

Foundations of Emergency Medicine
 Comprehensive Board Review

Cardiology	
Bizz	Buzz
What underlying pathologic process distinguishes myocardial infarction from angina/unstable angina?	Atherosclerotic plaque rupture → exposed endothelium → clot attaches → reduced blood flow; if cell death occurs (usually due to complete vascular obstruction) then positive trop and MI; if no cell death occurs then negative trop and angina/unstable angina
What is the difference between transmural and non-transmural infarction?	<u>Transmural</u> : usually STEMI, large vessel affected, benefit from thrombolytics/PCI; <u>Non-Transmural</u> : usually NSTEMI, smaller subendocardial artery, may benefit from PCI but no thrombolytics
What defines Unstable Angina?	Stable Angina + pain at rest, new pain, increasing pain severity, hemodynamic changes with pain
Acute chest pain at night, EKG with STEMI, all symptoms and EKG changes resolve with nitro?	Prinzmetal's Angina (coronary spasm, most do not have CAD; treat with CCBs)
What are early to late EKG changes with ACS?	Hyperacute T's and Giant R (very early and transient), STE, STD (ischemia or reciprocal), Q waves (1 square wide, 1/3 height QRS), TWI
Biphasic T-wave in V2/V3	Wellen's Syndrome: biphasic (type A) or deeply inverted, symmetric (type B) TW in septal leads = early signal of proximal LAD lesion
Chest Pain with STE V1-V4 with STD II, III, aVL	Anterior MI 2/2 LAD occlusion, may affect large territory of LV, septum and conduction system (high grade blocks, wide complex bradycardias), commonly have shock, possible ruptures
Chest Pain with STE I, aVL, V5, V6 with STD V1	Lateral MI 2/2 LAD vs LCx occlusion, may affect LV
Chest Pain with STE II, III, aVF with STD V1-V4	Inferior MI 2/2 occlusion of PDA (RCA > LCx), may affect AV node (usually transient narrow complex bradycardias), may cause papillary muscle rupture
Chest Pain with STE III > II and V1 > V2	Right Ventricular MI, should get R-sided leads (STE in V4R, V5R), 2/2 proximal RCA lesion, associated with Inferior MI
Chest Pain with STD V1-3	Posterior MI, get posterior leads to dx (req only 0.5 mm elevation for STEMI dx), 2/2 occlusion of Posterior Descending (RCA > L circ)
What meets STEMI criteria for leads V2-V3 versus all other leads?	V2-V3: ≥2mm in MEN ≥ 40yrs, ≥2.5mm in MEN < 40yrs, or ≥ 1.5mm in WOMEN; All other leads: STE at the J-point of ≥ 1mm in two contiguous lead
What distinguishes Type I-Type V MI?	Type I: MI caused by acute atherothrombotic CAD, usually due to plaque rupture or erosion; Type II: MI 2/2 mismatch of oxygen supply and demand; Type III: typical MI presentation but death before biomarkers obtained; Type IV: MI 2/2 PCI; Type V: MI 2/2 CABG
How can you detect MI in patients with paced rhythm or old LBBB	Sgarbossa Criteria: STE >1mm with concordant (same direction) QRS, concordant STD >1mm V1-V3, STE >5mm with discordant (opposite direction) QRS (modified Sgarbossa changes this last rule to discordant STE >25% preceding S wave)

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What is unique about the management of Inferior MIs?	With Inferior MI, always consider RV involvement and get right-sided EKG leads
What is unique about the management MI with right-ventricular involvement?	They are preload dependent and will become very hypotensive with nitroglycerin - avoid this, give IVF for hypotension
What are potential early complications (<24hr) of MI?	arrhythmia, shock 2/2 pump failure or valve dysfunction
What are potential late complications (>24hr) of MI?	Thromboembolism, myocardial rupture, valve rupture, CHF, pericarditis
Pleuritic chest pain 4wks after MI?	Dressler's syndrome: autoimmune pericarditis, typically 2-6wks s/p MI. Tx it with NSAIDs like any other pericarditis
What artery typically supplies the SA node and AV node?	SA- RCA 60%, LCx 40%; AV- RCA 90%, LCx 10%; concern for bradycardias if Inferior MI
Cardiac Tamponade after MI	Myocardial wall rupture, give IVF and dispo to OR
What EKG finding is classic in Cardiac Tamponade?	Electrical Alternans
Shock + new murmur after recent MI	Papillary muscle rupture, Rx- reduce afterload and dispo to OR; same treatment if septal wall rupture
What potential treatments for AMI have been shown to reduce mortality?	Defibrillation for VF/VT (30% mortality reduction), Aspirin (25% mortality reduction)
What is the only contraindication to aspirin in ACS?	True aspirin allergy (anaphylaxis)
What is better for treatment of STEMI, thrombolytics or PCI?	PCI is better. Thrombolytics should only be considered if PCI is not available at center within 90min or after transfer within 120min
What EKG changes are included under indications for thrombolysis?	STEMI (STE >2mm for men, >1.5mm for women in V2-3, STE >1mm in 2+ other leads), STD V1-3 (posterior MI), old LBBB + Sgarbossa
What are absolute contraindications for thrombolysis?	Any previous brain bleed or known mass, ischemic stroke or closed head injury within 3mo, known bleeding disorder, current active bleeding, major surgery in the last 2 months, BP > 180/110 *after treatment, suspected aortic dissection
What are concerning complications of thrombolysis and how often do they occur?	Intracranial hemorrhage (1/70 to 1/100, >50% mortality), major bleeding (e.g. GI bleed) in 5%
What EKG changes may occur with reperfusion?	Accelerated idioventricular rhythm, NSVT, PVCs; these should be transient, are overall benign and do not require additional treatment
What is the appropriate treatment of ST elevation after cocaine use?	First treat with benzos, aspirin, nitrates, calcium channel blockers or alpha blockers (e.g. phentolamine) for HTN, thrombolysis only if ST does not return to baseline after these treatments
What is the appropriate treatment for HTN after cocaine use?	Benzos, CCBs or phentolamine; NO Beta Blockers (may theoretically lead to unopposed alpha stimulation and worsened HTN)
What are key risk factors for Infective Endocarditis?	Diseased valves, artificial valves, IV drug use, dental extractions

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What heart valve and what organism is most common in Infective Endocarditis?	Left sided (mitral most common valve affected overall) > right sided (tricuspid > pulmonary); Staph aureus is most common pathogen but viridans strep if s/p tooth extraction); Tricuspid is most common with IV drug use (Staph aureus)
Describe the classic physical exam findings in Infective Endocarditis?	Osler Nodes (painful nodules on fingertips), Janeway Lesions (nontender hemorrhagic lesions on palms/soles), Roth Spots (retinal hemorrhages), Splinter hemorrhages (linear on nails), Petechiae, New Murmur
What is the appropriate management and treatment of a patient with suspected Infective Endocarditis?	Blood cultures x 3 (different locations), Echo (transesophageal best), broad spectrum antibiotics to cover staph/strep/gram negatives (Vanco + PCN + Gent)
When should a patient receive antibiotic prophylaxis for Infective Endocarditis prior to a procedure?	<u>High-risk procedures: dental or invasive respiratory</u> ; GI/GU procedures don't need abx. <u>High risk pts</u> : Artificial or damaged valves, ANY congenital heart dz hx; Rx: Amoxicillin (dental procedures)
What left sided murmurs are systolic?	Aortic stenosis and mitral regurg
What left sided murmurs are diastolic?	Aortic insufficiency and mitral stenosis
Syncope + systolic murmur radiating to neck	Aortic Stenosis, syncope is poor prognostic sign, requires surgical consult; causes L heart strain. Patients generally present with angina, then syncope and then heart failure.
Chest pain with new diastolic murmur	Aortic dissection causing aortic insufficiency, may have "water-hammer" pulse
Pregnant women with sudden cardiovascular collapse during labor	Mitral Stenosis, high output during labor causes LA enlargement, AFib and arrhythmia
Treatment for decompensating patient with diastolic murmur, opening snap	Cardiovert, suspect mitral stenosis and AFib
MI followed by hypotension and new murmur	Mitral Regurgitation 2/2 ruptured cordae tendineae/papillary muscle; Tx decrease afterload and cardiac surgery
What are the most likely causes and signs/symptoms of Right and Left sided Heart Failure?	Right: MC 2/2 L-sided failure, lung disease, PE, symptoms include JVD, peripheral edema, hepatic congestion; Left: 2/2 ischemia, valves, HTN, symptoms include SOB, orthopnea, PND, potential R-sided failure
What distinguishes systolic vs diastolic heart failure?	Systolic: failed forward flow; Diastolic: failed filling
What is the general approach for treatment of decompensated heart failure?	Decrease LVEDV to improve SV and CO (Starling curve); Reduce preload with nitroglycerin and diuretics (Lasix; caution if diastolic failure), BiPAP; consider afterload reduction (nitroglycerin); give inotropes for shock
What are classic causes of high output cardiac failure?	Hyperthyroidism, Beriberi, AV fistula, Paget's dz, severe anemia, pregnancy

