
-Contact dermatitis or eczema
-Dermatitis herpetiformis
-Drug reaction
-Lichen planus
-Stevens-Johnson or TEN
-Viral infection
-Many more

Workup
-Lesion biopsy needed

Management
-High potency topical steroid
-Systemic steroid
-May need long-term therapy with mycophenolate mofetil, azathioprine, or methotrexate as this disease often persists for years

Prognosis
-Remission may occur after months or years
-May be fatal secondary to infection or use of immunosuppressive agents

ACNEIFORM LESIONS

Acne Vulgaris

Etiology
-Multifactorial, involving hormones, keratin, sebum, and bacteria
-Proliferation of P. acnes in this environment → foreign body reaction

Types
-Comedonal
-Inflammatory
-Cystic: characterized by cysts, fissures, abscess formation, deep scarring

Differential
-Hydradenitis suppurativa (acne inversa): usually occurs in the axillae, inguinal folds, and perianal area; hallmark is double comedones
-Steroid acne
-Meds: Li, tetracyclines (paradox), phenytoin, OCPs, isoniazid
-Infectious folliculitis
-Cutting oils and other occlusives
-Rosacea
-Perioral dermatitis

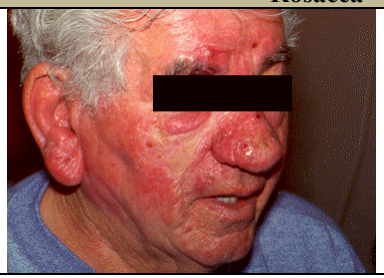
Management
1.) Behavioral modification: no picking, mild cleanser BID, oil-free non-comedogenic products
2.) Topical comedolytics (allow 4-6 weeks to work): retinoid (pregnancy D), azelaic acid (better for pregnancy), glycolic acid, salicylic acid
3.) Topical antibacterials: benzoyl peroxide (DOC, no bacterial resistance), clindamycin, erythromycin (lots of resistance)), sulfur-containing preparations, metronidazole, dapsone (for inflammatory acne)
4.) Oral therapies: antibiotics (minocycline, doxycycline, tetracycline, erythromycin; need 2-4 weeks to work), 5 mo course of isotretinoin (regulated by FDA iPledge due to pregnancy X), OCPS (for adult acne, hirsutism, PCOS, premenstrual flares), spironolactone (for poor OCP candidates, pregnancy X)

Prognosis
-Usually ends by age 25

Rosacea

Etiology not well understood: spicy food, alcohol, exercise, sun?

Signs & symptoms
-Resembles acne but also has flushing, telangiectasia, lingering erythema on forehead, chin, ± eyes
-No comedones
-Late manifestation is rhinophyma (big bulb-shaped nose)



Treatment
-Topical metronidazole, sulfacetamide + sulfur, or azelaic acid
-Time-released doxycycline
-Laser therapy

VERRUCOUS LESIONS

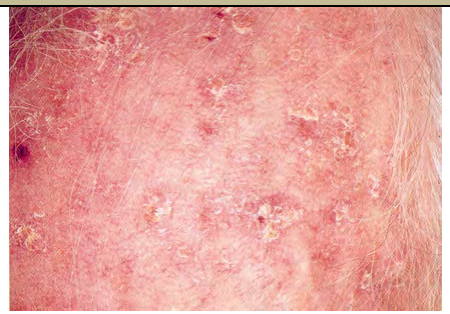
Actinic Keratosis

-Common cutaneous lesions as a result of proliferation of atypical epidermal keratinocytes

Risk factors
-Chronic sun exposure
-Fair skin
-Advancing age
-Male sex

Signs & symptoms
-Scaly, erythematous macules or papules on sites of chronic skin exposure
-May be pigmented, nonerythematous, or free of scale
-Common sites are scalp, face, lateral neck, dorsal forearms, dorsal hands
-Actinic cheilitis is a variant involving the lip

Differential
-Lentigo maligna
-SCC



Workup
-Biopsy uncertain lesions, esp if > 1 cm, indurated, ulcerated, rapidly growing, or unresponsive to therapy

Management
-Liquid nitrogen cryotherapy for isolated lesions
-Surgical curettage or shave removal
-Multiple lesions in a given area: 5-FU, imiquimod, or photodynamic therapy
-Avoid sun exposure and monitor lesion recurrence or malignancy transformation

Prognosis
-May progress to SCC

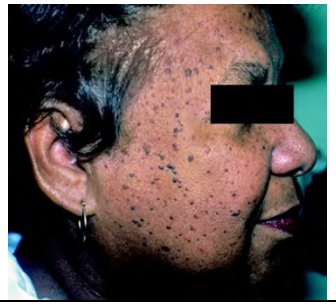
Seborrheic Keratosis

-Common epidermal tumor from benign proliferation of immature keratinocytes

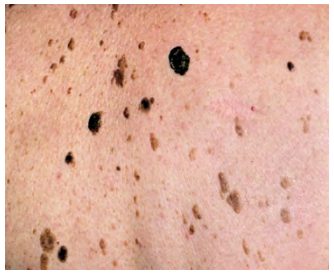
Sign of Leser-Trelat = sudden appearance of multiple seborrheic keratoses in association with skin tags and acanthosis nigricans; a sign of GI or lung cancer

Dermatosis papulosa nigra = variant commonly seen on the face of black patients, may be pedunculated

Risk factors
-FH
-Inflammatory skin disease



Signs & symptoms
-May be 1 isolated lesion or hundreds
-Stuck-on, warty, well-circumscribed, often scaly hyperpigmented lesions
-Most commonly on the trunk, face, and upper extremities



Differential
-Nevus: will not have stuck on or warty appearance, no scale
-Melanoma: look for blurred borders, asymmetry, or h/o change
-Pigmented BCC: h/o change, waxy appearance, dilated blood vessels, ulceration

Management
-Liquid nitrogen therapy ± curettage
-Shave excision with 1% lidocaine
-Electric removal

INSECT & PARASITIC INFESTATIONS

Lice

Signs & symptoms
-Scalp or neck pruritus
-May be asymptomatic
-Cervical and nuchal lymphadenopathy
-Secondary infection

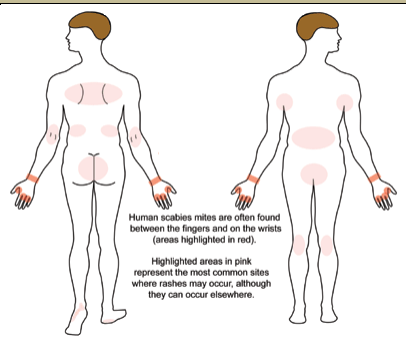
Workup
-Diagnosis is with visualization of the nits on hair shaft as well as crawling nymphs and adults

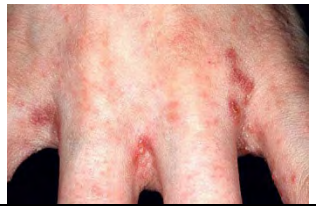
Management
-Resistance has been reported and varies geographically
-First-line treatments are pyrethroids, malathion, benzyl alcohol, or spinosad
-Mechanical wet combing is an alternative therapy for kids too young for medical therapy (< 2 months)
-Lindane is restricted due to neurologic effects
-Treat bedmates prophylactically and examine all housemates and close contacts for nits
-Wash all clothing and bedding used in last 2 days in hot water
-Store non-washable items in a plastic bag for 2 weeks
-2nd treatment with insecticide 7-10 days after first treatment to kill any surviving nits
-Kids may return to school after first application of insecticide

Scabies

-Caused by mites
-Spread by skin to skin or sex, with incubation period of 21 days
-Can live for 48 hours on clothing, bedding, and furniture

Signs & symptoms
-Intensely pruritic papules and pustular rash that is worse at bedtime
-Predilection for finger webs, wrist flexors, elbows, axillae, penis, external genitalia, feet, ankles
-Babies < 1 year can get scabies from the neck up
-"Norwegian" scabies make severe crusting and have heavy infestation





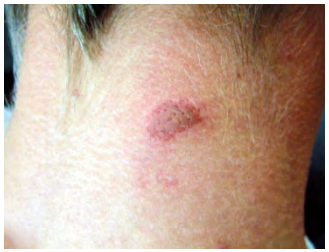
Workup
-Skin scraping under oil immersion for mites, feces, and eggs

Treatment
-Permethrin cream, usually only single application needed
-Treat all family members
-Wash all bedding and clothing in hot water

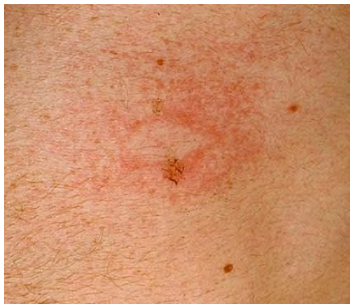
Spider Bites

-Most lesions attributed to spider bites are caused by something else

Signs & symptoms
-Solitary papule, pustule, or wheal
-Systemic symptoms of evenomation with certain spiders
-Recluse bite: malaise, n/v, fever, myalgias, progression of lesion to necrosis



-Widow spider: muscle pain or spasm, local paresthesia, HA, n/v, no necrosis



Differential
-Infection
-Other bite or sting: triatomid, ant, flea, bedbug, blister beetle, tick, mite, mosquito, biting fly
-Scorpion sting
-Common dermatosis: poison ivy, poison oak

Management
-Widow bite: depends on severity of evenomation; pain control, wound cleansing, tetanus prophylaxis, antiemetics, benzos, widow antivenom
-Recluse bite: local wound care, pain meds, tetanus prophylaxis, antibiotics for cellulitis, dapsone for necrosis

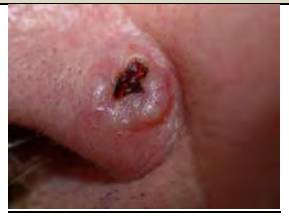
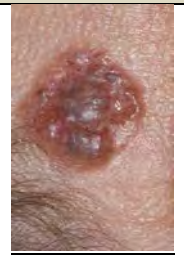
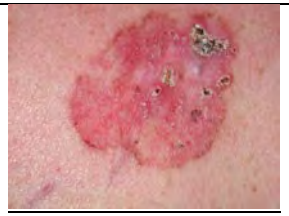
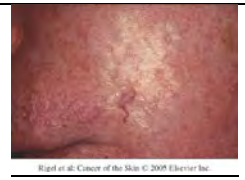
Medically relevant spiders by geographic location

Geographical location	Common name and species	Appearance	Typical habitat
Widows			
Southeastern United States (Maryland, Southern Ohio and lower states) Western half of the United States from Canada to Mexico	Southern black widow *L. mactans* Western black widow *L. hesperus*	Shiny black spider with some form of red on body	Clutter surrounding homes (eg, gardens, sheds, garages) Rarely indoors
New Zealand (coastal areas) Australia (coastal areas) Japan (Osaka prefecture)	Australian red back *L. hasselti*	Shiny black body with dorsal red stripe	
South America	*L. curacaviensis*		
Mediterranean	Black hag, black wolf *L. tredecimguttatus*	Smattering of 13 red dots on dorsal abdomen, **no** red hour glass	
World wide and in United States (from South Carolina to Texas and California)	Brown widow (can bite humans, but envenomation is usually mild) *L. geometricus*	White stripes on a tan abdomen with orange fangulars, abdomen color can vary from cream to almost black	
False Black Widows			
United States: Pacific coast and Colorado Canada: British Columbia Australia Europe	False black widow *S. grossa* *S. pavulliana* *S. grossa*	Similar shape to widows Chocolate brown color with tan stripes or markings on abdomen **do not** have red markings	Clutter surrounding homes Also indoors (in cupboards and undisturbed places)
Recluses			
United States: Mid-west and Southern states extending westward (see map in text) Worldwide infestations of buildings South America (Brazil, Chile, others) Isolated reports in South Africa, Australia	Brown recluse *L. reclusa* Mediterranean recluse *L. rufescens* Chilean recluse *L. laeta* *L. intermedia* *L. gaucho*	Non-descript brown spiders Recluses have 3 pairs of eyes (6 total), monochromatic abdomen and legs, and very fine hairs on legs	Mostly inside homes: attics, basements, cupboards Outdoors: in rock piles and under tree bark, **not** in live vegetation
Phoneutria			
South America	Brazilian wandering spider *P. nigriventer* *P. keyserlingi* *P. fera*	Large, extensively haired spider (up to 95 mm legspan)	Forage at night and may enter homes to take refuge under household items during the day Found in urban environments in piles of clutter, vegetation, or rubbish.
Australian Funnel Web			
Australia - Southeastern coastal regions (including Sydney and Brisbane)	Australian funnel web spider *Atrax robustus* 5 species of *Hadronyche*	Large spider (25 mm body length), shiny black coloration on body	Moist areas such as basements

NEOPLASMS

Basal Cell Carcinoma

-Most common skin cancer, and most common human cancer
-Slow growing, locally destructive
-No mets
-Risk factors: sun, sunburns < age 14, arsenic ingestion, radiation
-More common in males
-Usually after age 40
-Several subtypes with different treatments

Type of BCC	Info	Investigation & Treatment	Picture	Type of BCC	Info	Investigation & Treatment	Picture
Nodular BCC	Most common BCC. Pearly white or pink dome shaped papule with overlying telangiectasias → ulceration, raised borders, bleeding, scaling.	ED&C, excision, Mohs.		**Pigmented BCC**	Resembles melanoma.	ED&C, excision, Mohs.	
Superficial BCC	Least aggressive BCC. Erythematous scaly plaques or papules +/- rolled borders. Can look like psoriasis, eczema, others.	ED&C, excision.		**Morpheaform BCC**	Least common variant. White to yellow patch with poorly-defined borders.	Mohs needed.	