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What are classic CXR findings with heart failure?	Big heart, fluffy infiltrates, Kerly B lines, blunted CVA (effusion)
What does BiPAP help patients with heart failure?	Decreases work of breathing, decreases preload (positive pressure increases intrathoracic pressure and decreases venous return)
What is the most common cause of acute Right Heart Failure (Cor Pulmonale)?	Pulmonary Embolism; Left heart failure is most common chronic cause
Dx and Tx of Dilated Cardiomyopathy	H/o HTN or ischemia, big heart on CXR, low EF on echo; Tx underlying cause/CHF/dysrhythmias, anticoagulate if mural thrombus, transplant if severe
Dx and Tx of Restrictive Cardiomyopathy	2/2 fibrosis, radiation, TB causing stiffness; normal heart on CXR, poor filling on echo; Tx underlying cause/CHF/dysrhythmias
Dx and Tx of Hypertrophic Cardiomyopathy	Classically with septal hypertrophy, severe symptoms/syncope with exercise, EKG with LVH (tall QRS, needle-like Q waves); Tx avoid exertion, beta blockers (slow rate and ↑ ventricular filling), AICD for ventricular arrhythmias, surgical ablation
Describe the typical murmur of Hypertrophic Obstructive Cardiomyopathy (HOCM)	Harsh, systolic crescendo-decrescendo murmur; ↑ with Valsalva, standing up (↓ LV blood volume); ↓ squatting, trendelenberg (↑ LV blood volume)
What is the typical time frame for developing peripartum cardiomyopathy?	Last trimester to 5 months postpartum
What are the classic clinical clues for diagnosis of Pericarditis?	Triad: fever + dyspnea + chest pain (pleuritic chest pain radiating to neck, worse with laying flat), recent viral syndrome; exam- intermittent friction rub, clear lungs; may have evidence of pericardial effusion/tamponade; unlikely to have trop leak
What are classic EKG changes with Pericarditis?	PR depression (most specific), PR segment elevation (aVR), diffuse STE, TW flattening followed by TW inversion
What is the appropriate treatment for Pericarditis?	Must get Echo to r/o pericardial effusion; NSAIDs, ± colchicine (↓ recurrent pericarditis)
What are the classic clinical clues for diagnosis of Myocarditis?	Dyspnea = most common sx, chest pain, viral prodrome, CHF sx (wet lungs, edema), arrhythmias, *unresolving sinus tachycardia*; usually (+) troponin; Echo usually with global hypokinesis and dilated chambers
What are classic EKG changes with Myocarditis?	Sinus tachycardia, non-specific STE; can be similar to pericarditis
What is the appropriate treatment for Myocarditis?	Supportive care, avoid early NSAIDs or steroids, ICU admit if severe/CHF
What are the most common causes of Myocarditis?	Idiopathic (most common overall), Parvovirus (most common viral cause), Chagas disease (most common worldwide)
JVD, decreased heart sounds, and hypotension	Beck's Triad of Pericardial Tamponade; Rx- IVF to increase preload, pericardiocentesis ± surgery
What are the clinical features of hypertensive emergency?	<u>CNS</u> : dizziness, n/v, confusion, weakness, encephalopathy, ICH, SAH, CVA; <u>Eyes</u> : ocular hemorrhage, papilledema, vision loss; <u>Heart</u> : ACS, Ao dissection, shock; <u>Kidneys</u> : hematuria, proteinuria, acute renal failure

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Review the definitions of Asymptomatic HTN, HTN Urgency and HTN Emergency	<u>Asymptomatic HTN</u> : BP >140/90 without apparent symptoms; <u>HTN Urgency</u> : BP >180/110 but WITHOUT signs of end organ dysfunction; <u>HTN Emergency</u> : BP >180/110 WITH signs of end organ dysfunction
Review appropriate ED management of Asymptomatic HTN, HTN Urgency and HTN Emergency	<u>Asymptomatic</u> : no workup needed (Cr = only screening test shown to change mgmt for ITE), no treatment needed, restart home meds (if any), refer to PMD; <u>HTN Urgency</u> : rule out end organ dysfunction (based on sx), gradually lower BP over 1-2d with PO meds (restart home or HCTZ, BB/CCB); <u>HTN Emergency</u> : if end organ dysfunction then goal 20-30% BP reduction (Nicardipine, Esmolol, Labetalol, Nitroglycerin, etc. based on sx)
What are the JNC 8 recommendations for BP meds in asymptomatic HTN	NOT a requirement to start in ED, but recommended (esp. for boards). <u>Non-African-American</u> : Thiazide, CCB, ACEI, ARB; <u>African-American</u> : Thiazide, CCB; <u>CKD</u> : ACEI or ARB
What medications are best to lower BP in patients with severe HTN and the following: Encephalopathy, Aortic Dissection, Cocaine use, pregnancy, ACS/CHF	<u>Encephalopathy</u> : Nicardipine; <u>Aortic Dissection</u> : Beta blockers FIRST (Esmolol or labetalol to reduce rate & shear stress), ± Nitroprusside AFTER rate reduction); <u>Cocaine use</u> : benzos and phentolamine (no beta blockers); <u>Pregnancy</u> : IV Mg, Hydralazine, Labetalol (preeclampsia); <u>ACS/CHF</u> : Nitroglycerin
How should HTN be managed for Ischemic and Hemorrhagic Strokes?	<u>Ischemic Strokes</u> : permissive HTN to protect penumbra up to 220/120 BUT reduce to <185/110 if considering tPA; <u>Hemorrhagic</u> : varied guidelines for BP control (good goal SBP 140 or MAP < 130), use CCBs to prevent vasospasm (PO Nimodipine = classic for boards)
What are potential non-cardiac causes of syncope?	Aortic (Aortic dissection, ruptured AAA), neurologic (CVA, SAH, sz), bleeds (RP bleed, ruptured ectopic or AAA, GI bleed), orthostatic (meds), reflex (vasovagal)
What is the differential for potential life-threatening cardiac causes of syncope?	Dysrhythmias (VT), structural abnormalities (HOVM, critical aortic stenosis), electrical abnormalities (Brugada, WPW, Prolonged QT, arrhythmogenic right ventricular dysplasia), others (PE, MI); Screen all EKGs for these findings
What minimum workup should be completed on young female patients with syncope	Pregnancy test (ruptured ectopic may only present with syncope)
How is near syncope treated differently than syncope?	They aren't, they have the same causes and should be worked up the same way
What is the overall most common cause of syncope?	Idiopathic (40-50%) > Vasovagal (~20%)
What factors make someone with syncope "high risk" requiring admission and significant workup?	San Francisco Syncope Rule: CHES - Admit patients with CHF , Hct < 30, EKG that is abnormal, Shortness of Breath or Systolic BP < 90, as they are high risk for serious outcomes. Other high risk features: family history of sudden death, syncope with exertion, structural heart disease.
What EKG changes may be seen in a patient with Wolff-Parkinson-White?	Slurred upstroke of QRS (delta wave), wide QRS (QRS > 120ms), short PR interval (most common, PR < 120ms)

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What EKG changes may be seen in a patient with Brugada Syndrome?	Pseudo-RBBB, STE V1-3 (types: coved/downsloping STE followed by TWI, or "saddle-back" STE)
What EKG changes may be seen in a patient with Long QT?	End of T wave > 1/2 R to R interval
What EKG changes may be seen in a patient with Arrhythmogenic Right Ventricular Dysplasia?	Epsilon wave (postive notch at end of QRS)
What is the underlying pathology in Arrhythmogenic Right Ventricular Displasia?	Genetic abnormality, autosomal dominant, causes fibro-fatty infiltrate in RV (best seen on Cardiac MRI) that causes arrhythmogenic focus in RV (30% with epsilon wave) and predisposes for fatal arrhythmias; Rx- antiarrhythmics, AICD
What EKG changes may be seen in a patient with Hypertrophic Obstructive Cardiomyopathy (HOCM)?	LVH, LARGE voltages (tall QRS), deep/narrow Q waves ("needle-like") in lateral (I, aVL, V5-6) and inferior (II, III, aVF) leads
What patients are higher risk for Aortic Dissection?	Prolonged HTN, connective tissue disease (e.g. Marfan syndrome); also pregnancy, congenital heart disease, trauma
What are the classic clinical clues for diagnosis of Aortic Dissection?	Acute onset severe pain radiating in direction of propogation (neck/arms vs back/abdomen); chest pain (most common) + something else = TAD; HTN = most common risk factor AND exam finding; can be associated with any sx linked to sequelae of dissection including new murmur, MI, CHF, renal insufficiency, mesenteric ischemia, new neuro deficits. Note BP can be high, low or normal
What is the most common XR finding in acute aortic dissection?	Mediastinal widening
What is the appropriate management and treatment of a patient with suspected Aortic Dissection?	HR and BP control to decrease shear stress (Esmolol followed by Nitroprusside, or Labetalol), control pain, T&C x 10-15; if unstable consult cards/thoracic surgery with dispo to OR, consider bedside echo; if stable get CTA aortogram; **NEVER send unstable patient to CT**
What is the difference between Type A and Type B aortic dissections?	<u>Type A</u> : Ascending Aorta, managed surgically; <u>Type B</u> : Descending Aorta, usually managed medically
What patients are higher risk for Ruptured AAA?	Disease of arteriosclerosis (all the same risk factors): age > 60, males, family history, HTN, HL, smoking, CAD, connective tissue disease
Most common presentation for unruptured AAA?	Asymptomatic
What are the classic clinical clues for diagnosis of Ruptured AAA?	Cooper's triad: sudden abdominal/flank pain + pulsatile abdominal mass + hypotension; Others: peripheral ischemia, syncope, sudden death
What size AAA is higher risk for rupture?	>3cm is pathological, >5.0cm is high risk and requires surgery

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What is the appropriate management and treatment of a patient with suspected Ruptured AAA?	Bedside US to eval for AAA, possible free fluid (though bleeding may be retroperitoneal), T&C x 10-15, emergent vascular surgery consult with dispo to OR ASAP; **DO NOT send unstable patient to CT scan**
History of repaired AAA with massive GI bleed	Aortoenteric fistula
Dx and Tx of Acute Arterial Occlusion	Look for medical problems related to thromboemboli (Afib = most common embolic cause, MI, or endocarditis); <u>6 P's</u> : pain (out of proportion to exam), pallor, pulselessness, poikilothermia, paresthesias, or paralysis; Dx: CT angio vs duplex US; emergent vascular surgery consultation; Rx: heparin vs thrombolysis vs embolectomy
How sensitive is Homan's sign for DVT?	Homans (pain in the calf on dorsiflexion of ankle while the knee is fully extended) has 50% sensitivity
What are risk factors for DVT?	Classic triad (Virchow's): stasis, + hypercoagulability + endothelial damage; <u>acquired (persistent)</u> : age, active cancer, hx DVT/PE, antiphospholipid Ab; <u>acquired (transient)</u> : recent surgery or major trauma, pregnancy, OCPs/HRT, paralysis/immobilized (3d within last 4wks); <u>inherited</u> - ATIII deficiency, Protein C/S deficiency, Factor V Leiden; exam- tender vein or distended superficial veins, unilateral calf swelling >3cm, unilateral pitting edema
What is the appropriate workup for patients with clinical symptoms and low versus high risk of DVT?	<u>Low risk</u> : d-dimer ok; <u>Moderate-high risk</u> : d-dimer & duplex US (alt CT venography); if high risk may require serial dopplers & whole leg US
What is the appropriate management of isolated calf DVT?	Anticoagulation not required unless within 5cm of popliteal vein (ASA otherwise); repeat ultrasound (in 2-5d) to r/o propagation. This is only true for low risk pts w/ transient risk factors (such as recent travel).
what are considered distal veins in the evaluation of a DVT?	Anything in the calf - infrapopliteal veins: posterior tibial, peroneal, anterior tibial. If a pt is low risk (e.g. NOT obese or prothrombic for any reason) and has a transient risk factor (e.g. recent travel, prolonged immobilization, ect ...) distal DVTs can be monitored w/ repeat US and no anticoagulation.
Review the definitions and EKG changes of 1st, 2nd, and 3rd degree AV Block	<u>1st</u> : PR >200 and otherwise normal; <u>2nd</u> : Mobitz Type I (Wenckebach-increasing PR interval then dropped beat), Mobitz Type II (stable long PR, then sudden, non-conducted PR → dropped beat); <u>3rd</u> : P waves entirely dissociated from QRS
What types of heart block typically require pacemaker placement?	2nd degree (Mobitz Type II) and 3rd degree
What is the appropriate treatment for unstable bradycardia?	Atropine (may help if narrow QRS); Others: Dopamine, Epinephrine, Isoproterenol; <u>Unstable</u> : transcutaneous pacing (± transvenous pacing); Definitive Rx: permanent pacemaker
Review the general steps for transcutaneous pacing	Sedate/pain control if able, place pacer pads, turn on pacing function, set rate 70-80, increase voltage until capture noted

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Review the general steps for transvenous pacing	Place IJ or SC cortis, introduce catheter and inflate balloon, set pacer at 80 bpm and voltage to 20 mA, advance catheter to RV (will show LBBB pattern), deflate balloon, secure and decrease voltage to lowest setting with continued capture. **Preferred sites: RIJ and left subclavian**
Treatment for arrhythmia and unstable patient	Electricity
DDX for narrow complex tachyarrhythmia WITH P waves; Treatment for stable patient	Sinus tach, AFlutter, MAT; Treat underlying problem If present, and control rate with AV nodal blockers (and shock if unstable!)
DDX for narrow complex tachyarrhythmia WITHOUT P waves; Treatment for stable patient	AF, SVT, AVNRT, orthodromic AVRT; Treat with Adenosine or AV nodal blockers (and shock if instable!)
Differential for wide complex tachyarrhythmia without P waves; Treatment for stable patient	VT (VF possible but likely unstable pt), SVT with BBB, Antidromic AVRT (e.g. WPW); Rx: Procainamide or Amiodarone; AVOID AV nodal blockers; Unstable- shock!
EKG with chaotic P waves, irregularly irregular rhythm	Atrial Fibrillation; Rx- CCB or BB
EKG with "sawtooth" symmetrical P waves, regularly irregular rhythm	Atrial Flutter; Rx- CCB or BB
EKG with multiple types of P waves, irregularly irregular rhythm	Multifocal Atrial Tachycardia; 2/2 pulmonary disease (COPD = most common cause); Rx- CCB or BB
EKG with regular tachycardia and narrow QRS	AV nodal reentrant tachycardia (AVNRT) or Orthodromic Atrioventricular Reentrant Tachycardia (AVRT); Rx- Adenosine (first line), consider BB or CCB, shock (cardioversion)
EKG with regular tachycardia and wide QRS	Antidromic Atrioventricular Reentrant Tachycardia (AVRT) OR Ventricular Tachycardia; Rx- Procainamide vs Amiodarone, consider Mag (shock if unstable); Avoid AV nodal blockers in these patients
What is the difference between Orthodromic and Antidromic Atrioventricular Reentrant Tachycardia (AVRT)?	Reentry circuit with accessory pathway (WPW- Bundle of Kent); <u>Orthodromic</u> travels anterograde down AV node and back up accessory pathway resulting in regular and narrow QRS complex (looks like SVT); <u>Antidromic</u> travels anterograde down accessory pathway and back up AV node (retrograde) resulting in regular and wide QRS complex (looks like VT). Tx antidromic w. Procainamide.
What is the appropriate management of a patient with tachydysrhythmia and suspected WPW?	Procainamide or Amiodarone if stable; Shock if unstable; AVOID AV nodal blockers. If any signs of WPW (delta wave, short PR) or borderline wide QRS, presume WPW and avoid AVNBs
EKG with multiple chaotic ventricular foci that are wide and irregular	Ventricular Fibrillation; shock (defibrillate)

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What BP measurements define Stage I and Stage II HTN?	<u>Stage I HTN</u> : systolic 140-159 mmHg or diastolic 90-99 mmHg; <u>Stage II HTN</u> : systolic >160 mmHg or diastolic >100 mmHg
What EKG findings suggest Ventricular Aneurysm?	Persistent STE > 2wks after known MI (and lack of reciprocal changes), most often in precordial leads (V3-5); Others: Q or QS waves, T waves small relative to QRS, reciprocal changes absent
What is "Holiday Heart Syndrome" and how should it be treated?	Typically atrial arrhythmia (Afib) after excessive ETOH intake; Rx- observation (if stable), typically self-resolves within 48hr
What EKG findings confirm a diagnosis of Ventricular Tachycardia?	AV dissociation, QRS > 120, HR > 100, fusion beats & capture beats (both help distinguish from SVT)
What is the most common cause of Cor Pulmonale?	Cor Pulmonale = R heart failure 2/2 respiratory disease; COPD = most common CHRONIC cause; PE = most common ACUTE cause; others- pulm fibrosis, ILD, pulmonary HTN, sleep apnea
What are contraindications for Coumadin with known AFib?	Alcoholism, recent trauma or surgery, respiratory bleeding, active GI bleeding, GU bleeding, ICH, or significant risk of falls
Review CHA2DS2VASc scoring to determine need for anticoagulation with AFib	CHF (1), HTN (1), Age ≥75 (2), DM (1), Stroke (2), Female (1); Rx- Low risk (0)- ASA or none, intermediate risk (1)- consider AC, high risk (≥ 2)- start AC
How are vitals assessed on a patient with an LVAD?	Blood pumped by machine from LV to aorta, no pulse will be present, need to check with BP cuff to obtain MAP (goal 70-80)
Pt with an LVAD and elevated LDH?	Think pump thrombosis. Thrombosis leads to hemolysis which then leads to elevated LDH. LDH levels are typically > 1000
what is the most common site for infection in a LVAD?	Drive line, followed by pump pocket
What characterization of chest pain is most consistent with a cardiac cause?	Radiation of pain down to Right arm > radiation down both arms > radiation down Left arm
What is the path of electrical conduction during a normal cardiac cycle?	SA node → R atrium → AV node → Bundle of His → Bundle branches → Purkinje fibers
What are the most appropriate locations for central line placement prior to transvenous pacing?	Right IJ (preferred), L Subclavian (these offer the most direct routes to the heart)
What happens when a magnet is placed over an AICD?	It disables defibrillation and switches to pacing mode; should be done if pt is receiving inappropriate shocks. Note all AICDs are also pacemakers (on XR AICDs have a thicker wire in the distal lead)
What is oversensing in a pacemaker?	When a pacemaker interprets signal noise as native heart beats and does not generate a paced beat. Tx: place magnet over the pacemaker to switch it back to pacer mode. This will present as an individual with a pacemaker that is bradycardic and symptomatic and w/o pacer spikes on the ekg.
What is the appropriate management of a patient with an AICD with unstable VT?	Immediate electrical cardioversion for AICD/pacemaker malfunction