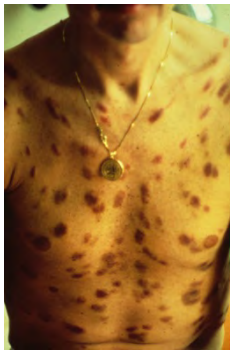
Kaposi Sarcoma

-Neoplasm characterized by abnormal angiogenesis
-Requires prior infection with HHV-8
-Most common in elderly men of Mediterranean and European descent

Epidemiologic and clinical types of Kaposi sarcoma

Type	Predominant risk groups	Cutaneous presentation	Visceral involvement	Clinical course
Classic (sporadic)	~3:1 male:female ratio Age >60 Mediterranean or Central/Eastern European Origin; Middle East	Distal lower extremities	Uncommon	Usually indolent; rarely aggressive & disseminated
Endemic (African)	Male adults Children of both sexes Equatorial Africa	Various (may be similar to classic or more locally aggressive); lower extremity lymphedema in adults; cutaneous erosive often absent in children	Internal organs involved in a subset of adult patients Common (lymph nodes and viscera) in children	Indolent to locally invasive in adults Occasional rapid progression with visceral disease in adults Aggressive in children
Iatrogenic (immunosuppression-related)	Exogenous immunosuppression, esp. solid organ transplant Older patients (>50) Use of cyclosporin A	Distal lower extremities; may be disseminated	Relatively common	May regress with modification of immunosuppression May be aggressive
AIDS-associated	Men who have sex with men (developed countries) Heterosexual men & women (Africa)	Localized or disseminated	Common with poor HIV control	Aggressive or indolent May regress with effective HIV treatment

Signs & symptoms
-Purplish, reddish blue, or dark brown or black lesions on the lower extremities, often with lymphedema
-Slow growing and localized, but can become disseminated
-Visceral involvement with HIV



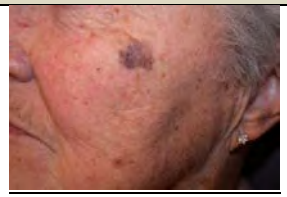
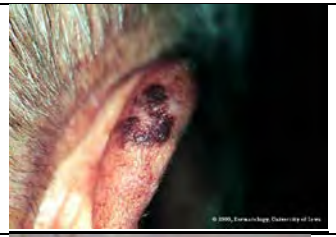
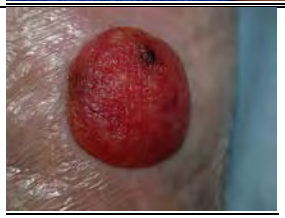
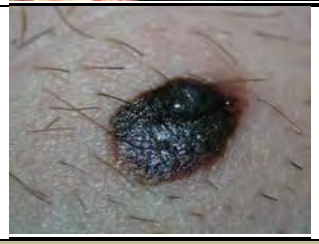
Workup
-Biopsy required

Management
-Observation for limited asymptomatic lesions that do not impair function
-Compression stockings for LE edema
-Local treatment: radiation therapy, excision, cryotherapy, laser ablation
-Chemo: PLD

Melanoma

-Flat, raised, nodular, or ulcerated
-Variable color
-Consider in any new mole or a mole changing shape, size, or color

Workup
-Lymph node palpation
Punch or incisional biopsy

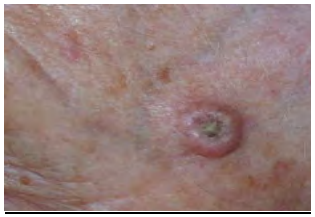
Type of Melanoma	Info	Picture	Type of Melanoma	Info	Picture
Lentigo maligna (melanoma in situ)	Melanoma restricted to epidermis.		**Acral lentiginous melanoma**	Primarily on hands, feet, nails. Most common type of melanoma in blacks and Asians. Common in males.	
Superficial spreading melanoma	Most common type of melanoma. Asymmetric, flat lesions > 6 mm. Vary in color. Lateral spread.		**Amelanotic melanoma**	Innocent-appearing pink to red colored papules that enlarge to plaques and nodules. Scary.	
Nodular melanoma	Rapid growth vertically from and through skin. Most common on extremities.				

Squamous Cell Carcinoma

-AKA Bowen's disease if SCC in situ
-Potentially invasive malignancy of keratinocytes in the skin or mucous membranes
-Most caused by UV radiation but other risks are chemicals, tobacco, infection, burns, HPV
-Erythroplasia of Queyrat is SCC of the penis

Presentation
-Flesh, pink, yellow, or red indurated papules plaques, or nodules with scale
-Can have ulcerations and erosions

Workup & Management
-Palpate regional lymph nodes for mets.
-ED&C, excision, Mohs.
-Bowen's: 5-FU, cryo, ED&C, excision, Mohs.



HAIR & NAILS

Alopecia

Signs & symptoms
-Alopecia areata: smooth, circular, discrete areas of complete hair loss developing over a few weeks, may have nail pitting
-Male pattern baldness: slow, progressive transition of terminal hairs on the frontal scalp to shorter, thinner, more vellus hairs

Differential
-Scarring (cicatricial) alopecia: chemical or physical trauma, lichen planopilaris, bacterial or fungal infection, shingles, discoid lupus, scleroderma, ionizing radiation
-Nonscarring: male pattern baldness, telogen effluvium (stress-related), alopecia areata (immune-related, exclamation point hairs), trichotillomania, drug-induced, SLE, secondary syphilis, hyperthyroid, hypothyroid, iron-deficiency anemia, pituitary deficiency

Workup
-May need microscopic examination of hair or scalp biopsy

Management
-Alopecia areata → topical steroids
-Male pattern baldness → minoxidil or finasteride, hair transplant, antiandrogen in women (spironolactone)

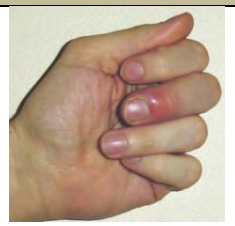
Paronychia

-Infection of nail base

Agents
-Acute: *Staph*
-Chronic: *Candida*

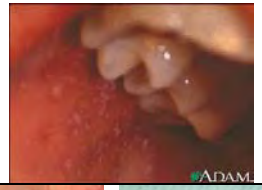
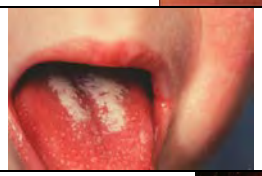
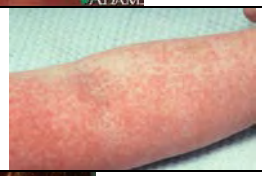
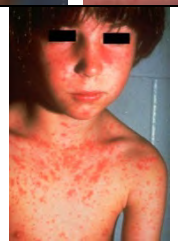
Signs & Symptoms
-Pain
-Erythema
-Abscess formation
-Swelling

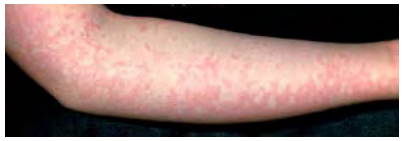
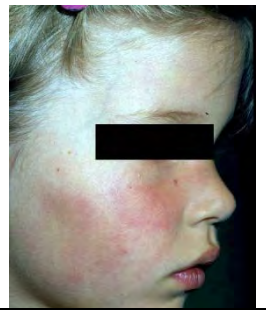
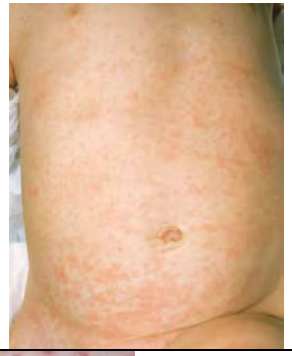
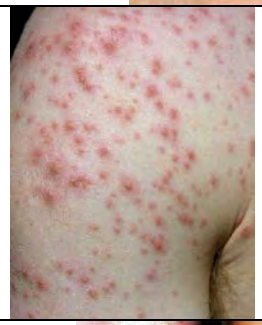
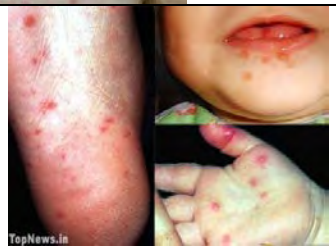
Management
-I&D for abscess
-Raise nail to express pus
-Warm moist compresses for 24 hours followed by dry dressings for 3-4 days

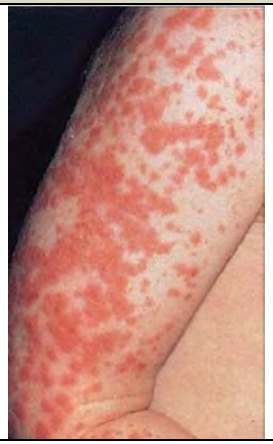


VIRAL DISEASES

Childhood Exanthems

Disease	Information	Signs & symptoms	Picture	Treatment
Measles (Rubeola or First Disease)	-Agent is measles virus -Prevent with MMR vaccination (indicated to prevent death)	-Cough -Coryza -Conjunctivitis -Koplik's spots -Maculopapular rash starting at hairline and spreading down to confluence		-Self-resolution in 7-10 days -Supportive care -Complication: subacute sclerosing panencephalitis, a rare fatal infection years after initial infection
Scarlet Fever (Second Disease)	-Agent is GAS	-Pharyngitis -Strawberry tongue -Sandpapery rash that is worse in the groin and axilla with desquamation of palms and soles	 	-Penicillin VK or amoxicillin administered to prevent sequelae of rheumatic fever
Rubella (German Measles, 3 Day Measles, Third Disease)	-Agent is rubella virus -Prevent with vaccination (indicated to prevent congenital rubella syndrome)	-Mild fever -Conjunctivitis -Arthralgias -Postauricular and occipital adenopathy -Maculopapular rash on face that spreads		-Resolves in 3 days -Sequelae of arthralgias

Disease	Information	Signs & symptoms	Picture	Treatment
Erythema Infectiosum (Fifth Disease)	-Agent is human parvovirus B19	-Mild flulike illness -Rash at days 10-17: initially appears as flushed cheeks, then encompasses whole body as a maculopapular rash, becoming lacy in the arms and legs -Low grade fever -Migratory arthritis in older patients that can last 6-8 weeks -"Papular purpuric glove & sock syndrome" in older adolescents, lasts 1-2 weeks	 	-Treatment is supportive with NSAIDs for arthralgias and fever
Roseola (Sixth Disease)	-Agent is HHV-6 or HHV-7 -Affects young children, 6 mo to 3 years	-High fevers to 104 for 3-7 days with no rash -Rash after fever goes away		-Antipyretics and hydration
Varicella	-Caused by HHV-3 (VZV) -Child will be contagious for 1 week	-Intensely pruritic lesions on the trunk first, then face, head, extremities, possibly mucous membranes -Lesions come in crops over 3-4 days and crust over in 3-5 days		-Symptomatic only -Consider acyclovir in teens
Hand-Foot-Mouth Disease (Herpangina)	-Agent: Coxsackie A16 virus -Highly contagious	-Vesicles on tongue, oral mucosa, hands, possibly feet -May have generalized scarlatiniform rash -Low-grade fever -Overall child feels well		-Symptomatic -Sequelae of myocarditis, substernal chest pain, dyspnea

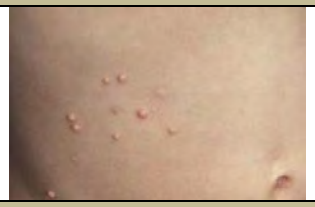
Disease	Information	Signs & symptoms	Picture	Treatment
Gianotti-Crosti Syndrome (Papular Acrodermatitis of Childhood)	-Usually caused by EBV, Hep B, or HHV-4 -Affects 6-14 month olds	-Symmetric red-purple papules and papulovesicles on the face, buttocks, and extremities -Lymphadenopathy -Low-grade fever		-Self-limiting over 3-4 weeks
Enterovirus Exanthems		-Varied rash; may be maculopapular, vesicular, petechial, or urticarial -May involve other organ systems		

Molluscum Contagiosum

-Agent is a poxvirus
-Spread by autoinoculation

Signs & symptoms
-Flesh-colored dome-shaped lesions with umbilicated central core

Management
-Self-limiting after about 1 year
-Cryotherapy



Verrucae

-Agent is HPV, which infects the skin keratinocytes to cause warts
-Occur in areas of skin trauma
-100 serotypes = many infections possible over a lifetime
-Don't have "roots", rather they are confined to the epidermis
-Cause necrosis of capillaries
-Have oncogenic potential

Subtypes
-Verruca vulgaris: the common wart; verrucous surface, thrombosed capillaries, loss of dermatoglyphics, may have fingerlike projections
-Verruca plana: the flat wart; flat-topped pink to brown papules, usually in linear formation, with predilection for the face, dorsal hands, wrists, and knees, commonly spread by shaving
-Verruca plantaris: the plantar wart; verrucous surface, thrombosed capillaries, often coalesce into a "mosaic", with predilection for pressure points of the feet

Differential
-Callus: won't interrupt skin lines

Management
-Wait for regression via cell-mediated immunity
-Destruction: cryotherapy, laser, cautery, duct tape occlusion, excision, beetle juice, podophyllin gel, retinoids, salicylic acid, 5-fluorouracil
-Immunomodulating agents

Condyloma Acuminatum

-Anogenital warts associated with HPV subtypes 6 and 11
-The most common STD in the US

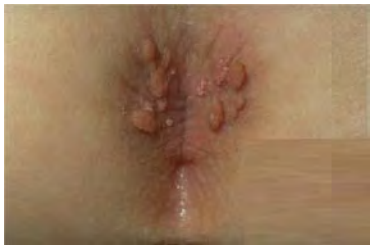
Transmission
-Sex, including digital/anal, oral/anal, and digital/vaginal contact
-Possibly fomites