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What medication decreases mortality after an MI?	Aspirin
With cardiac arrest, what drugs can be given to adult and pediatric patients by ET tube?	NAVEL (adults): Narcan, Atropine, Vasopressin, Epinephrine, Lidocaine LANE (peds): all except vasopressin

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Trauma	
Bizz	Buzz
Difference between tension PTX and pericardial tamponade (for boards)	<u>Tension pneumothorax</u> : tracheal deviation, decreased breath sounds, subQ air. <u>Pericardial tamponade</u> : decreased heart sounds. BOTH will have JVD, hypotension, tachycardia
How to determine GCS	E4 V5 M6; <u>Eyes</u> : 4- Spontaneous, 3- Voice, 2- Pain, 1- None; <u>Voice</u> : 5- Normal, 4- Confused, 3- Words, 2- Sounds, 1- None; <u>Motor</u> : 6- Follows commands, 5- Localizes pain, 4- Withdraws to pain, 3- DeCORTicate (arms to CORE; flexed), 2- Decerebrate (extended) posturing, 1- None; GCS < 8 intubate
How to determine ASC Class of Hemorrhagic Shock	I : Normal vitals (<15% loss, 750cc), II : Tachy, but normal BP with ↓ PP (15-30% loss, 750-1.5L), III : Hypotension (30-40% loss, 1.5-2L), IV : AMS-confused/lethargic (>40%, >2L)
Vital Sign changes with Brain Herniation	Cushing's reflex (2/2 inc ICP): Hypertension, Bradycardia, Irregular Respirations
Compare subfalcine, uncal and tonsillar herniation	<u>Subfalcine</u> : most common, frontal lobe under falx, ssx abnormal gait; <u>Uncal</u> : temporal lobe under cerebellar tentorium, ssx CN3 palsy (blown pupil, down and out), ipsilateral hemiparesis, coma; <u>Tonsillar</u> : rare, brainstem herniation, coma and death
Injuries CT can commonly miss	Diaphragmatic injury, pancreas injury, basilar skull fracture, hollow viscus injuries
Classification of LeFort Fractures	Midface fx resulting in detachment of maxilla from skull all the fractures involve pterygoid plate; Dx : CT; I : palate mobile (fx below nose); II : palate + nose mobile (inferior orbits); III : entire face is mobile (zygoma bone), ± CSF rhinorrhea. IV : a III that involves the frontal bone
Dx and Tx of Mandibular Fractures	SSx: malocclusion, trismus, lower lip paresthesias; BODY = most common; Dx : CT or panorex; Rx : manage non-condylar fx as open fx with empiric PCN/Clinda, ENT/OMFS consult
Dx and Tx of Orbital Fractures	SSx: Diplopia, proptosis, limited EOM, ↓ visual acuity; check for infraorbital paresthesia, inhibited upward gaze , diplopia, globe injury; Dx : CT orbit; Rx : consult ophtho/ENT, decongestants, abx (Augmentin) for sinus involvement
Which facial bone fx has the lowest rate of infection?	Zygomatic.
Dx and Tx of Nasal Septal Hematomas	Dx : dark red mass/hematoma associated with nasal fx/trauma; Rx : MUST incise & pack (NO needle) to prevent saddle nose deformity/pressure necrosis
Classification of Neck Zones	I : sternum/clavicles to cricoid cartilage; II : cricoid to angle of mandible, (most common site of injury); III : angle of mandible to base of skull

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Management of Penetrating Neck Injury	Intubate early, straight to OR if unstable vitals or HARD signs of vascular injury ("HARD BRUIT": H ypotension (shock), A rterial bleeding, R apidly expanding hematoma, D eficit [pulse/neuro], B ruit/thrill), airway obstruction; Soft signs: CT angio, possible scope/exploration if stable
Possible complications of blunt neck injury	Pseudoaneurysm, carotid artery dissection, tracheal injury; Dx: CT angio, if unstable, intubate/ENT consult; blunt neck trauma + neuro findings = carotid artery dissection until proven otherwise
Dx and Tx of Traumatic Aortic Dissection	Most die in field; Consider when- high speed deceleration, chest pain/back pain, new murmur, pulse deficits BUT exam often unremarkable; Dx: stable- CXR (1/3 normal, look for mediastinal widening, obscured aortic knob, L apical pleural cap, R tracheal deviation, ↓ L bronchus/↑ R bronchus, loss of AP window, R displaced NGT), VERY stable- get CTA; Rx: if (+) dispo to OR on beta blocker for BP control; aortic isthmus = most common location
Dx and Tx of Flail Chest	≥3 adjacent rib fractures at 2 different points; leads to paradoxical chest motion with respirations; Association: pulmonary contusion; Rx: early intubation, ± chest tube
Appropriate imaging to eval for sternal fracture	Must get lateral CXR; consider CT if high suspicion and XR (-)
Identify high risk rib fractures	1-2: associated with vascular and broncheal injuries; 9-11: associated with liver and spleen lacerations; 4-9: most common location; multiple ribs: associated with underlying lung contusion
Indications for OR Thoracostomy with Hemothorax	Unstable vitals, initial chest tube output >1.5L (20cc/kg) OR >200/hr over 3-4hr (3cc/kg), persistent bleeding >7cc/kg/hr, persistent air leak
Management of Traumatic Pneumothorax	<u>Small</u> : O2, repeat CXR; <u>Large</u> : chest tube; **Pearl: if intubating with ptx, do chest tube first to prevent tension ptx**
Indications for ED Thoracotomy	<u>Penetrating</u> traumatic cardiac arrest: field arrest with initial vitals, ED arrest, SBP <50 after IVF; <u>Blunt</u> trauma: ED arrest; <u>Other</u> : suspected air embolism
General approach to traumatic abdominal injury	OR (penetrating): unstable vitals, peritonitis w/ (+) FAST, evisceration, or transabdominal GSW; STABLE- get CT, DO NOT send unstable patient to CT; Rx blood for shock even if normal initial H/H
Most common injury sites for abdominal GSW and stab wound	<u>GSW</u> : small bowel; <u>SW</u> : Liver (Blunt abdominal trauma: spleen > liver)
Dx and Tx of Diaphragmatic Injuries	Both sides are injured equally, but historically L>R for blunt (more common) & penetrating; consider with any injury nipple to navel; frequently missed/delayed dx; SSx: SOB, chest/abd pain, n/v, Kehr sign (referred L shoulder pain); Dx: CXR with coiled NGT in chest = pathognomonic, blurred hemidiaphragm, air/fluid level in chest; CXR & CT miss 50%; Definitive dx: laparoscopy/otomy in OR
Abdominal pain 2/2 bike handlebar injury	Duodenal/pancreas injury

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Abdominal pain after lap belt injury	Small bowel injury. Part of the “seat belt syndrome” Which includes: 1. Transverse abdominal wall contusion 2. Chance fracture 3. Abdominal visceral trauma (e.g. small bowel injury).
What volume of fluid/bleeding is required for positive FAST?	250ml
What traumatic injuries would NOT be identified on FAST?	Poor sensitivity for solid-organ injury, hollow viscous injury, and retroperitoneal injury
When is DPL considered positive?	Use if unstable + no US or equivocal FAST; (+) DPL if (Rule of 10's)- 10 mL initial gross blood/bile/feces; if no gross contents, infuse 1000cc and then aspirate: (+)DPL if >10,000 RBCs (penetrating) or >100,000 RBCs (blunt)
How to dx retroperitoneal injuries	CT with IV contrast, FAST will be negative
Dx of scrotal/testicular injuries	Hx: straddle injury, hematuria, scrotal ecchymosis/hematoma/ttp; Dx: doppler US, urology consult
Time limit to reimplant amputated penis	8-12hr max
Dx and Tx of Bladder/Urethral Injuries	<u>Red flags</u> : pelvic fx, gross hematuria, blood at meatus, urinary retention, high prostate, perineal bruising, females may have vaginal bleeding; Rx: NO FOLEY, RUG first to eval for urethral injury, CT cystogram for bladder injury, urology consult; Partial urethral lacs typically treated with Foley, complete urethral lacs require surgery
Interpretation of RUG for possible urethral injuries	<u>Anterior Urethral Injury</u> : distal to UG diaphragm, usually external signs of trauma, RUG: small extravasation with bladder filling; <u>Posterior Urethral Injury</u> : proximal to UG diaphragm, usually normal external exam, RUG large extravasation into pelvis
Dx and Tx of Bladder Rupture	Red flags: pelvic fx, gross hematuria (most common); Dx: retrograde cystogram; Rx: Intraperitoneal (contrast posterior to bladder) → OR (non-emergent), Extraperitoneal (flame pattern) → Foley (no OR)
Management of Renal Injuries	Blunt trauma causes 90% renal injuries; SSx: gross hematuria (microscopic hematuria is rare for significant injury); Dx: CT with IV contrast; Rx: All ureteral injuries go to OR, most blunt renal injuries are nonoperative; Renal injury is rarely in isolation - look for other injuries
Management of severe head trauma	Intubate with GCS ≤ 8; remove c-collar + hold inline cervical stabilization for intubation; hyperventilation → cerebral vasoconstriction → ↓ ICP
NEXUS Criteria	Used to clear c-spine; DOES NOT HAVE "Cervical PAIN": <u>C</u> ervical ttp, <u>P</u> ain that distracts, <u>A</u> MS, <u>I</u> ntoxication, <u>N</u> euro deficits
How to read C-spine XR	<u>Lateral</u> : Ant/Post spinal line & spinal laminar lines should be smooth; normal pevertebral space- 6mm at C2 & 22mm at C6; <u>Open mouth</u> : C1 and C2 lateral edges should align, look at odontoid for fx

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Identify unstable C-spine injuries	"Jefferson Bit Off A Hangman's Thumb"; <u>Jefferson fx</u> (C1 burst fracture 2/2 axial load), <u>Bilateral facet joint dislocation</u> (2/2 hyperflexion), <u>Odontoid fx</u> (Type I - tip [stable fx]; II - neck [most common]; III - body), <u>Atlanto-axial dislocation</u> (C1/C2 dislocation), <u>Hangman's fx</u> (bilateral C2 pedicle fracture 2/2 hyperextension), <u>Teardrop fx</u> (anterior & inferior vertebral body fx with interspinous ligament rupture, 2/2 flexion > extension)
Identify landmarks for anterior, middle and posterior spinal column (Denis model)	Classification for thoracolumbar fx; Ant column: anterior half of vertebral body, Middle column: posterior half of vertebral body, Post column: posterior to vertebral body; > 2 columns = unstable
Most common location of spinal fractures	T11-L2 (50%). **Spinal fractures often occur in multiples**
Describe wedge, burst and Chance fractures	<u>Wedge</u> : compression of anterior column. <u>Burst</u> : crush with multiple fragments involving anterior & middle columns. <u>Chance</u> : fracture through all columns, associated with lap belt injuries
Identify spinal cord syndromes: Central Cord, Anterior Cord, Brown-Séquard	<u>Central cord</u> : 2/2 hyperextension, usually elderly person hitting chin, sensory (cape distribution) & motor deficit, UE > LE; <u>Anterior cord</u> : 2/2 hyperflexion, bilateral motor paralysis, loss of pain/temp, intact vibration & proprioception; <u>Brown-Séquard</u> : penetrating trauma to 1/2 spinal cord, ipsilateral loss of motor, vibration & proprioception, contralateral loss of pain/temp
Identify landmarks for dermatomes: C6/7/8, T4, T10, L1, L4/L5/S1, S3-5	C6 - 1st dorsal web space, C7 - middle finger, C8 - pinky finger, T4 - nipple, T10 - umbilicus, L1 - inguinal ligament, L4 - patella, L5 - big toe, S1 - 5th toe, S3-5 - anus
What are the clinical findings in Neurogenic Shock?	Loss of vasomotor & sympathetic tone; Classic features: hypotension (vasodilation), bradycardia (unopposed vagal tone), poikilothermia (peripheral vasodilation, "warm shock"); Rx: IVF, pressors, Atropine
What is Spinal Shock?	Not true shock, more of a spinal "stun" 2/2 no circulatory involvement; SSx: areflexia & flaccid paralysis (all transient), relative bradycardia; first reflex to return is bulbocavernosus
Perimortem C-Section	Indication: gestation ≥ 24 weeks + loss of maternal vital signs Best if delivery performed ≤ 5 minutes after arrest Does not worsen maternal outcome
What population is at highest risk for intimate partner violence?	Pregnant women
Differential for serious complications in Pregnant Trauma?	Placental abruption, maternofetal hemorrhage, uterine rupture, preterm labor
Review Blast Injury Types I-IV	1° : blast shock wave (hollow viscus injury; TM rupture = most common blast injury, blast lung), 2° : projectiles from explosion (penetrating trauma, amputations, lacs); 3° : blunt trauma from explosion (crush injuries, blunt trauma); 4° : environmental contamination (burns, inhalation injury, smoke, radiation, etc.)

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Review concerning specialized blast-related injuries	<u>TM rupture</u> : most common injury, CXR if (+) to look for blast lung; <u>Blast Lung</u> : pulmonary barotrauma, most common cause of death, Dx: CXR (patchy opacities in butterfly pattern); <u>Others</u> : delayed intraabdominal injuries, compartment syndrome
Immediate and Delayed possible complications with myocardial contusion	<u>Immediate</u> : arrhythmia (sinus tach most common); <u>Delayed</u> : pericardial effusion (most common complication), MI/CHF, valvular injuries (aortic valve + AR most common), ventricular wall rupture (rare; most common cause of death in nonpenetrating cardiac injuries); Sternal fx = most common associated fx. Screen with ekg not troponin
What is the most common cause of in hospital death following near-hanging?	Pulmonary edema
What criteria require transfer of a patient to a Trauma Center?	Abnormal vitals, GCS < 14, penetrating trauma, severe blunt injuries (flail chest, multiple long bone fx), pelvic or skull fx, neurological deficits, high mechanism MVC or ped vs auto, elderly or kids, anticoagulant use, pregnant > 20wks
Potential complication of not repairing a galeal laceration?	Loss of the frontalis muscle function
2 days after a femur fracture, pt presents altered, hypotensive, hypoxic and w/ petechiae. They rapidly progress into ARDS and DIC.	Fat embolism to the lungs. Supportive care. Aggressively intubate because of the development of ARDS and AMS. There is no specific treatment.
what type of brain bleed is associated with basilar skull fractures?	Epidural hematoma. Temporal bone is most fractured bone in the base of the skull. Middle meningeal artery runs along the temporal bone.
Fat protruding through an eye lid laceration makes you suspicious for what?	Globe injury. Eyelid do not have fat therefore the presence of fat is concerning for deeper injury.
Tourniquets	Wider tourniquets are more comfortable and less likely to cause soft tissue/nerve injury. The Windlass tightening mechanism is a good mechanism when applying tourniquets.

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Gastrointestinal	
Bizz	Buzz
What pain medication is best for biliary colic?	NSAIDS, it is prostaglandin mediated pain
US with +gallstone and dilated common bile duct	Cholelithiasis, ± Jaundice, Rx: ERCP
Gold standard for diagnosing choledocholithiasis?	MRCP. ERCP and endoscopic US are good as well but they are invasive.
How sensitive if Murphy's sign for Acute Cholecystitis?	65-70%
What are possible US findings in Acute Cholecystitis?	Gallstones, gallbladder wall thickening (>3mm), pericholecystic fluid, sonographic Murphy's
Who is most at risk for Acalculous Cholecystitis?	Inflamed GB but NO stone; typically in very sick (hospitalized) or elderly
Fever + RUQ pain + Jaundice	<u>Charcot's Triad</u> ; <u>Reynold's Pentad</u> : add AMS, hypotension; <u>Cholangitis</u> : biliary obstruction with ascending bacterial infection; HIGH Mortality, Rx: abx, ERCP vs surgery
Chronic RUQ abd pain, Jaundice, Weight Loss	Cholangiocarcinoma
What is the risk of cancer in patients with a Porcelain Gallbladder?	25%
What arthropod is associated with pancreatitis?	Scorpion
Abdominal pain with bruising around the flank and umbilicus?	Hemorrhagic Pancreatitis; Ecchymosis of left flank (Grey-Turner sign), umbilical ecchymosis (Cullen sign)
Does lipase level correlate with severity of disease in Pancreatitis?	No
What are the components of Ranson's Criteria in Acute Pancreatitis?	Predicts mortality; <u>At admission</u> : Age > 55, WBC > 16k, Glucose >200, LDH > 350, AST > 250; <u>At 48hr</u> : Ca < 8, Hct drop > 10%, PO2 < 60, BUN increase >5, Neg base excess > 4, Fluid sequestration > 6L
What is a potential consequence of Chronic Pancreatitis?	Malabsorption when 90% affected
Painless jaundice and palpable gallbladder (Courvoisier sign)	Pancreatic Cancer; most common at head of pancreas, high mortality, high CA 19-9; also may have "Trousseau's sign" (migratory thrombophlebitis)
What is the difference between incarcerated and strangulated hernias?	<u>Incarcerated</u> : stuck; <u>Strangulated</u> : ischemic (requires surgery)