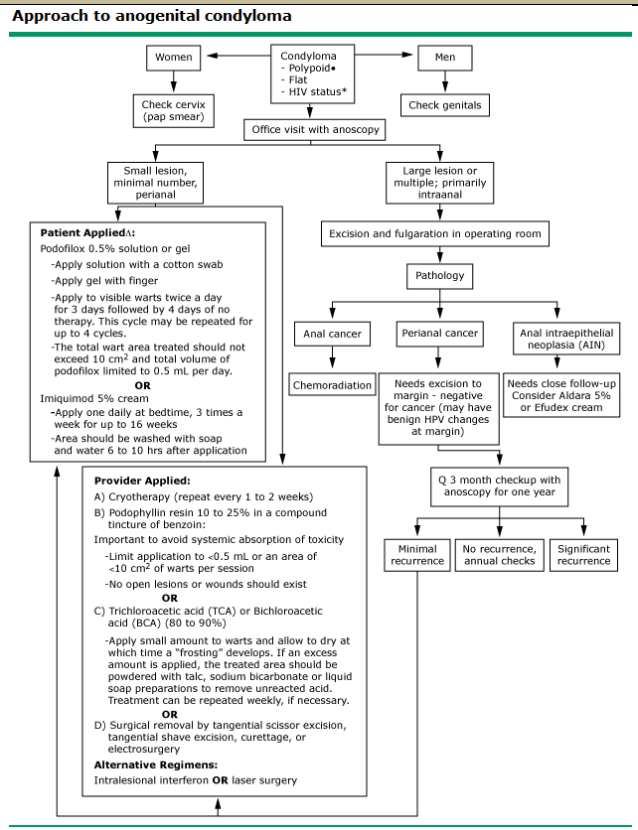
Prevention
-Gardasil vaccine

Signs & symptoms
-Many are asymptomatic
-Smooth papules or verrucous lesions
-Pruritus
-Bleeding
-Burning
-Tenderness
-Vaginal discharge

Differential
-Condyloma lata of 2° syphilis
-Micropapillomatosis of the vulva
-SCC
-Vulvar intraepithelial neoplasia
-Molluscum contagiosum
-Skin tag

Management
-Chemical or physical destruction
-Immunologic therapy
-Surgical excision
-For recurrence → surgical excision or fulguration therapy followed by adjuvant treatment





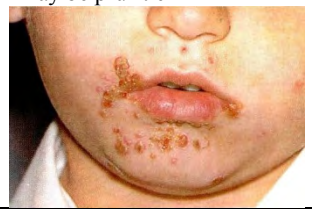
BACTERIAL INFECTIONS

Impetigo

Etiologies
-Usually *Staph aureus*
-Also *Strep*
-If deep and extending into the dermis with ulceration and a tender yellow-gray crust, it is called ecthyma (agents are *Strep*, *Pseudomonas*, *Staph*)

-Risk factors: trauma, underlying eczema or HSV, poor hygiene, previous antibiotics, warm temps, high humidity
-Lesions are spread by auto-inoculation

Signs & symptoms
-Oozing lesions
-May be pruritic



Management
-Topical mupirocin for small number of non-bullous lesions
-Oral therapy for anything else: dicloxacillin, cephalexin, or clindamycin
-Suspect MRSA → clindamycin or linezolid

Sequelae
-May be followed by poststreptococcal glomerulonephritis or rheumatic fever

Cellulitis		
-Risk factors: skin trauma, inflammation (eczema or radiation), pre-existing impetigo or tinea pedis, edema **Agents** -Most commonly GAS or GBS -*Staph aureus* -Gram negs -*H. flu* -*Clostridium* -*Pseudomonas* **Differential** -Erysipelas: more acute onset that involves the upper dermis and superficial lymphatics while cellulitis involves the deeper dermis and subcutaneous fat; lesions will be raised above surrounding skin with clear demarcation between involved and uninvolved tissue -If it involves the ear, it's erysipelas! (no deep tissue here)	**Signs & symptoms** -Skin erythema, edema, and warmth -Indolent onset with development of symptoms over days -May have purulent drainage or exudate **Differential** -Necrotizing fasciitis -Gas gangrene -TSS -Bursitis -Osteomyelitis -Herpes zoster -Erythema migrans **Workup** -Diagnosis is clinical -Cultures if there is systemic toxicity, extensive skin involvement, underlying comorbidities, unusual exposures like animal bites or water-associated injury, or recurrent or persistent cellulitis	**Management** -Can usually be treated with oral antibiotics -Careful, erysipelas must be covered with IV therapy for initial management -IV AB for systemic toxicity or rapid progression -Empiric therapy for nonpurulent cellulitis to cover *Strep* and MSSA → oral dicloxacillin (or cephalexin or clindamycin), IV cefazolin (or oxacillin, nafcillin, or clindamycin) -If risk factors for MRSA (recent hosp, LTC resident, recent AB, HIV, MSM, IVDU, HD, incarceration, military, DM) with nonpurulent cellulitis →oral clindamycin (or amoxicillin + Septa or doxy, or linezolid) -Purulent cellulitis (= risk for MRSA) → clindamycin, Septra, doxy, or linezolid -May need suppressive therapy for recurrent cellulitis

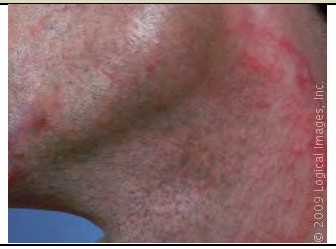
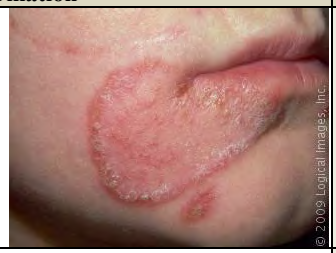
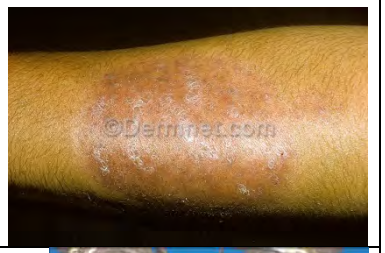
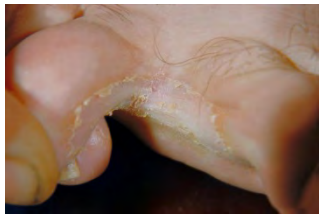
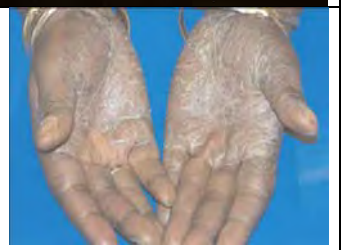
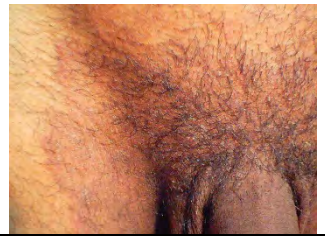
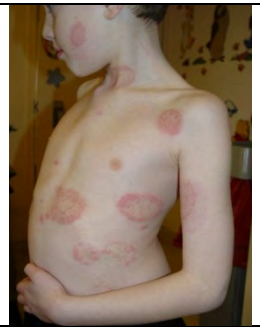
FUNGAL INFECTIONS

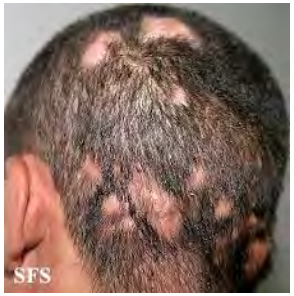
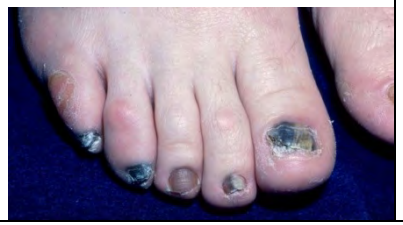
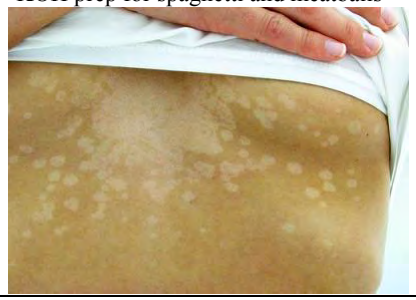
Dermatophytoses

-Caused by *Microsporum, Trichophyton*, or *Epidermophyton* spp
-May be transmitted person-to-person, soil-to-person, or zoonotically
-Risk factors: atopy, immunosuppression, existing skin condition, DM, sweating, humidity
-AKA "tinea" or "ringworm"

Differential

-Atopic dermatitis
-Dyshidrotic dermatitis
-Lichen simplex chronicus
-Psoriasis
-Trichotillomania
-Traction alopecia
-Alopecia areata
-Erythema chronicum migrans
-Pityriasis versicolor
-Pseudofolliculitis barbae
-Seborrheic dermatitis
-Acne rosacea
-Discoid lupus erythematosus
-Contact dermatitis
-Candidal intertrigo
-Erythrasma
-Friction blister
-Onychogryphosis

Disease	Information	Management	Information	Disease
Tinea barbae: beard		-Oral antifungal: griseofulvin, terbinafine, or itraconazole -Air exposure -Topical antifungal: terbinafine, naftifine, butenafine ± Soaks with aluminum acetate -2nd line: topical azole	-Located on the non-bearded face -Children often acquire from cats or dogs -May be brought on by sunlight exposure 	**Tinea faciale**
Majocchi (trichophytic) granuloma	-Deep cutaneous infection 		-Erythema, scaling, vesicles -Maceration of web spaces -May have toenail involvement or bacterial 2° infection -"Moccasin" pattern with involvement of heels, soles, and lateral feet -May be inflammatory with bullae 	**Tina pedis**
Tinea manuum: hand	Similar to tinea pedis but on the hand -May be more aggressive 		-Erythematous, well-demarcated scaling plaques -Must differentiate from erythrasma or candidiasis (will fluoresce differently under Wood's lamp) 	**Tina cruris:** "jock itch"; groin and thighs
Tinea corporis: infection of neck, trunk, or extremities	-Sharp-bordered erythematous plaques of varying sizes -May have pustules or vesicles within the border -Lesions will enlarge peripherally and may have an area of central clearing 			

Disease	Information	Management	Disease	Information	Management
Tinea capitis: head	-Black dot hair loss -Scalp erythema and scaling -May see kerions 	-Oral antifungal: griseofulvin, terbinafine, or itraconazole ± Antifungal shampoo: selenium sulfide, ketoconazole	**Tinea unguium** (onychomycosis)	-3 forms: distal subungual, proximal subungual, and white superficial -Most pts will also have tinea pedis -Infection may be yeast or nail dystrophy = must to KOH scrape to be sure before starting therapy as terbinafine won't cover candidiasis 	-1st line is oral terbinafine -2nd line is oral azole or ciclopirox topical lacquer -3rd line is repeat therapy or nail removal
Tinea versicolor: caused by *Malasezzia furfur* = not really a tinea	-Hypo or hyperpigmented macular lesions -Especially on trunk -Fine rim of scale -KOH prep for spaghetti and meatballs 	-Topical selenium sulfide, pyrithione zinc, propylene glycol, ciclopirox, azole, or terbinafine ± UV light therapy -Systemic ketoconazole if recurrent or refractory			

OTHER DERMATOLOGY TOPICS

Acanthosis Nigricans

-19% of primary care patients will have this, with rates up to 47% in Native Americans

Etiologies
-Acquired or inherited
-Most commonly a result of obesity or endocrine or metabolic disorders, but can also be associated with genetic syndromes, familial acanthosis nigricans, malignancy, or drug reactions

Signs and symptoms
-Velvety, hyperpigmented plaques that most frequently occur on the neck and axillae but can appear on other skin sites or mucosal surfaces
-Rapid onset if associated with malignancy

Management
-Treat underlying disorder
-Weight loss, metformin or rosiglitazone
-D/c offending meds
-Topical retinoids, vitamin D analogs, and keratolytics

Lipomas

-Collections of mature fat cells encased in thin fibrous capsules
-The most common benign soft tissue neoplasm

Signs & symptoms
-Small golf ball under skin
-Soft, asymptomatic, mobile
-Do not enlarge quickly
-Multiple sites with familial multiple lipomatosis

Management
-Treatment only indicated for pain, cosmesis, or concern for malignancy
-Surgical excision

Prognosis
-Recurrence of excised lipoma is uncommon

Burns

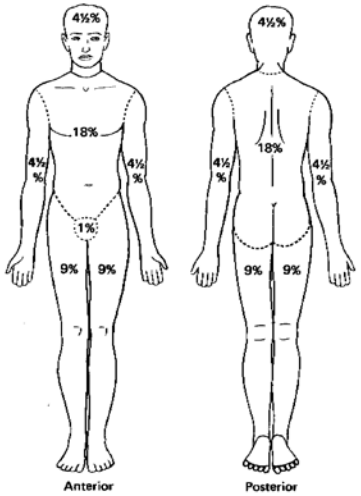
First Degree Burns
-Superficial burns only involving the epidermis
-Ex. sun burns, "flash" burns
-No blisters or edema, skin is pink or red, dry
-Will heal on their own in 3-6 days
-Are not included in burn calculations

Second Degree Burns
-Partial thickness burn that involves the dermis
-Superficial partial = small amount of dermis involved; caused by flame, scalding, or chemicals; moist, pink/red, edema, blistering, extremely sensitive to touch; heals in 10-21 days
-Deep partial = significant amount of dermis involved, more than 50%; caused by grease, flame, or chemicals; fewer capillaries left = appears white, dry, moderate edema, decreased sensation & circulation → minimal pain; may scar; may convert to full thickness burn; healing takes > 21 days
→ Tell these apart by degree of pain and pressure sensation

Third Degree Burns
-Full thickness burn, entire epidermis and dermis is gone, extends to subcutaneous fat
-A result of prolonged exposure to any heat source
-Extensive edema, dry, leathery, charred skin, no sensation or circulation
-Will not heal spontaneously, requires skin grafting