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What is the underlying pathology in Achalasia?	Impaired relaxation of the lower esophageal sphincter (LES), absence of peristalsis; most common esophageal motility disorder. Pts will present with dysphagia and they will “raise their arms above their heads” or “straighten their backs” after eating to increase intraesophageal pressure
Chest pain after vomiting, ill-appearing	Boerhaave's Syndrome: full-thickness perforation of esophagus causing mediastinitis; <u>Mackler's Triad</u> : SQ emphysema + chest pain + vomiting; "Hamman's Crunch" (crunching sound around heart); Dx: esophagram (water soluble) or CT w/ contrast; Rx: abx, surgical consult
On what side of the esophagus is rupture most common	Left side (distal posterolateral esophagus)
What condition predisposes to spontaneous rupture of the esophagus?	Esophageal Candidiasis (consider in HIV patient); Rx: oral fluconazole, IV fluconazole if pt is septic or cannot tolerate PO.
Regurgitating food and recurrent aspiration pneumonia	Esophageal Diverticula (Zenker's is pharyngeal mucosa above UES)
Kid with witnessed choking episode	Esophageal (or tracheal) foreign body; do thorough workup so this is not missed
What is the most common location of obstruction in esophageal foreign body ingestion?	Cricopharyngeus (C6) > Aortic Arch (T4) > GE junction (T11)
What foreign bodies in the esophagus require immediate/emergent removal?	Button batteries, sharp objects, multiple objects. OR has been present in the esophagus 24hrs or more, airway compromised or evidence of perforation.
What is the appropriate management for a Food Impaction?	EGD. You can try Glucagon 1mg IV (relaxes LES and causes vomiting) while you wait for GI; if glucagon works, patients must followup for endoscopy after to r/o underlying structural abnormality
What is the most common structural abnormality found in patients with food impaction?	Schatzki's Ring: ring of mucosal or muscular tissue in the distal esophagus causing narrowing; SSx: dysphagia, impacted food bolus; Rx: endoscopy
Small volume blood after frequent emesis	Mallory-Weiss Syndrome: longitudinal, partial-thickness esophageal tear in the distal esophagus and proximal stomach
Pediatric patient with respiratory distress with feeding and recurrent pneumonia	Tracheoesophageal Fistula
Pediatric patient who presents not being able to tolerate solid foods but can have liquids.	esophageal web
Smoker with chest pain and dysphagia	Esophageal Cancer, increased risk for men, heavy ETOH, smoking, chronic GERD/Barrett's esophagus, most likely Squamous Cell
HIV patient with chest pain and dysphagia	Candida Esophagitis; risk of perforation. Tx w/ oral fluconazole or IV fluconazole if they cannot tolerate PO.

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What medications are more likely to cause Pill Esophagitis and what is the appropriate management?	Large pills or those coated with gelatin; Meds: antibiotics (tetracycline, doxycycline —think of pts getting treatment for acne, clinda), anti-inflammatories (NSAID/ASA), bisphosphonates, iron, vitamin c, potassium chloride; Rx: stop inciting medication, endoscopy if severe or persistent symptoms
What type of caustic ingestion is worse and why?	Alkali ingestions are worse (cause liqueactive necrosis and deeper burns) than Acid ingestions (cause coagulative necrosis and more superficial damage)
What is the appropriate management for Caustic Ingestion?	Do NOT induce vomiting or attempt decontamination, get upright CXR to r/o perforation, consult GI for endoscopy, consult surgery pm
What is the most common cause of Cirrhosis?	ETOH in US; Hepatitis C outside the US
Most common complication of cirrhosis?	Ascites
Cirrhosis + GIB (Dx and Tx)	Esophageal Varicies 2/2 portal HTN; Rx: airway protection, blood transfusion, PPI, octreotide, antibiotics (mortality benefit!), GI consult for endoscopy vs IR for TIPS
What medication improves mortality when given for variceal bleeding?	Ceftriaxone; likely increased translocation of bacteria (causing SBP) during GI bleed in cirrhotics
What are options for tamponade of massive GI bleeding?	Sengstaken-Blakemore tube, Minnesota tube, Linton tube
Liver disease and new renal dysfunction	Hepatorenal syndrome (acute renal failure without other reversible cause); most commonly associated with SBP. may be 2/2 large fluid shifts and renal hypoperfusion; high mortality; Rx: Albumin decreases mortality (as it prevent large fluid shifts after large volume paracentesis).
Liver disease + AMS	Hepatic Encephalopathy; accumulation of nitrogenous waste (e.g. ammonia); Triggers: infection (SBP common), GI bleed, meds, or constipation; Rx- lactulose and find/Tx underlying cause
What is the usual source of infection and diagnostic criteria for Spontaneous Bacterial Peritonitis (SBP)?	E. coli translocation from gut; Paracentesis positive if: total WBC > 500, PMN (neutrophils) > 250, pH < 7.34, low glucose, (+) gram stain/culture
What are the two main types of Liver Abscess and what is the correct treatment?	<u>Pyogenic</u> : (80% in US), sepsis/RUQ pain/JAUNDICE, mixed bacteria (staph/strep), Rx: broad spectrum antibiotics (Ceftriaxone, Ampicillin, metronidazole), surgical drainage; <u>Amoebic</u> : (10%) usually subacute presentation, 2/2 entamoeba histolytica, no jaundice, complication-amoebic dysentery, Rx: metronidazole, medical management; BOTH may cause biliary obstruction
What lab abnormalities are expected with Acute Viral Hepatitis?	Elevated AST/ALT (to 1000s), high conj and unconj bili, high Alk Phos, Coagulopathy
Which is more common: Hep B or Hep C?	Hepatitis C (85%), Hepatitis B (15%)

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Which hepatitis virus is most likely to cause chronic infection?	Hepatitis C - 80% cause chronic infection, 20% of these progress to cirrhosis
What is the risk of liver cancer in patients with Alcoholic Cirrhosis vs Hep B/C?	Alcoholic - 80%, Hepatitis - 25%; the most common cause of hepatocellular carcinoma is still chronic Hep B/C virus
How do LFTs help distinguish acute viral hepatitis from alcoholic liver disease?	ALT > AST with acute viral hepatitis; AST > ALT with alcoholic liver disease (Mnemonic: Scotch & Tonic, AST)
What antibody is diagnostic for Acute Hepatitis A virus?	Anti-HAV IgM in acute infection; Anti-HAV IgG in prior infection/vaccination
Markers for Hepatitis B Virus	<u>HBsAg</u> : active infection; <u>Anti-HBs</u> : recovered or immunized; <u>Anti-HBc IgM</u> : early marker of infection, (+) in window period, <u>Anti-HBc IgG</u> : best marker for prior HB; <u>HBeAg</u> : high infectivity; <u>Anti-HBeAb</u> : low infectivity
Severe abdominal pain in patient with AFib, nonfocal abdominal exam	Mesenteric Ischemia - 2/2 embolism (50%), a/t thrombosis in the SMA; jejunum = most common location, usually severe pain out of proportion to exam (nonfocal abd exam), lactic acidosis = late finding, high mortality; Dx- XR to rule out perf/free air, CTA = gold standard; Rx: abx, surgery consult
What is the most common location of injury with Mesenteric Ischemia?	Superior Mesenteric Artery
Describe Rovsing sign, Psoas sign and Obturator sign associated with Appendicitis	<u>Rovsing</u> : most sensitive, RLQ pain with palpation to LLQ; <u>Psoas</u> : RLQ pain with passive extension of the hip; <u>Obturator</u> : RLQ pain with internal rotation of the hip (very low sensitivity)
What are diagnostic criteria for diagnosis of Acute Appendicitis on US?	Non-compressible, tubular structure with a diameter \geq 6 mm must be visualized; Others- fluid, target sign, appendicolith
What is the most common type of bezoar?	Phytobezoar (food, fiber); others- Trichobezoar (hair), Pharmacobezoar (antacids, aspirin)
What is a common contributing cause of Gastric Adenocarcinoma	H. pylori; GI CA associated with left supraclavicular lymph node; MC cancer with H.pylori is Mucosa-associated lymphoid tissue lymphoma (MALToma)
Periumbilical Lymph Node	Metastatic spread of CA to peritoneum (Sister Mary Joseph node)
Describe the intestinal and extraintestinal manifestations of Crohn's Disease	<u>Intestinal</u> : terminal ileitis (classic) [remember yersinia can also have terminal ileitis], "skip lesions" of normal bowel between disease, can involve ANY part of the GI tract, complications- abscess, fistula, stricture, toxic megacolon; <u>Extraintestinal</u> - arthritis (most common), uveitis, erythema nodosum; Rx- steroids, immunosuppressive
Describe the intestinal and extraintestinal manifestations of Ulcerative Colitis	<u>Intestinal</u> : continuous disease (no skip lesions) of rectum and colon ONLY, complications- toxic megacolon, increased risk of cancer; <u>Extraintestinal</u> : arthritis, uveitis, erythema nodosum; Rx: steroids, less often antibiotics
What is the most common cause of Small Bowel Obstruction?	Adhesions (very common with prior surgery) > tumor/mass > hernia