Fourth Degree
-Penetration to the bone
-Usually requires amputation

Calculation of Burn Area
-"Rule of 9s" for adults



Management
-Remove clothes
-Irrigate with room temp saline (not cold, will cause vasoconstriction)
-Cover wound to prevent heat loss
-Use LR for IVF using Parkland formula to estimate needed amount
-Keep room warm
-IV morphine
-NG decompression
-Tetanus booster
-Assess for smoke injury: facial burn, singed nose or facial hair, carbonaceous deposits in oropharynx
-Surgical management may be needed
-Biologic dressings for several weeks with daily cleansing and debridement, topical antibiotics
-Vaseline cause during exudative phase
-Indications for referral to burn center: > 10% body surface are with 2nd degree burns, any 3rd degree burns, electric or chemical burn, inhalation injuries, burns accompanied by trauma

Hidradenitis Suppurativa

-Chronic inflammatory skin disorder characterized by pustules, inflammatory nodules, and sinus tract development, usually in intertriginous areas

Prevention
-Avoiding skin trauma
-Careful skin hygiene
-Smoking cessation
-Reducing carb intake
-Weight reduction

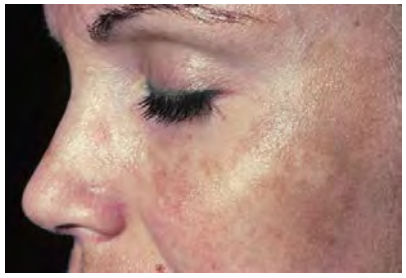
Signs & Symptoms
-Affected areas may be in the axillae, genitofemoral region, gluteal folds, or perianal areas
-Small, painful subcutaneous nodules can be palpated
-Pruritus
-Erythema
-Burning pain
-Local hyperhidrosis
-Sinus tract formation
-Hyperpigmentation, scars, and pitting of the skin

Management
-Hot packs for mild cases
-Topical or systemic antibiotics: clindamycin or doxycycline
-Retinoids
-Accutane
-Surgical removal, I&D, or skin grafting for severe cases
-I&D will not alter course of disease and should be reserved for pt comfort in times of tight skin abscess formation

Melasma

-Disorder of hyperpigmentation affecting sun-exposed areas of skin

Causes
-Pregnancy (occurs in up to 75% of pregnant women)
-OCPs
-Genetics
-Sun exposure
-Cosmetics
-Thyroid dysfunction
-Antiepileptics



Signs & Symptoms
-Usually appears on the face
-More pronounced in those with darker complexions

Workup
-Diagnosis is clinical

Management
-Broad spectrum sun protection
-Hydroquinone 4% cream

Epidermal Inclusion Cysts (Epidermoid Cysts)

-Discrete nodules with keratin-producing cell wall
-Erroneously referred to as sebaceous cysts but there is no sebaceous component

Gardner's syndrome = rare condition of multiple epidermal inclusion cysts associated with colon cancer

Signs & symptoms
-Discrete, freely movable cyst or nodule, often with central punctum
-Can have spontaneous inflammation and rupture
-May grow larger

Differential
-Pilar cyst: derived from hair root sheath

Management
-Be cautious of cysts that have been present on scalp since birth as these may communicate with CNS
-Kenalog injection for inflamed cysts
-I&D of infected cyst
-Surgical excision of non-inflamed cyst

Prognosis
-May resolve spontaneously without therapy

Pilonidal Disease

-An acquired condition likely related to mechanical forces on the skin overlying the natal pilonidal cleft → cavity formation containing hair, debris, and granulation tissue

Signs & Symptoms
-May have asymptomatic cyst
-Tenderness, erythema, and abscess formation just above the gluteal cleft

Management
-Acute abscess needs I&D with debridement of all visible hair
-Refer for surgical excision of recurrent pilonidal disease
-Primary closure associated with faster healing but delayed closure associated with lower likelihood of recurrence
-Antibiotics only for cellulitis

Decubitus Ulcers (Pressure Ulcers)

-Infections of these are typically polymicrobial, including staph and strep, enterococci, *Enterobacter*, *Proteus*, and anaerobes

Risk factors
-Shearing forces, friction, moisture, immobility, incontinence, poor nutrition, decreased skin perfusion

Staging
-Stage 1 = intact skin but with non-blanchable redness for > 1 hour after relief of pressure
-Stage II = blister or other break in the dermis ± infection
-Stage III = full thickness tissue loss, may see subcutaneous fat, destruction extends into muscle ±infection, may have undermining or tunneling
-Stage IV = full thickness skin loss with involvement of bone, tendon, or joint ± infection, often with undermining or tunneling
-Unstageable = full thickness tissue loss in which ulcer base is covered by slough or eschar
-Suspected deep tissue injury = purple or maroon localized ara of discolored intact skin or blood-filled blister due to damage of underlying tissue from pressure or shear

Differential
-Diabetic neuropathy ulcer
-Arterial or venous insufficiency ulcer

Workup
-MRI is best for evaluation of osteomyelitis
-Wound cultures: gold standard sample is of a deep tissue specimen from a surgically cleaned and debrided ulcer; superficial cultures tend to represent colonization rather than true wound infection

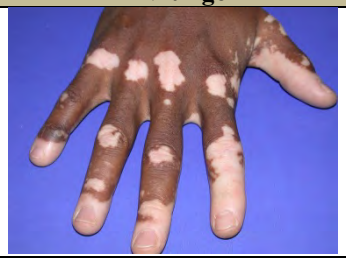
Management
-Comprehensive analysis of patient's general medical condition and evaluation of the wound
-Daily monitoring using healing and staging scales
-Adequate pain control
-Nutritional supplementation if needed
-Repositioning every 2 hours
-Dressing choice depends on wound characteristics: wet to dry, semiocclusive, occlusive, sterile maggots, etc.
-Stage 1 ulcers need transparent film dressing
-Stage 2 ulcers need a moist wound dressing
-Stage 3 and 4 ulcers need infection treatment, debridement, and other special dressings
-For superficial infections, topical antibiotics such as sulfadiazine are used to reduce bacterial counts
-For deeper infections or complicated infections, systemic therapy is needed

Prognosis
-Complications: bacteremia, sepsis, death

Vitiligo

-Acquired skin depigmentation as a result of autoimmunity against melanocytes

Signs & symptoms
-Usually is generalized (vitiligo vulgaris): widespread macules and patches that are often symmetric
-Comorbid autoimmune disease: autoimmune thyroiditis, pernicious anemia, SLE, Addison disease



Differential
-Postinflammatory hypopigmentation
-Chemically-induced depigmentation
-Tinea versicolor
-Pityriasis alba
-Morphea
-Lichen sclerosus
-Leprosy

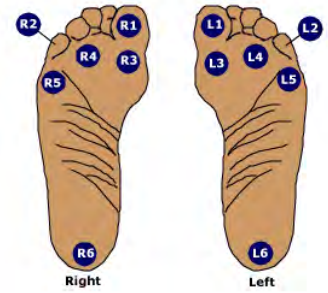
Management
-Repigmentation therapies
-Topical steroids
-Topical calcineurin inhibitors
-UV light

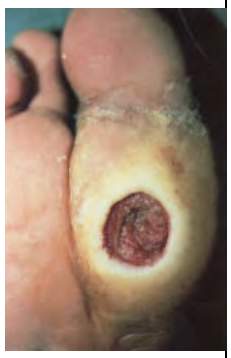
Leg Ulcers

Risk factors
-Poor circulation, venous insufficiency, disorders of clotting, diabetes, sickle cell, neuropathy, renal failure, HTN, lymphedema, inflammatory skin diseases, smoking, genetics, malignancy, meds

Diabetic (Neurotrophic) Ulcers

Screening
-Recommended annually with visual examination and monofilament test (checks most common sites of ulceration)





Signs & symptoms
-Ulcers with punched-out borders with calloused surrounding skin
-Underlying neuropathy

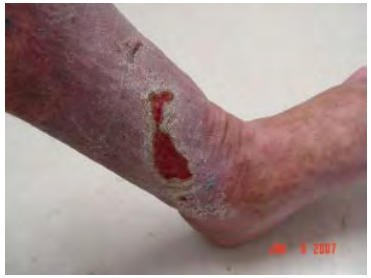
Workup
-Ankle-brachial index with symptoms of PAD

Management
-Comprehensive assessment of ulcer and patient's overall medical condition
-Classification of wound at each follow-up
-Debridement, local wound care, pressure relief, infection control, and proper dressing selection
-Negative pressure wound therapy following debridement after infection, necrosis, or amputation
-Revascularization for critical wound ischemia

Venous Insufficiency Ulcers

-Classified by CEAP system, which helps distinguish initial disease severity as well as changes over time
-Varicose veins in the absence of skin changes are NOT chronic venous insufficiency!

Risk factors
-Advancing age, FH, increased BMI, smoking, h/o LE trauma, prior DVT, pregnancy

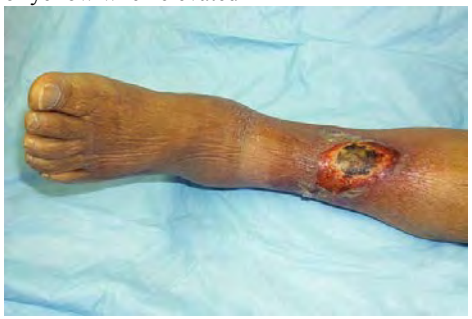


Signs & symptoms
-C/o tired, heavy legs, leg pain, or leg swelling
-Telangiectasias, reticular veins, and varicose veins
-Edema, inflammation, pruritic dermatitis
-Ulcers with irregularly shaped borders along the medial ankle or saphenous veins that are tender, shallow, exudative, and have a base of granulation tissue
-Skin discoloration or redness, may appear shiny or tight
-20% of symptomatic patients will have no visible clinical signs

Management
-Leg elevation, exercises, and graduated compression stockings
-SCDs for patients refractory to stockings
-Horse chestnut seed extract for patients who can't tolerate or are noncompliant with compression therapy
-Skin moisturizers
-Wound debridement PRN
-Barrier creams to protect adjacent skin
-Selection of proper wound dressing
-NOT effective: topical antibiotics, debriding enzymes, growth factors, or honey
-Compression bandages for severe edema, weeping, eczema, or ulceration
-Aspirin therapy accelerates healing
-Referral to subspecialty for slowly healing ulcers, persistent dermatitis, or recurrent cellulitis

Arterial (Ischemic) Ulcers

Signs and symptoms
-PAD symptoms: pain and claudication with walking that is relieved by rest (however may have more pain with leg elevation in severe disease)
-Ulcers are usually on the feet at points of friction and appear punched-out
-Feet will turn red when dangled and pale white or yellow when elevated



Human & Animal Bites

-Risk of infection in cat bites > human bites > dog bites

Workup
-Check involved animals for rabies
-CBC, blood and wound cultures if febrile or wound appears infected
-Head CT for deep dog bite to the scalp

Management
-Primary closure of wounds that are cosmetically important, clinically uninfected, < 12 hours old (or < 24 on the face), and not located on hand or foot
-Leave wounds open that are crush injuries, puncture wounds, bites of the hand or feet, wounds > 12 hours old (or > 24 on the face), cat or human bites, or bite wound in immunocompromised host
-Tetanus or rabies vaccination if indicated
-Primary care f/u in 48-72 hours
-Empiric antibiotics for animal and human bites: Augmentin drug of choice
-Deep or severe wound infections from animal or human bites require IV antibiotics: Zosyn

HEMATOLOGY

ANEMIAS

Aplastic Anemia

-A result of bone marrow failure due to injury or suppression

Etiologies
-Idiopathic
-Drugs: phenytoin, sulfas, chemo, radiation, chemicals
-Viruses
-Pregnancy
-Hereditary (Fanconi's anemia)

Signs & symptoms
-Abrupt onset of fatigue, weakness, dyspnea, excess bleeding & bruising, petechiae, purpura, pallor, and infections

Workup
-Labs show pancytopenia, severe normocytic anemia, ↓ retics, reduced cells in BM with fat replacement

Management
-Based on severity of disease
-None if mild
-BMT or immunosuppression if severe

Microcytic Anemia = MCV < 80

Iron Deficiency Anemia

Etiology
-In an adult, this is due to blood loss, likely GI, until proven otherwise

Signs & symptoms
-Fatigue
-Dyspnea on exertion
-Tachycardia
-Cheilosis
-Spoon-shaped nails
-Pica
-Dysphagia due to webbing of the esophagus

Management
-Treat blood loss
-Oral iron with stool softeners, continue 3-6 months post Hb recovery
-Consider parenteral therapy by heme if pt does not tolerate oral therapy or it is not rapid enough
-Recheck CBC in 3-4 weeks and ferritin in 8 weeks

Chronic Inflammation Anemia (Anemia of Chronic Disease)

Etiology
-Usually from reduced erythropoietin stimulation of bone marrow

Workup
-A disease of exclusion

Management
-Treat only it pt is symptomatic with folate, iron, EPO

Sideroblastic Anemia

Etiology
-Inherited, acquired, or idiopathic heme synthesis from alcohol, lead, myelodysplasia, leukemia, TB, or drugs

Workup
-BM biopsy showing ringed sideroblasts

Management based on cause

Macrocytic Anemia = MCV > 100

Vitamin B 12 Deficiency

Etiology
-Inadequate diet: vegetarians
-Malabsorption
-Drugs

Signs & symptoms
-Abnormal sensation and peripheral neuropathy in stocking-glove pattern
-Glossitis
-Pallor
-Anorexia
-Diarrhea

Folate Deficiency

Etiology
-Inadequate diet: alcoholics
-Dialysis
-Malabsorption
-Impaired metabolism

Signs & symptoms
-Glossitis
-Diarrhea
-Malnourishment
-Cheilosis
-No neuropathies