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History of AAA repair with massive GI bleed	Aortoenteric fistula; Triad: GI bleed ("herald bleed") + abdominal pain + palpable mass; rare, but high mortality; Rx: blood, surgical consult. Duodenum is most commonly involved portion of the intestines.
Diarrhea after recent antibiotic use	C.difficile (anaerobic gram positive bacillus) causing Pseudomembranous Colitis; Dx: stool antigen; Rx: PO metronidazole or vanco PO vs stool transplant
What two signs/symptoms exclude a diagnosis of Irritable Bowel Syndrome	Fever or blood in stool
Review the typical presentation and treatment for Sigmoid vs Cecal Volvulus	<u>Sigmoid</u> : most common, elderly/nursing home, immobilized, chronic constipation, Triad: abdominal pain + distension + constipation, Dx: XR with inverted "U", Rx: sigmoidoscopy with rectal tube decompression (stable), surgery (definitive); <u>Cecal</u> : younger, marathon runners, Dx: XR with kidney-shaped/coffee-bean (massively dilated cecum in the LUQ), comma sign; Rx: surgery, abx for perforation
Sudden severe abdominal pain, abdominal distension and inability to pass NGT	Gastric Volvulus - closed loop obstruction, ischemia and perforation. Typically happens in the elderly or infants w/ congenital diaphragmatic defect. Tx: try to pass a NGT (on an adult). Call surgery.
Risk with chronic perirectal abscess	Fistula formation (classically with Crohn disease)
Abscess above gluteal cleft near midline	Pilonidal cyst/abscess; Rx: I&D in ED, surgical removal (definitive); recurrent disease = most common complication
Dx and Tx of Proctitis	Inflammation of lining of the rectal mucosa caused by STD (N. Ghonorhea most common) > radiation, Crohn's; SSx: tenesmus, rectal discharge; Dx: sigmoidoscopy, treat infection
What is the most common location for an anal fissure? What should be considered for anal fissures NOT at this location?	Posterior midline (90%); if anal fissure found not at midline (lateral) should consider systemic process: Crohn's, HIV, leukemia, tuberculosis or syphilis
Tx of thrombosed hemorrhoid	Excision of clot with elliptical incision
What are the classifications of internal hemorrhoid severity I-IV?	I: don't protrude through anus; II: prolapse but spontaneously reduce; III: prolapse but require manual reduction; IV: prolapse and cannot be reduced (± strangulation)
Dx and Tx of Rectal Prolapse	Seen in young and elderly (related to constipation), also with anal intercourse; Consider cystic fibrosis in Peds; Dx: red mass protruding from anus; Rx: manual reduction (can use granulated sugar), surgery consultation pm (ischemia)
Large bowel obstruction without identified obstructing lesion on CT?	<u>Ogilvie's Syndrome</u> : colonic pseudoobstruction, elderly/bedridden patients with comorbidities, massive dilatation of the colon (>10cm), absence of mechanical obstruction; Rx: colonic decompression and neostygmine
What is the most common cause of surgical and non-surgical abdominal pain in the elderly?	Surgical: Acute Cholecystitis (**present with milder symptoms); Non-surgical: Pancreatitis

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What is the most common cause of acute pancreatitis?	Gallstones (45%) > Alcohol (35%)
What is the appropriate treatment for epiploic appendagitis?	NSAIDs, supportive, likely discharge with outpt f/u
What is the appropriate management for intussusception in children and adults?	<u>Children</u> : barium or air enema to reduce if uncomplicated; <u>Adults</u> : surgery as most are associated with a mechanical cause (most often tumor)
What LFT abnormalities are expected in the following conditions: Gilbert's, Hemolysis, Alcoholic Hepatitis, Cholestasis, Ischemic Hepatitis	<u>Gilberts & Hemolysis</u> : elevated indirect bili; <u>Alcoholic</u> : AST>ALT in a 2:1 ratio; <u>Cholestasis</u> : elevated direct bili; <u>Ischemic</u> : very high AST & ALT (e.g. 10,000)
When is a G tube tract considered mature?	After 2-3 weeks. Before then, call a surgeon, consider antibiotics and imaging and DO NOT replace it as you could cause a false tract.

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Pediatrics	
Bizz	Buzz
Neonatal jaundice w/in 24hrs of life	BAD sign, ABO incompatibility, Rh incompatibility, TORCH infections, G6PD def. Admit, hydrate and order Coombs test.
Neonatal jaundice 24hr-72hrs	Physiologic (indirect hyperbili), sepsis, others
Neonatal jaundice > 72 hrs	sepsis, breast milk jaundice, breast feeding jaundice, Gilberts. Remember, breast feeding jaundice : suboptimal supply of breast milk, requires hydration and supplementation. Breast Milk jaundice is when the baby's liver is not developed enough to handle breaking down the supply of breast milk from mom.
Asian baby age 1 month with jaundice and direct hyperbile	Biliary atresia - dx of intra and extrahepatic bile ducts leading to obstructive jaundice, cirrhosis, and death. Typically diagnosed before 2months ago. Tx: surgery w/ Kasai procedure.
Baby with conjugated hyperbili	**Will require admission and work up**. DDx: biliary atresia, SEPSIS, cholelithiasis, cystic fibrosis, Wilson's, etc ...
What are the most concerning (and unique) causes of abdominal pain in the following age groups: 0-3mo, 3mo-2yr, school aged kids	<u>0-3mo</u> : Necrotizing Enterocolitis, Hirschprung's/Toxic Megacolon, Volvulus, Pyloric Stenosis; <u>3mo-2yr</u> : Intussusception, Meckel's Diverticulum, Foreign Bodies; <u>School age</u> : similar to adults including pregnancy (consider if >8)
Dx and Tx of Necrotizing Enterocolitis	Inflammation & necrosis of the bowel wall from translocation of gut bacteria; prematurity(greatest risk factor); SSx: bilious emesis, bloody stools, abdominal wall erythema; Dx: XR with pneumatosis intestinalis (pathognomonic), portal vein air (poor prognosis); Rx: IVF, broad spectrum antibiotics, NG tube (bowel rest), surgery consult, admit
Dx and Tx of Hirschsprung Disease	Lack of ganglion cells in the rectosigmoid colon; SSx: Delayed passage of meconium (>48 hr) → obstruction & bilious emesis (late finding); Complications: enterocolitis/toxic megacolon; Dx: rectal biopsy (gold standard), contrast enema (transition zone); Rx: surgery, admit
Dx and Tx of Midgut Volvulus	1st mo of life; Congenital malrotation → volvulus → midgut ischemia; SSx: bilious vomiting (always emergent), abd pain/distention, ± rectal bleeding/hematochezia (gut ischemia); XR "double bubble" can also be seen in duodenal atresia; Dx (definitive): upper GI series "corkscrew", US; Rx: NGT, surgery consult. Associated conditions: congenital diaphragmatic hernia, congenital heart disease, omphalocele
Dx and Tx of Intussusception	6mo-3yr; telescoping of bowel (ileoceal most common); Tumor, Meckel's, post-viral, HSP; SSx: colicky abd pain w/ LETHARGY + abd mass (sausage-shape in RUQ; RLQ usually empty) + "currant jelly" stools; AXR: obstruction, Dance's sign (pathognomonic); Dx (preferred): US "target sign"; Rx: OR (sick), air/contrast enema (not sick), abx