Hemolytic Anemia

Etiologies
-Hereditary spherocytosis: intrinsic inherited defective spectrins in RBC membrane → weak, deformed spherical RBCs prone to rupture in blood vessels or spleen
-Hereditary elliptocytosis
-G6PD deficiency: results in oxidation-prone RBCs → intracellular ppt of oxidized Hb into Heinz bodies → bite cells and hemolysis after journey through the spleen
-Sickle cell disorders
-Acquired disorders: TTP, HUS, giant hemangioma, artificial heart valves, sepsis, DIC, autoimmune hemolytic anemia, hemolytic disease of the newborn

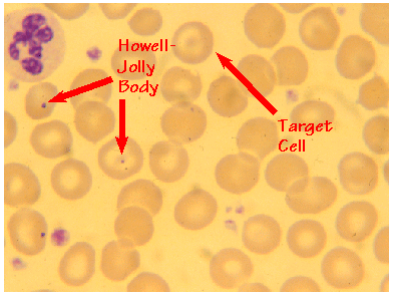
Signs & symptoms
-Rapid onset of pallor and anemia
-Jaundice with ↑ unconjugated bili
-Bilirubin gallstones
-Splenomegaly

Workup
-Increased hemolysis markers: LDH
-Reduced or absent serum haptoglobin (since it binds free Hb, which is abundant during hemolysis)
-May have + Coomb's test (DAT)
-Increased retic % or retic # as BM responds to anemia
-Abnormalities on peripheral smear

Management
-Treat underlying cause

Sickle Cell Disease

-Triggers for crisis can be dehydration, infection, and acidosis as they increase cell sickling
-Usually pts are functionally asplenic as their spleen becomes infarcted by a young age
-Disease can be AS (trait), SS, SC, or S + other Hb variant



Signs & Symptoms
-Anemia → pallor and fatigue
-Hemolysis → jaundice and gallstones
-Dactylitis
-Leg ulcers
-Priapism
-Pulmonary, cerebral, and splenic emboli → functional asplenia, stroke, PE
-Retinal artery obstruction → blindness
-Crisis: skeletal pain, fever, anemia, jaundice
-Acute chest syndrome
-Splenic sequestration crisis
-Aplastic crisis: worsening of anemia from parvovirus B19 infection
-Pulmonary HTN
-CKD (may progress to ESRD)
-Osteomyelitis

Workup
-Peripheral smear showing sickled cells and Howell-Jowell bodies reflecting splenic dysfunction
-Reticulocytosis from chronic hemolysis

Outpatient Management
-Vaccinations: Pneumovax, flu, Hib, Menactra, hep B
-Prophylactic penicillin until age 5
-Folate, MVI, hydroxyurea
-Funduscopic exam for proliferative retinopathy risk
-Screening echo and BNP every 2 years after age 21
-BMP and UA twice a year
-Mental health assessments
-Birth control discussion (risk of crisis ↑ during pregnancy, hydroxyurea is pregnancy cat D)

Crisis Workup
-↑bili and LDH from hemolysis
-Low haptoglobin (binds up the lysed Hb)
-Cultures for fever

Management of crisis
-Pain control for opioid naïve: NSAIDS or APAP + codeine
-Next step: oral morphine, oxycodone, or hydromorphone
-Severe pain: IV fentanyl, morphine, or hydromorphone
-Breakthrough pain: start PCA
-IVF
-Bronchodilators for wheezing
-Incentive spirometry to prevent atelectasis
-Supplemental O2
-May need transfusion for low Hb
-DVT prophylaxis (hypercoagulable state)
-Empiric ceftriaxone for fevers

Complications
-Acute chest syndrome: #1 cause of death in sickle cell, defined as a new radiodensity + fever/resp symptoms, caused by hypoventilation/pulmonary infarct/pulmonary edema/TRALI, treat with abx to cover CAP (because it can't be distinguished from pneumonia), IVF, bronchodilators, incentive spirometry, supplemental O2, transfusion for Hb < 10
-Chronic lung disease and pulmonary HTN
-Stroke
-Bone/joint: osteomyelitis, septic arthritis, osteonecrosis, vertebral body collapse, stunted growth, osteopenia
-Chronic kidney disease from renal infarcts
-Priapism

G6PD Deficiency

-X-linked disorder of the enzyme that catalyzes reactions to prevent oxidative injury RBCs

Screening
-Not performed routinely in newborns

Signs & symptoms
-Most affected patients are asymptomatic
-Neonatal jaundice
-Increased susceptibility to infection
-Episodic anemia following exposure to infection, drugs, or chemicals → HA, nausea, back pain, chills, fever, hemoglobinuria, jaundice
-Some may have chronic hemolysis

Workup
-Several screening tests available

Management
-Avoid fava beans
-Avoid certain meds: dapsone, nitrofurantoin

Prognosis
-May incur protection against malaria
-Severe episodes of hemolysis can be fatal

Thalassemia

Alpha Thalassemia

-Decrease or absence of alpha globulin
-At risk: SE Asian, Mediterranean, African

Signs & symptoms
-HBH disease → splenomegaly, pallor
-A thal trait → normal clinic presentation under non-stressful conditions

Management
-For full disease, splenectomy, folate, avoid Fe

Beta Thalassemia

-Decrease or absence of beta globulin
-Body may compensate by increasing % of HbA2 and HbF
-At risk: Italian, Greek, Asian, African

Signs & symptoms
-Major disease (Cooley's) → jaundice, hepatosplenomegaly, bony abnormalities, chipmunk face, growth retardation
-Intermediate disease → moderate chronic anemia, Fe overload
-Minor disease → rare anemia

Management
-Only severe disease is treated with transfusions, folate, Fe chelation therapy, splenectomy

COAGULATION DISORDERS

Thrombocytopenias

*Remember to distinguish from pseudothrombocytopenia in patients that are "EDTA clumpers" or in patients on aspirin, clopidogrel, prasugrel, NSAIDs, or other antiplatelets

Acute Thrombocytopenia	Immune Thrombocytopenic Purpura	Heparin-Induced Thrombocytopenias	Thrombotic Thrombocytopenic Purpura	Hemolytic Uremic Syndrome	Disseminated Intravascular Coagulation	Other Causes
-Sudden ↓ in platelets from destruction, less production, or sequestration -S/s: dried blood in nose, wet purpura in the mouth, splenomegaly, petechiae -Plt will be < 150k	-Due to anti-platelet Abs -Can be from viral infection in kids that is self-limiting -In adults tends to be chronic with no prior infection, but can also occur after valve replacement, cardiac cath, drugs -S/s: neurologic symptoms, fever, ± renal failure -W/u: normal PT/PTT, normal cell lines, normal marrow, normal spleen = dx of exclusion -Tx: steroids, immune modulation with IV Ig or splenectomy, plasmapheresis, EPO -90% fatal without treatment	-HIT type I is non-immune, transient, and improves upon d/c of heparin -HIT type II is due to IgG against PF4 →formation of immune complexes that activate platelets to form microthromboses in small vessels throughout body -S/s: MAHA, limb necrosis, pulseless extremities -W/u: plt ~50k, normal PT/PTT, PF4 Ab, schistocytes on peripheral smear -Tx: stop heparin, use direct thrombin inhibitors, and begin warfarin after plt normalize	-Caused by Ab to ADAMSTS13 → extensive microthromboses throughout body -Assoc with certain meds -S/s: AMS, fevers, chest pain, dyspnea, not urinating, similar to acute thrombocytopenia or ITP -W/u: plt ~20k, normal PT/PTT, schistocytes -Tx: plasmapheresis to remove Ab, steroids	-Classically caused by *E. coli* damage to endothelia and renal arterioles and inactivation of ADAMSTS13 → plt activation → thrombocytopenia, uremia -Very similar to TTP, distinguish it by renal dysfunction vs neuro impairments -Tx: supportive, volume repletion, pressors, plasma exchange; only give pRBCs with severe anemia; NO abx as it will worsen HUS, NO plt as this will worsen MAHA	-A pathological activation of coagulation that is always associated with an underlying disease such as sepsis, burns, cancer, head trauma, snake bite, or vasculitis -W/u: ↓ plt, schistocytes, + D-dimer, ↓ fibrinogen (all used up), prolonged PTT/PT (factors all used up) -Tx: treat underlying disease, replace blood products PRN, may need LMWH to inhibit further clotting	-Liver disease →low thrombopoietin (tx with FPP) -Vit K deficiency due to abx killing vit K synthesizing gut bacteria, malnutrition, or biliary tract disease (tx with vit K supp + FFP)

Thrombopathies

-Characterized by prolonged bleeding time despite normal platelet counts

Von Willebrand Disease	Glanzmann's Thrombasthenia	Bernard-Soulier Syndrome	Hemophilia	Other Thrombopathies
-The most common hereditary coagulation abnormality -A result of a functional or quantitative deficiency of vWF -A spectrum of disease -W/u: electrophoresis shows decreased vWF multimers (the opposite of TTP), hyperagglutination with ristocetin, prolonged closure time for collagen and ADP -Tx: only treat severe cases with vWF & factor 8 concentrates, or desmopressin for quick release of vWF from endothelial stores	-Rare genetic disease where platelets lack glycoprotein IIb/IIIa = no fibrinogen or vWF bridging can occur -W/u: platelet agg studies are the opposite of Von Willebrand disease	-Rare genetic disease that causes a deficiency of the receptor for vWF -Giant platelets dominate = functional thrombocytopenia -W/u: giant platelets on peripheral smear, platelet agg studies are the opposite of Von Willebrand disease	-Hemophilia A is factor 8 deficiency -Hemophilia B is factor 9 deficiency -S/s: hemarthroses, muscle bleeds, other abnormal bleeding -W/u: prolonged PTT, decreased factor 8 or 9 -Tx: factor concentrates	-Decreased factor 8 due to autoimmunity (differentiate from hemophilia by doing a mixing study, it won't correct) -Acquired thrombopathy from drugs, infection, renal disease, hepatic disease, AIDS, NSAIDs

Hypercoagulable States

Hereditary

Factor V Leiden
-Hereditary resistance to factor 5 inactivation by protein
-PTT may be shortened and does not correct
-Definitive PCR test

Prothrombin G20120A
-Inherited mutation that causes increased prothrombin levels
-Definitive DNA test

Protein C or protein S deficiency
-Deficiency of natural vitamin K-dependent anticoagulants

Antithrombin III deficiency
-Reduced inhibition of the conversion of fibrinogen to fibrin
-Fatal if homozygous

Acquired

Virchow's triad
-Vascular damage, hypercoagulability, vascular stasis = after surgical procedures

Anti-PL syndrome
-Recurrent spontaneous abortions
-Livedo reticularis
-Treat indefinitely with warfarin

Others
-Hormones: pregnancy, OCPs
-Malignancy
-Smoking
-Immobilization
-Surgery

Disseminated Intravascular Coagulation

-Can be acute or chronic

Etiologies
-Usually occurs when shock causes widespread activation of the clotting cascade
-Sepsis
-Trauma and tissue destruction
-Malignancy
-Obstetric complications: placental abruption, HELLP syndrome, hemorrhage, septic abortion

Signs & Symptoms
-Bleeding diathesis: petechiae, ecchymosis, oozing from wounds and IVs
-Thromboembolism
-Renal dysfunction: AKI
-Hepatic dysfunction: jaundice
-Respiratory dysfunction: hemoptysis, dyspnea
-Shock
-CNS involvement: coma, delirium, TIAs

Workup
-Peripheral smear shows microangiopathic hemolytic anemia
-Low platelets and clotting factors (may be near normal in chronic DIC)

Management
-Treat underlying disease
-Hemodynamic support
-Most coagulopathies are short-lived but some pts with severe bleeding may need platelets or FFP transfusions

MALIGNANCIES

Acute Lymphoblastic Leukemia

-Cancer of the lymphoid progenitor, affecting B or T cell lineage
-Incidence peaks @ 2-5 years and drops @ 8-10 years = more common in children

Signs & symptoms
-Illness over days to weeks
-Fever, pallor, petechiae, ecchymoses, lethargy, malaise, anorexia, bone or joint pain, meningitis, weight loss, lymphadenopathy, splenomegaly, dyspnea

Differential
-Infection
-Aplastic anemia
-Juvenile RA
-Other malignancy

Workup
-CBC with differential showing 1-2 cytopenias
-Confirmatory blood smear or bone marrow aspirate showing blasts
-LP to eval for CNS involvement

Management
-3-4 agent induction therapy
-Radiation if CNS disease is present
-Continuous therapy for 2-3 years

Prognosis
-Overall cure rate 20-40% for adults

Acute Myelogenous Leukemia

-Cancer of the myeloid progenitor (gives rise to all WBCs other than B/T and NK cells), where cells do not mature and do not die and take up the bone marrow space of other needed cells
-Most common in first 2 years at life, peaks again in adolescence numbers of blasts

Signs & symptoms
-From cell deficiencies: pallor, fatigue, dyspnea, thrombocytopenia, petechiae, hematomas, bleeding, neutropenia with sepsis, cellulitis, pneumonia
-From hyperleukocytosis: obstruction of capillaries and small arteries with high
-From CNS involvement: HA, AMS, CN issues
-Leukemia cutis lesions
-DIC
-Tumor lysis syndrome

Workup
-Differentiate from ALL by peripheral smear showing Auer rods

Management
-Aggressive chemo

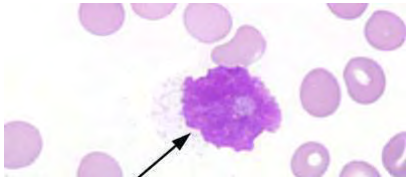
Prognosis
-Overall survival of 30%

Chronic Lymphocytic Leukemia

-Clonal proliferation and accumulation of mature-appear B cells
-The most commonly occurring leukemia
-Mostly occurs in those > 50, and more common in males
-RAI system for staging

Signs & symptoms
-Fatigue, night sweats, weight loss, persistent infections, lymphadenopathy, hepatomegaly, splenomegaly

Workup
-CBC showing lymphocytosis with WBCS > 20k with concomitant anemia and peripheral smear showing mature small lymphocytes and cobblestone-appearing smudge cells
-Coexpression of CD19 and CD5
-High IgG



Management
-Observation
-Chemo with tumor lysis prophylaxis
-BMT
-Radiation for lymphadenopathy

Prognosis
-Typically slow-growing, but has potential for Richter's transformation to aggressive disease
-Worse prognosis with deletion of chromosome 17
-Average 5 year survival rate of 50%

Chronic Myelogenous Leukemia

-Excess proliferation of the myeloblast or its progeny with no negative feedback
-Usually occurs in young to middle age adults

Categories
1.) Chronic: < 15% blast component of bone marrow or blood
2.) Accelerated: peripheral blood > 15% blasts or > 30% blasts + promyelocytes, or > 20% basophils
3.) Acute: when blasts comprise > 30% of BM; final phase of CML

Signs & symptoms
-Fever, bone pain, LUQ pain with splenomegaly, weakness, night seats, bleeding & bruising, petechiae

Workup
-Detection of Philadelphia chromosome via FISH or RT-PCR
-CBC showing leukocytosis and thrombocytopenia

Management
-Chemo
-BMT

Prognosis
-Average survival is 6 years with treatment

Multiple Myeloma

-Malignancy of plasma cells where replacement of bone marrow leads to failure
-Etiology is unknown, but there is increased incidence with h/o pesticides, paper production, lather tanning, nuclear radiation exposure, and abnormalities of chromosome 13
-Multi-hit hypothesis that development of MM requires 2 oncologic events: MGUS (a common, age-related medical condition characterized by accumulation of monoclonal plasma cells in the BM → moderate IgG spike on electrophoresis) + 2nd hit causing transition of MGUS to severe MM

Signs & symptoms
-Forms lytic lesions on bone → bone pain, pathologic fx, and hypercalcemia
-Renal failure from excretion of proteins
-Fatigue
-Recurrent infections
-Spinal cord compression
-Hyperviscosity syndrome from high circulating Ig of all kinds

Workup
-Bone marrow biopsy shows > 5% plasma cells
-Lytic lesions on metastatic bone survey x-ray series
-Spikes in M protein in protein electrophoresis (differentiate from MGUS, where M protein level will still be WNL)
-IgG and IgA spikes on electrophoresis
-Peripheral smear showing rouleaux formations (poker chips)
-Urine has Bence-Jones proteins (produced by malignant plasma cells)
-Hypercalcemia
-Anemia

Management
-Chemotherapy
-Local radiation for pain control
-Autologous BMT
-Bisphosphonates for hypercalcemia

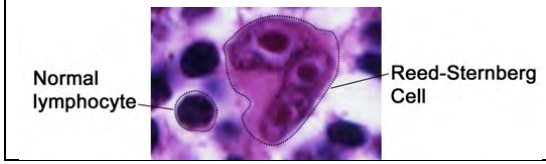
Prognosis
-Average survival with chemo is 3 years, 7 years for BMT

Lymphoma

Hodgkin Lymphoma

-A group of cancers characterized by orderly spread of disease from one lymph node to another and by the development of systemic symptoms with advanced disease
-Extranodal presentation in the lung, liver, or BM in some cases
-Peaks in adolescence and young adulthood, and in ages 50+
-Association with EBV

Signs & symptoms
-Painless, firm lymphadenopathy (often supraclavicular and cervical areas), mediastinal mass causing cough or SOB, fever, weight loss



Workup
-Peripheral smear showing Reed-Sternberg cells

-CT scans of chest, abdomen, and pelvis
-PET scan
-BM biopsy
-Lymph node biopsy

Management
-Chemo
-Low dose radiation

Prognosis
-Overall survival 90% but there are 3 separate risk groups

Non-Hodgkin Lymphoma

-A diverse group of blood cancers that include any kind of lymphoma except Hodgkin (includes CLL, Waldenstrom's, and multiple myeloma)
-Associated with congenital or acquired immunodeficiency
-Single or multiple areas of involvement
-Low, intermediate, and high grades
-Incidence increases with age

Signs & symptoms
-Lymphadenopathy → hydronephrosis, bowel obstruction, jaundice, wasting, SVC syndrome
-Abdominal pain
-Fever, weight loss, night sweats
-Edema

Workup
-CBC and smear are usually normal
-Lymph node biopsy
-CT scans of chest, abdomen, and pelvis

Management
-Systemic chemo

Prognosis
-70-90% survival rate

INFECTIOUS DISEASES

Fungal Disease

Cryptococcosis

-*Cryptococcus neoformans*
-Invasive fungal infection that is becoming increasingly prevalent in the immunocompromised population (AIDS, prolonged steroids, organ transplant, malignancy, sarcoidosis)
-Associated with soil frequented by birds and with rotting vegetation
-Worldwide distribution

Signs & symptoms
-Pulmonary infections → solitary, non-calcified nodules
-Meningoencephalitis: seen in HIV, sx occur over 1-2 weeks,

Workup
-Must culture organism from CSF for definitive dx of meningitis but can presumptively ID with CSF Ag testing

Treatment
-Amphotericin B and flucytosine for meningitis

Pulmonary Histoplasmosis

-*Histoplasma capsulatum*
-The most prevalent endemic mycosis in the US
-Associated with bird and bat droppings, chicken coops, farm buildings, abandoning buildings, caves, wood lots
-Most infections will be asymptomatic or self-limiting

Signs & symptoms
-Symptoms begin weeks to months following exposure
-Pneumonia with mediastinal or hilar lymphadenopathy
-Mediastinal or hilar masses
-Pulmonary nodule
-Cavitary lung disease
-Pericarditis with mediastinal lymphadenopathy
-Arthritis or arthralgia + erythema nodosum
-Dysphagia from esophageal narrowing
-SVC syndrome
-With dissemination: fever, fatigue, weight loss, GI, CNS, adrenal manifestations

Differential
-Sarcoidosis
-TB
-Malignancy

Workup
-CXR looks just like sarcoid
-Histo serologies
-Special stains on biopsy specimens
-EIA: urine, blood, or BAL

Management
-Itraconazole for moderate infection, amphotericin B for severe

Candidiasis

-Candidiasis of the esophagus, bronchial tree, or bladder are a result of severe immunosuppression → think HIV

Oral Candidiasis (Thrush)

-Risk factors: inhaled steroid use, AIDS, antibiotic use, radiation therapy

Presentation
-Pseudomembranous candidiasis: most common; white plaques with underlying red mucosa
-Erythematous candidiasis: no white component; agent of denture stomatitis
-Hyperplastic candidiasis: thick white patches that can't be scraped off
-Angular cheilitis: corners of mouth
-Glossitis with broad spectrum antibiotic use
-Cottony feeling in mouth
-Loss of taste
-Pain with eating and swallowing
-May be asymptomatic

Workup
-KOH prep of mouth scrapings

Management
-Nystatin swish or cream
-Disinfect dentures
-Oral fluconazole if severe
-Infants: oral nystatin swabs for 7-14 days, boiling of bottle nipples and pacifiers

Vaginal Candidiasis

See section on vaginitis

Cutaneous/Intertriginous

-Commonly affected sites are under the breasts, abdominal folds, axillae, groin, web spaces, diaper areas

Presentation
-Beefy red plaques with satellite lesions

Management
-Nystatin powder

Pneumocystis Pneumonia

-*Pneumocystic jiroveci*
-Occurs in immunosuppressed individuals: HIV, stem cell or organ transplant, cancer pts, chronic steroid therapy, chemotherapy

Prevention
-PCP prophylaxis for HIV+ individuals with h/o PCP, CD4 < 200, or h/o candidiasis with Bactrim DS daily

Signs & symptoms
-Fever
-Dry cough
-Fulminant respiratory failure

Workup
-CXR typically shows diffuse, bilateral interstitial infiltrates
-Sputum induction or BAL with Gram stain

Management
-Begin empiric therapy if suspecting with Bactrim, atovaquone, or IV pentamidine
-Adjunct steroids with PaO2 < 70 or hypoxemia

Bacterial Disease

Acute Rheumatic Fever

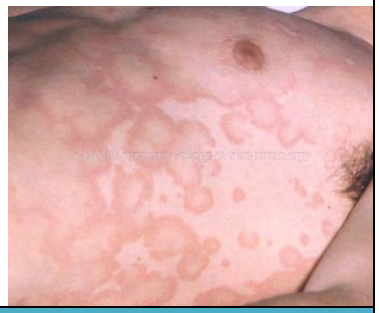
-Sequelae of GAS pharyngitis 2-4 weeks after infection (does not occur with impetigo) → inflammatory lesions of heart, joints, and subcutaneous tissue
-Peak incidence in 5-15

Screening
-Echo for children and young adults in countries where rheumatic fever is endemic

Signs & Symptoms
-Onset of symptoms 1-5 weeks from start of infection
-Major Jones criteria: migratory arthritis, carditis, valvulitis, CNS involvement (chorea), erythema marginatum, subcutaneous nodules
-Minor Jones criteria: arthralgia, fever, elevate ESR or CRP, prolonged PR interval

Workup
-2 major or 1 major + 2 minor criteria indicate a high probability of acute rheumatic fever

Management
-Treat whether or not pharyngitis is present with penicillin, cephalexin, or azithromycin, and treat all household contacts with + throat cultures
-Aspirin is DOC for anti-inflammatory, even in kids
-HF management if present
-Continuous prophylaxis against GAS (due to increased severity with subsequent infection) with IM penicillin G after end of initial treatment until 18-25 years of age, indefinitely if valvular disease is present, and for one year if reactive arthritis is present



Botulism

-Rare but life-threatening neuroparalytic syndrome resulting from a neurotoxin released by the bacterium *Clostridium botulinum*
-Clostridia are ubiquitous in the environment, including soil, seafood, and on the surfaces of fruits and vegetables
-110 cases per year occur in the US

Foodborne botulism
-Ingested preformed botulinum toxin
-Incubation of hours to 1 week
S/s: n/v/d, abdominal pain, dry mouth, followed by CN involvement and weakness

Infant botulism
-Accounts for most US cases of botulism
-S/s: constipation, weakness, feeding difficulties, hypotonia, drooling, irritability, weak cry

Wound botulism
-Can occur with all kinds of wounds, not just puncture wounds
-S/s: no prodromal GI symptoms

Adult enteric infectious botulism
-Enteric colonization without source of preformed toxin ingestion
S/s are same as foodborne botulism

Inhalational botulism

Signs & symptoms
-Acute onset of bilateral cranial neuropathies
-Symmetric descending weakness
-Absence of fever
-Patient remains responsive
-Normal or slow HR and normal BP
-No sensory deficits except for blurred vision
-Nonspecific GI symptoms

Differential
-Myasthenia gravis
-Tick paralysis
-Guillain-Barre syndrome
-Polio
-Stroke
-Heavy metal intoxication

Management
-Botulinum antitoxin
-Additionally, penicillin G for wound botulism

Chlamydia

-Most commonly reported STI in US
-Frequent coinfection with gonorrhea

Screening
-Every year for women < 26
-When there is a new sex partner in last 60 days
-With > 2 new sex partners in a year

Signs & symptoms
-May be asymptomatic
-Vaginal discharge
-Dysuria
-Cervical friability or ectropion
-Pelvic or lower abdominal pain
-Ectopic pregnancy
-Perihepatitis
-Lymphogranuloma venereum with L serotypes

Workup
-Cervical swab with PCR is best
-Urine test for men

Treatment
-1st line is azithromycin or doxycycline
-2nd line is erythromycin or levofloxacin
-Sexual abstinence for 7 days from initiation of therapy
-Treat for gonorrhea as well

Prognosis
-Need retesting 3 months after treatment
-Complications: PID, epididymitis, urethritis, sterility

Cholera

-*Vibrio cholerae*
-US cases are only acquired overseas or via consumption of contaminated seafood

Prevention
-Dukoral vaccine available

Signs & symptoms
-Severe, watery diarrhea
-Vomiting

Workup
-Stool Gram stain for curved Gram neg rods
-PCR for toxinogenic strains

Management
-Begin treatment before definitive diagnosis!
-Rehydration
-Antibiotics: doxycycline, FQ if resistant

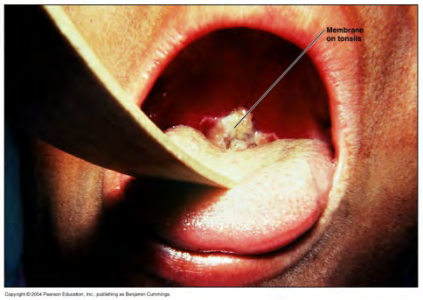
Diphtheria

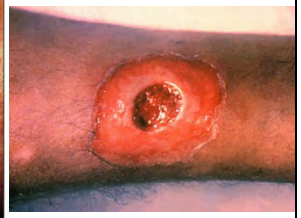
-Agent is *Corynebacterium diphtheriae*
-Transmission is direct or droplet
-Humans are the only reservoir and immunization does not prevent carriage
-Some strains produce respiratory toxin → heart and nervous system damage (why we vaccinate)

Signs & symptoms
-May be asymptomatic
-Sore throat
-Low grade fever
-Malaise
-Cervical lymphadenopathy
-Diphtheria membrane
-Cutaneous diphtheria

Workup
-Throat and membrane cultures

Treatment
-Erythromycin or penicillin
-Airway management
-Prophylax close contacts





Gonorrhea

Screening
-Every year for women < 26
-When there is a new sex partner in last 60 days
-With > 2 new sex partners in a year

Signs & symptoms
-Vaginal discharge
-Abdominal pain
-Cervicitis
-Most men will be symptomatic with purulent discharge, dysuria, urethritis
-Pharyngitis

Workup
-Cervical swab with PCR is best
-Urine test for men
-May need to culture rectum

Treatment
-1st line is ceftriaxone injection
-2nd line is cephalosporin
-If pharyngitis is present add azithromycin or doxycycline
-Treat for chlamydia as well

Prognosis
-Complications: PID, tubo-ovarian abscess, perihepatitis, vertical transmission

Tetanus

-Caused by the soil anaerobe *Clostridium tetani*

Signs & Symptoms
-Muscle spasms
-Inadequate vaccination history

Workup
-Diagnosis is clinical

Management
-Neutralize unbound toxin using tetanus Ig
-Metronidazole to eradicate remaining *Clostridia*
-Benzos to control spasms
-Labetalol for autonomic hyperactivity

Methicillin Resistant Staph Aureus (MRSA)

-Healthcare-associated MRSA (HA-MRSA) is associated with severe, invasive disease in hospitalized patients
-Community-acquired MRSA (CA-MRSA) can occur in skin and soft tissue infection in young healthy adults with no recent healthcare exposure
-Transmission occurs via contact with a colonized individual or contaminated fomite

Management
-Invasive infections → vancomycin or daptomycin
-Osteomyelitis → vancomycin or daptomycin
-Outpatient management of MRSA skin and soft tissue infection → clindamycin, Septra, or doxycycline
-Inpatient treatment of severe MRSA skin and soft tissue infection → vancomycin, linezolid, or daptomycin

Pertussis

Prevention
-Dtap vaccine series for kids
-Tdap vaccination for adults to protect kids

Signs & symptoms
-Initial: cold-like; rhinorrhea, lacrimation, dry cough with episodes of severe cough, low-grade fever; post-tussive emesis
-Paroxysmal stage: coughing becomes more severe and may persist up to 10 weeks at this stage; paroxysmal whooping may be heard
-Convalescent stage: coughing diminishes as patient recovers and disappears over 2-3 weeks but may recur with subsequent URIs

Workup
-*Bordetella* culture or PCR from nasopharyngeal swab

Management
-Macrolides are DOC
-Septra is an alternative

Prognosis
-May be infectious for several weeks if untreated

MYCOBACTERIAL DISEASE

Tuberculosis

Signs & symptoms
-Latent or primary infection: Asymptomatic
-Active infection: cough, fever, weight loss, night seats, hemoptysis, fatigue, decreased appetite, chest pain

Workup
-If high suspicion, most clinics don't workup but put a mask on and send to ER
-CXR: active infection (infiltrates in mid or lower fields, hilar adenopathy, cavitation, emyema) or previous (pulmonary nodules, apical fibrosis, Ghon lesion)
-TB skin test, AFB smear

Active TB drug regimens
-Initial for 2 months: isoniazid, rifampin, pyrazinamide, ethambutol
-Continuation for 4-7 months: isoniazid and rifampin

Latent TB drug regimens
-9 months of isoniazid or 4 months of rifampin

Monitoring
-Sputum smears and cultures throughout treatment
-Vision checks and color vision testing with ethambutol
-CMP, CBC, and bili

Atypical Mycobacterial Disease

-*Mycobacterium tuberculosis*
-*Mycobacterium leprae*
-Non-tuberculous mycobacteria: MAC, *Mycobacterium kansasii, Mycobacterium abscessus*

Signs & symptoms
-MAC: pulmonary disease with cough, fatigue, malaise, weakness, dyspnea, chest discomfort, occasional hemoptysis
-*M. kansasii* presents as lung disease that is very similar to TB
-Superficial lymphadenitis
-Disseminated disease in the immunocompromised
-Skin and soft tissue infection from direct inoculation

Workup
-Sputum or BAL culture

Management
-3 drug regimen for 12 months+

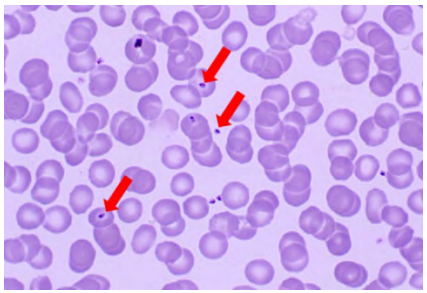
PARASITIC DISEASE

Malaria

-At greatest risk for severe malaria are young children and pregnant women
-Older children and adults typically develop partial immunity after repeated infection and are at low risk for severe disease

Prevention
-Mosquito bite prevention
-Chemoprophylaxis for travelers to endemic areas

Signs & symptoms
-*Plasmodium falciparum* incubation is 12-14 days, *P. vivax* and *P. ovale* can cause illness weeks or months after initial infection
-Fever, chills, malaise, fatigue
-Tachycardia and tachypnea
-Diaphoresis
-HA
-Cough
-Anorexia
-N/v/d
-Abdominal pain
-Diarrhea
-Arthralgias
-Myalgias
-Severe malaria (hyperparasitemia) → cerebral malaria, hypoglycemia, acidosis, renal impairment, noncardiogenic pulmonary edema, anemia, liver dysfunction

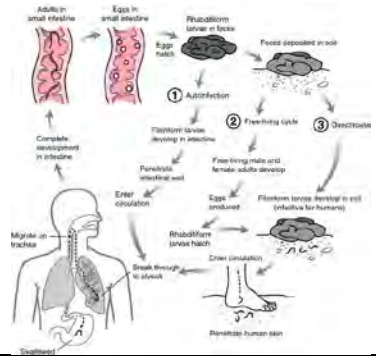
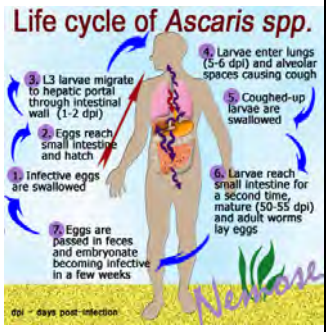
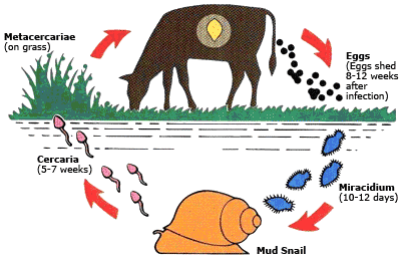


Differential
-Viral infection
-Meningitis
-Pneumonia
-Bacteremia
-Leptospirosis
-Typhus
-Enteric fever

Workup
-Light microscopy of peripheral smear is test of choice but can't detect low parasitemia
-Antigen or antibody tests

Management
-Uncomplicated malaria: chloroquine or quinine in drug-resistant areas

Helminth Infestations

Helminth	Information	Treatment	Helminth	Information	Treatment
Cestodes (Tapeworms)	-Flat hermaphroditic worms that can live in the human GI tract -*Taenia saginata* = beef tapeworm → mostly asymptomatic, may have nausea, anorexia, epigastric pain, or peripheral eosinophilia -*Diphyllobothrium lata* = fish tapeworm → megaloblastic anemia from B12 deficiency -*Hymenolepis nana* = rodent/arthropod tapeworm, associated with poor sanitation → asymptomatic infection or abdominal pain, diarrhea, anorexia, pruritus ani -Dx: eggs or proglottids in stool 	-Praziquantel	**Hookworms**	-*Ancylostoma duodenale* -*Necator americanus* -Penetrate skin directly -Spread by feces -S/s: acute GI sx, chronic nutritional deficiency, anemia -Dx: stool exam for eggs 	-Albendazole or mebendazole
Nematodes (Roundworms)	-*Ascaris lumbricoides* = tropical worm, associated with poor sanitation, migrate from small intestine to lungs then back to intestine → asymptomatic infection or transient pulmonary symptoms, bowel obstruction, biliary colic, acalculous cholecystitis, ascending cholangitis, obstructive jaundice, pancreatitis -Dx: stool microscopy 	-Albendazole or mebendazole -Prophylactic albendazole q 3-4 months in endemic areas	**Trematodes**	-Schistosomiasis: fresh water transmission → localized dermatitis, fever, intestinal, hepatic, urinary, neurologic, or pulmonary disease -*Clonorchis sinensis* = liver fluke, ingestion of infected fish → fever, anorexia, abdominal pain, myalgia, arthralgia, malaise, urticaria, bile duct obstruction, weight loss, diarrhea, pancreatitis, recurrent cholangitis, liver abscess, cholangiocarcinoma 	-Praziquantel
	-*Strongyloides stercoralis* → direct penetration of skin → waxing/waning GI symptoms, cutaneous or pulmonary symptoms, unexplained eosinophilia -Dx: at least 2 stool specimens for larvae -Occurs in the southern US	-Ivermectin			

Pinworms

-Parasite is *Enterobius vermicularis*, a roundworm -The most common parasitic intestinal infection -Transmission is fecal-oral or by inhalation	**Signs & symptoms** -Severe rectal itching -UTI -Vaginitis	**Workup** -Diagnosis is usually clinical -Tape test with microscopy	**Management** -Albendazole, with repetition in 2 weeks

Toxoplasmosis

-Agent is parasite *Toxoplasma gondii*
-Transmission is through ingestion of contaminated meat or produce, vertical, via blood transfusion or organ transplantation, or by handling contaminated animal feces (cats)

Signs & Symptoms
-Infections are generally asymptomatic
-Fevers, chills, sweats
-Cervical lymphadenopathy
-Congenital toxoplasmosis: chorioretinitis, intracranial calcifications, seizures, jaundice, HSM, lymphadenopathy, anemia, thrombocytopenia, abnormal CSF, hearing loss, intellectual disability, motor abnormalities, hydrocephalus

Differential
-Lymphoma
-Primary HIV
-Mono

Workup
-Toxo IgG antibodies will be present in pts previously exposed/immunized, while IgM indicates active infection

Management
-Usually not required in adults
-Congenital toxoplasmosis → treat with pyrimethamine + sulfadiazine for 1 year

Prognosis
-Infection will persist in latency for lifetime of infected host but can reactive in times of immunosuppression
-Treated infants remain at risk for long-term sequelae

Amebiasis

-*Entamoeba histolytica*

Signs and symptoms
-Intestinal amebiasis has a subacute onset of 1-3 weeks with mild diarrhea or dysentery, abdominal pain, bloody stools, can have fulminant colitis with bowel necrosis → perf and peritonitis or toxic megacolon
-Extraintestinal manifestations present as liver abscesses or pulmonary, cardiac, or brain involvement

Workup
-Serology or Ag testing along with parasitic stool exam

Management
-Treat with metronidazole, then paromomycin to kill the cysts

SPIROCHETAL DISEASE

Lyme Disease

-*Borrelia burgdorferi* with a tick vector
-Transmitted by *Ixodes spp* deer tick
-Transmission usually does not occur until 72 hours after attachment

Signs & symptoms
-Early localized disease: erythema migrans ~1 mo after exposure, nonspecific flulike viral syndrome
-Disseminated disease: acute neurologic or cardiac involvement weeks to months after tick bite, secondary skin lesions, malaise, fatigue, fever, HA, neck pain, myopericarditis, facial palsy
-Late disease: months to years after disease; arthritis, subtle encephalopathy or polyneuropathy, acrodermatitis chronicum atrophicans
-HA, fatigue, arthralgias may persist for months after treatment but don't represent active disease

Workup
-Dx can be clinical if erythema migrans is present or in endemic areas
-Serology for antibodies (warning, pts may be antibody negative for first several weeks of disease)
-Confirmatory Western blot

Management
-Treat with doxycycline, amoxicillin, or cefuroxime, for 10-21 days for erythema migrans, 14-21 days for facial nerve palsy, 28 days for meningitis or arthritis
-Ceftriaxone for CNS manifestations
-IV antibiotics needed for patients with cardiac symptoms or late neurologic disease
-No evidence for extended-course antibiotics for presumed chronic Lyme
-Can give single dose doxycycline for prophylaxis if attached tick is identified, estimated to be present > 36 hours, local tick *Borrelia* infection rate > 20%

Rocky Mountain Spotted Fever

-Most cases in NC, SC, Tennessee, Oklahoma, and Arkansas
-Agent in US is *Rickettsia rickettsii*
-Transmitted by *Dermacentor variabilis* (dog tick)

Signs & symptoms
-Chills, fever, headache, nausea, vomiting, myalgias, restlessness, insomnia, and irritability
-Rash begins as macules then progresses to maculopapules and petechiae, beginning on wrists and ankles, spreading to arms, legs, and trunk, also soles and palms
-Facial flushing, conjunctival injection, and hard palate lesions may occur
-Splenomegaly, hepatomegaly, jaundice, and myocarditis may occur
-Possible delirium
-Pneumonitis with respiratory failure

Workup
-PCR of skin lesions
-Serology will not be positive until 2nd week of disease

Treatment
-Doxycycline

Prognosis
-High mortality rate if untreated

Syphilis

-*Treponema pallidum*
-Most cases are MSM
-Can be transmitted vertically from mother to fetus

Screening
-Recommended for pregnant women at the first prenatal visit, with repeat at 28 weeks

Signs & symptoms
-Primary/acute infection lasts 5-6 weeks: contagious chancre, painless rubbery regional lymphadenopathy, followed by generalized lymphadenopathy
-Secondary infection 6 weeks-6 months after exposure (not all pts will develop this): fever, malaise, HA, arthralgias, bilateral papulosquamous rash on the palms and soles, alopecia, denuded tongue, condyloma lata
-Tertiary infection occurs in disease > 4 years' duration: end organ manifestations, CV symptoms, gummas, neurosyphilis
-Latent infection has no clinical manifestations but serology will be reactive
-Congenital syphilis of infant: stillbirth, prematurity, low birth weight, hydrops fetalis, large or pale placenta, inflamed umbilical cord, fever, HSM, lymphadenopathy, failure to thrive, edema, syphilitic rhinitis, maculopapular rash, condyloma lata, jaundice, anemia, thrombocytopenia, leukopenia or leukocytosis, pneumonia

Workup
-Remember that negative tests do not exclude a diagnosis of syphilis
-Darkfield microscopy of chancre sample
-LP for neurosyphilis
-Direct fluorescent antibody testing
-Serology: RPR (has a 3-6 week latency period)
-HIV test recommended as syphilis facilitates this infection

Management
-Mandatory reporting within 24 hours
-Penicillin G
-Recheck serologies at 6 and 12 months after treatment to look for fourfold reduction in titer

VIRAL DISEASE

Cytomegalovirus

-Transmission may be sexual, close contact, or blood and tissue exposure
-HIV patients and transplant patients are at increased risk of reactivation disease

Signs & symptoms
-Generally asymptomatic or nonspecific in immunocompetent host
-Can have CMV mononucleosis with fever (distinguish from EBV by absence of lymphadenopathy and pharyngitis)
-Rare associations with colitis, encephalitis, myocarditis
-Can have reactivation in critically ill patients

Workup
-CBC shows lymphocytosis
-PCR test
-Serologies
-Viral culture

Management
-Antivirals only for immunocompromised with severe manifestations

Epstein-Barr Virus

-Cause of infectious mononucleosis
-Persists as an asymptomatic latent infection for life in most adults

Signs & symptoms
-Majority of primary infections are asymptomatic
-Malaise and anorexia
-N/v
-HA
-Low-grade fever
-Pharyngitis and palatal petechiae
-Cervical lymphadenopathy
-Splenomegaly
-Young children/infants: OM, diarrhea, URI
-Morbiliform rash if ampicillin is used

Workup
-CBC
-Rapid monospot test
-Peripheral smear will show lymphocytosis with atypical lymphocytes

Management
-Supportive
-Pain control
-Steroids with emergent ENT referral if impending airway obstruction
-No contact sports for 3 weeks

Prognosis
-Associated with development of lymphoma and nasopharyngeal carcinoma
-Risk of splenic rupture

Human Immunodeficiency Virus (HIV)

-Progresses from primary infection with seroconversion → clinical latency → early symptomatic disease → AIDs
-Transmission is mostly heterosexual in developing countries while both MSM and heterosexual in the US
-Patients are most infectious during primary infection

Signs & symptoms
-Only lymphadenopathy during asymptomatic disease
-May have mononucleosis-like syndrome during primary infection
-Febrile illness
-Aseptic meningitis

Workup
-Serologies are + 3-7 weeks after infection
-Drug resistance testing
-Definition of AIDS is when CD4 count drops to < 200

Management
-Large debate about aggressive treatment of primary infection vs waiting until disease is symptomatic

Herpes Simplex

-Over 85% of adults will be + for HSV-1 and 20% will be + for HSV-2 by serology
-Precipitating factors: sunlight, dental surgery, cosmetic surgery, wind, trauma, fever, stress
-Transmission can be through asymptomatic shedding
-First outbreak will be the worst and can last up to 21 days

Signs & symptoms
-Prodrome of burning or neuralgia
-Swollen regional lymph nodes
-Pain with urination

Differential
-Chancroid
-Syphilis
-Pyoderma
-Trauma

Workup
-Viral culture is gold standard
-Serology is questionable, as not all + cultures will have + serology and vice versa, and many are asymptomatically +

Complications
-Eczema herpeticum: severe infection in the immunocompromised
-Herpetic whitlow: fingernail or hand infection
-Herpes gladiatorum: infection anywhere not covered by underwear
-Pyoderma
-Proctitis, esophagitis
-Keratitis
-Encephalitis

Management
-Acyclovir
-Valacyclovir
-Famciclovir
-Topical corticosteroid for orolabial herpes
-7-10 days for first outbreak and 3-5 days for subsequent outbreaks
-Suppressive therapy if needed

Human Papilloma Virus

-Small DNA viruses that are sexually or contact transmitted
-Sexually transmitted strains are associated with squamous neoplasia of the anogenital region and oropharynx

Risk Factors
-Multiple sex partners
-Young age at first sexual activity
-H/o STDs
-Multiparity
-Immunosuppresion
-Uncircumcised male partner

Prevention
-HPV vaccines cover most of the sexually transmitted subtypes

Signs & Symptoms
-Condyloma acuminata: caused by HPV type 6 and 11, can also be 16 or 18
-Cervical dysplasia and oropharyngeal lesions: usually HPV 16 and 18
-Common cutaneous warts: HPV types 1, 2, 4
-Anal carcinoma in MSM

Workup
-Pap cytology
-Colposcopy

Management
-Most sexually transmitted HPV infections will self-resolve
-Follow resolution of infection with sequential Paps

Influenza

Influenza vs. common cold
-Flu = abrupt onset (sx worsen over 3-6 hours) with fever > 101.5, severe myalgias, headache, malaise, painful dry cough, sore throat, rhinitis, secondary *Staph aureus* pneumonia may follow
-Cold = slow, insidious onset, usually no headache or chills, sore throat, stuffy nose, sneezing, mild aches

Prevention
-Inactive vaccine starting at 6 months (first-time vaccination in kids under 9 requires 2 doses)
-Live vaccine if 2-49 and healthy (warning: viral shedding)

Workup
-Nasopharyngeal swab (may be done just for epidemiologic purposes)

Treatment
-Antipyretic/analgesic
-Albuterol neb
-Ipratropium inhaler (Atrovent) for secretions
-Consider steroids
-Consider antivirals (oseltamivir, zanamivir) for influenza A or B only with hospitalization, severe or progressive disease, age under 2 or over 65, and for outbreak control in institutions or health care workers, AND MUST BE within 48 hours of start of symptoms to help at all

Mumps

-Agent is paramyxovirus
-Prevent with MMR vaccine (indicated to prevent severe pain)

Signs & symptoms
-Parotitis
-Stenson's duct inflammation with yellow discharge
-Orchitis 7-10 days later, with abrupt fever, testicular swelling and tenderness
-Possible CNS involvement

Management
-Scrotal support and ice packs
-NSAIDs

Prognosis
-Rare chance of sterility with orchitis

Varicella

Signs & symptoms
-Intensely pruritic lesions that come in crops over 3-4 days → see lesions in all different stages
-Acute neuritis with rash most commonly presenting in the thoracic and lumbar dermatomes
-Disseminated lesions in immunocompromised host

Management
-Oral acyclovir for adults with uncomplicated varicella
-IV acyclovir for immunosuppressed host or with disseminated disease such as pneumonia or encephalitis
-Acetaminophen for fever
-Antihistamines for pruritus

Prognosis
-Can be fatal in adolescents and adults
-Will be contagious for 1 week

Varicella Zoster (Herpes Zoster or Shingles)

-Primary infection is varicella, secondary is zoster

Prevention
-Zostavax vaccine indicated after age 60

Treatment
-High dose acyclovir within 72 hours of onset
-Prednisone if over age 50
-Analgesics

Complications
-Post-herpetic neuralgia
-Ophthalmic complications
-Hemiplegia

SURGICAL INFECTIOUS DISEASE

Postoperative Fever

Etiologies
-Not always infectious!
-Wind: atelectasis, pneumonia
-Wound infection: usually occur several days to 1 week after operation
-Water: UTI
-Walking: DVT or thrombophlebitis
-Wonder drugs: medication-induced fever (heparin or abx)
-Women: postpartum fever, endometritis
-Blood transfusion

Prevention
-Avoid atelectasis: early ambulation, incentive spirometry
-Avoid pneumonia: use humidified O2
-DVT prophylaxis
-Judicious use of Foley catheters with d/c ASAP
-Clear instructions for home care of wound sites

Workup
-Fever in patient < 2 days out from surgery who is otherwise doing well is usually self-limiting and does not require workup
-CBC
-CXR: may lag behind PE findings
-Consider LE US for DVT
-Blood cultures?

Management
-Change out infected or thrombosed lines
-Wound infection: open infected area, start antibiotics
-D/c unnecessary meds, NGT, catheters
-Treat fever with acetaminophen
-Broad spectrum antibiotics only for hemodynamically unstable pts while source is being identified

Surgical Site Infection

-SSI is defined as infection related to the operative procedure occurring at or near the surgical incision within 30 days of an operative procedure or within 1 year of an implant
-Occur in 2-5% of patients undergoing surgery
-Nonteaching hospitals have lower rates than teaching hospitals
-Most common source is direct inoculation of pt's endogenous flora at the time of surgery
-Incidence of resistant pathogens cultured from SSIs is increasing: MRSA, MRSE, VRE
-Incidence of fungi cultured from SSIs is increasing: Candida albicans

Risk Factors
-Obesity
-Smoking
-DM
-Systemic corticosteroids
-Immunosuppression
-Malnutrition
-Preoperative nasal carriage with Staph aureus
-Presence of remote focus of infection
-Long duration of preoperative hospitalization
-Preoperative severity of pt illness
→ Can predict pt risk of SSI with National Healthcare Safety Network risk index

Prevention
-Preop showering with antimicrobial soaps
-Preop prepping of operative site with antiseptics (chlorhexidine superior to iodine)
-Washing and gloving of surgeon's hands (alcohol rubs may be as effective as traditional soap scrubbing)
-Use of sterile drapes
-Use of gowns and masks by OR personnel
-Good surgical technique: gentle traction, effective hemostasis, removal of devitalized tissue, obliteration of dead space, irrigation with saline, use of fine nonabsorbable monofilament suture, judicious use of closed suction drains, wound closure without tension
-Antibiotic prophylaxis: should be administered within 60 min of first incision; may need to be repeated more than once depending on length of surgery
-Hair removal: may increase risk of surgical site infection, must use clippers or depilatories if removing hair vs razor
-Tight glucose control in diabetic pts
-Perioperative warming (bear huggers) to prevent hypothermia, warmed IVF, hats and booties
-Minimally invasive and laparoscopic procedures associated with ↓ risk of SSI

Components of Optimal Wound Healing
-Well-vascularized wound bed
-Wound free of devitalized tissue
-Wound clear of infection
-Moist wound

Management of SSI
-Opening, exploration, draining, irrigation of wound
-Sharp surgical debridement of devitalized tissue
-Wound can be closed or allowed to heal by secondary intention once granulation tissue is apparent

ADDITIONAL EMERGENCY MEDICINE TOPICS

Near Drowning

Signs & Symptoms
-Pulse detection may be difficult due to hypothermia

Management
-Ventilation is most important
-If pt does not respond to 2 rescue breaths, CPR should begin
-Intubation if needed
-Rewarming techniques

Prognosis
-Neuroprotective effects of hypothermia can result in complete recovery of patients despite cardiac arrest and prolonged resuscitation of several hours

Carbon Monoxide Poisoning

Causes
-Poorly functioning heating systems
-Improperly vented kerosene heaters, charcoal grills, camping stoves, gasoline powered generators, or running cars
-CO outcompetes with O2 binding Hb

Signs & Symptoms
-Headache
-Malaise
-Nausea and dizziness
-AMS, seizures, or coma
-Cardiac ischemia

Workup
-ABG cooximetry (pO2 will look normal)

Management
-100% O2 via nonrebreather
-?Hyperbaric therapy for severe intoxication

Electrical Injury

Causes
-Lightning
-AC current: more dangerous
-DC current: travels in one direction so individual is thrown off of electrical source

Signs & Symptoms
-Manifestations range from mild superficial burns to severe multiorgan dysfunction and death
-Cardiac: arrhythmias
-Skin: superficial to full-thickness burns
-Msk: periosteal burns, destruction of bone matrix, osteonecrosis, rhabdo, compartment syndrome
-Renal: AKI from rhabdo
-Neuro: coma, cognitive deficits, autonomic dysfunction
-Vascular: small vein coagulation, major artery thrombosis
-TM rupture
-Entry and exit sites for DC current exposure

Management
-Trauma evaluation
-Telemetry
-Monitoring for compartment syndrome, rhabdo, and AKI

Anaphylaxis

-Typically an IgE-mediated reaction
-Common triggers are foods, insect stings, and medications

Signs & Symptoms
-Cardiopulmonary arrest: occurs in 5 minutes with iatrogenic anaphylaxis, 15 minutes in stinging insect anaphylaxis, and 30 minutes in food-induced anaphylaxis
-Generalized urticaria
-Angioedema
-Flushing
-Pruritus

Workup
-Transiently elevated plasma histamine or total tryptase

Anaphylaxis is highly likely when any ONE of the following three criteria is fulfilled:
1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (eg, generalized hives, pruritus or flushing, swollen lips-tongue-uvula)
AND AT LEAST ONE OF THE FOLLOWING:
A. Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced PEF in older children and adults, hypoxemia)
B. Reduced BP* or associated symptoms of end-organ dysfunction (eg, hypotonia, collapse, syncope, incontinence)
2. TWO OR MORE OF THE FOLLOWING that occur rapidly *after exposure to a LIKELY allergen for that patient* (minutes to several hours):
A. Involvement of the skin-mucosal tissue (eg, generalized hives, itch-flush, swollen lips-tongue-uvula)
B. Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced PEF in older children and adults, hypoxemia)
C. Reduced BP* or associated symptoms (eg, hypotonia, collapse, syncope, incontinence)
D. Persistent gastrointestinal symptoms (eg, crampy abdominal pain, vomiting)
3. Reduced BP* *after exposure to a KNOWN allergen for that patient* (minutes to several hours):
A. Infants and children: low systolic BP (age specific)* or greater than 30 percent decrease in systolic BP
B. Adults: systolic BP of less than 90 mmHg or greater than 30 percent decrease from that person's baseline

Management
-Epinephrine
-Immediate intubation if signs of impending airway obstruction from angioedema
-Oxygen via face mask
-Rapid NS infusion of 1-2 L
-Also consider albuterol, diphenhydramine, ranitidine, and/or methylprednisolone
-Refractory symptoms → epinephrine infusion, other pressors, glucagon
-Referral to allergist/immunologist to confirm diagnosis and triggers