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Dx and Tx of Meckel Diverticulum	Most common congenital GI malformation. Incomplete closure of vitelline duct → heterotopic gastric mucosa; SSx: painless rectal bleeding 2/2 ulceration → obstruction (2/2 intussusception/volvulus/hernia; <u>Rule of 2s</u> : 2% of population, 2% symptomatic, 2ft proximal to terminal ileum, 2x more often in males, 2yo most common; Dx: Meckel scan; Rx: surgical consult
Where do ingested foreign bodies usually get stuck?	Cricopharyngeus C6 (60-80%), GE junction T11 (10-20%), Aortic Arch T4 (5-20%); Coin most common object swallowed. CXR (AP): coin appears flat if in esophagus
What are indications for emergent endoscopy for ingested foreign body?	High-grade obstruction, object in esophagus >24hr, object >6cm, sharp objects, multiple objects swallowed, button battery in esophagus, button battery in stomach >48hr or if symptomatic (earlier)
Dx and Tx of Pyloric Stenosis	Age 2-8 wks. Hypertrophied pylorus. Most common congenital GI disorder. Risk factors: first-born males, macrolide abx exposure. SSx: nonbilious projectile vomiting, "hungry vomiter"; Labs: hypoCl, hypoK, metabolic alkalosis (2/2 vomiting), dehydration. Exam: palpable "olive-shaped" mass. Dx: US (target sign), upper GI series "string sign". Rx: IVF, surgery
What is the most likely location of traumatic C-spine injury in young children?	Age < 8yrs more susceptible to upper cervical spine injuries (C1-3).
What are normal variants in pediatric c-spine imaging?	Pseudosubluxation (C2 on C3), growth plates can look like fractures, anterior wedging
What is SCIWORA?	"Spinal cord injury without radiographic abnormalities." May present with missed old injury leading to significant subsequent injury after relatively minor trauma. XR/CT without abnormalities, MRI will show problem area. Most commonly seen in children and the elderly.
Review common causes of anemia in young children	Physiologic nadir (Hgb 9 at 6wks), B12/folate deficiency (high MCV, hypersegmented polys, seen in vegans), Iron deficiency (1-2yr, low MCV, associated with <i>pica</i> , breath holding, high milk intake (more than 28-32 ounces per day), Sickle Cell dz (hemolysis, high retic count), Lead Poisoning (basophilic stippling, abd pain, AMS)
Approximate weight for newborn, 1yr, 5yr, 10yr	Newborn: 3.5kg, 1yr: 10kg, 5yr: 20kg, 10yr: 40kg
How do you determine ETT size, depth, and blade size in young children?	Newborn: 3.5 ETT, otherwise ETT = Age/4 + 4 (minus 0.5cm for cuffed); Depth = 3x tube size; Blade = 1 for newborn and 2 from 2-12yr, 3 > 12yr
ETT size for premature neonates or small neonates?	premature: 2.5 uncuffed / small (weight 1.6-3kg) 3.0 uncuffed
What are the general cutoffs for abnormal vitals in a newborn/infant?	Pt is SICK if SBP < 60, RR > 60, HR > 180; Normal SBP = Age x 2 + 70
Dx and Tx of Breath Holding Spells	6mo-6yr; associated with pain/emotion, ± turn blue then pass out, but child returns to normal after this and is otherwise well. Rx: reassurance. rule out Fe deficiency anemia, otherwise pt will grow out of it

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Dx and red flags of Tic/Movement Disorders	More common in males, suppressible but involuntary in otherwise normal child. Red flags: head bobbing, neuro deficits, nystagmus, choreoathetoid movements
What is the approximate blood volume in a child?	80cc/kg
At what level of blood volume loss does a child drop their BP?	30%
Peds trauma + hypotension, what should initial bolus of blood and IVF be?	pRBC: 10cc/kg, crystalloid: 20cc/kg
Review distinguishing characteristics of the following viral exanthems: Measles/Rubeola, Rubella, Erythema Infectiosum/5th Disease, Varicella, Roseola, Hand/Foot/Mouth Dz	<u>Measles (Rubeola)</u> : cough/coryza/conjunctivitis (3 C's), Koplik's spots, rash: maculopapular, head → feet; complications: AOM (most common), encephalitis. <u>Rubella</u> : "3d Measles"; suboccipital/posterior auricular LNs, petechiae on hard palate, rash: maculopapular: face → trunk. <u>Erythema Infectiosum</u> : Parvovirus B19, URI sx (3-5d) → rash: "slapped cheek" with circumoral pallor; aplastic crisis in sickle cell dz. <u>Varicella</u> : vesicles (dew drop on rose petal) in crops at different stages, spares palms/soles. Rx: Acyclovir for immunosuppressed/age>12, encephalitis/pneumonitis. <u>Roseola</u> : HIGH fever → rash (blanching maculopapular), HHV-6, assoc. febrile sz, mimics sepsis/meningitis. <u>Hand/Foot/Mouth</u> : Coxsackievirus, URI prodrome, vesiculopapular lesions (hands/feet), anterior mouth ulcers (most common, tongue/buccal mucosa). <u>Herpangina</u> : posterior painful, papulo-vesiculo-ulcerative exanthem (fever)
Distribution and Tx of Tinea Infections	T. capitis: head, T. corporis: body, T. cruris: groin, T. pedis: foot, T. unguinum. Rx: topical antifungals UNLESS in hair (PO griseofulvin x 8wks)
Dx and Tx of Kerion	Inflammatory head/hair fungal lesion on scalp. Rx: PO griseofulvin. Complication: scarring alopecia
Distinguish, Dx, and Tx staph/strep infections including Impetigo, Bullous Impetigo, Staph Scalded Skin	<u>Impetigo</u> : age < 6, honey-crusted lesions on face, pruritic NOT painful; Rx: topical mupirocin. Complication: glomerulonephritis. <u>Bullous Impetigo</u> : bullae formation, Rx: topical mupirocin + PO Keflex. <u>SSSS</u> : severe form of bullous impetigo (extensive bullae), infants/young children, rash: erythroderma (perioral is classic), NO muosal involvement, (+) Nikolski's, Rx: PCN (e.g. Dicloxacillin) ± MRSA coverage (e.g. Vanc), admit
Dx and tx of acute rheumatic fever?	Child w/ recent hx of strep throat that has: fever, migratory polyarthralgia, signs of pericarditis or carditis/CHF. Sydenham's chorea is rare but often tested. Jones Criteria exists but could be too specific for the test. Tx: Penicillin. ASA/NSAIDs for the arthritis.
Complication of untreated rheumatic fever?	Mitral stenosis as an adult
Dx and Tx of Scarlet Fever	Age 2-10. Group A Strep infxn. SSx: pharyngitis, strawberry tongue, circumoral pallor, rash: sandpaper feel, groin/axilla → trunk/ext, spares palms/soles, pastia lines (linear petechiae). Rx: PCN to reduce incidence of rheumatic fever (not glomerulonephritis)

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Dx and Tx of Erysipelas	Upper dermal infection with GAS. SSx: erythematous plaque, sharp border, ± ears. Rx: Amox/Keflex (mild), CTX (systemic dz)
Dx and Tx of Pityriasis Rosea	SSx: Herald patch → Christmas tree distribution of rash on back; Rx: improved with sunlight, antihistamines (pruritis), otherwise self-resolves
Distinguish, Dx, and Tx Scabies vs. Lice	<u>Scabies</u> : linear burrows (pathognomonic), pruritic rash (hand/feet/groin); Rx permethrin (NOT lindane for peds/pregnant → seizures), Ivermectin. <u>Lice</u> : nits (eggs attached to hairs) with extreme pruritis on head; Rx: permethrin (alt. malathion), scrape out nits, repeat tx 7-10d
Dx and Tx of Kawasaki Disease	Vasculitis. SSx: fever >5 days (most common sx) + 4/5 hallmarks-bilateral conjunctivitis, oral mucosal changes (lip cracking, "strawberry tongue"), ext. changes (hand/foot erythema), polymorphous rash, cervical LAD (at least 1 > 1.5cm) concern for cardiac aneurysms (get Echo); Rx: high-dose ASA, IVIG. Complication: coronary artery aneurysm
Dx and Tx of Henoch-Schönlein Purpura	Post-infectious vasculitis (IgA deposition); most common vasculitis in Peds. Age 6mo-5yrs. SSx (TRIAD): palpable purpura + colicky abd pain + arthralgia. Dx: hemolytic anemia, normal/high plt , AKI, sz, lethargy. Rx: If no renal failure, supportive care with NSAIDs. NO ABX. Admit if renal failure/involvement. Complications: intussusception (heme pos. stool), renal failure (micro. hematuria, proteinuria, elevated BUN/Cr)
what type of intussusception is seen in Henoch-Schonlein Purpura?	ileo-ileo. The most common type of intussusception outside of HSP is ileocecal.
Dx and Tx of Cat Scratch Disease	<i>Bartonella henselae</i> . SSx: cat scratch/bite 1-3 weeks prior, causes regional LAD. Rx: Doxycycline (Azithromycin in pregnancy)
Distinguish Simple from Complex Febrile Seizures	<u>Febrile sz criteria</u> : convulsions + fever, 6mo-5yr (NOT < 6mo), no CNS infxn/inflamm, no metabolic abnormalities, no h/o afebrile sz. <u>Simple</u> : 6mo-5yrs, single episode/24hr, <15 min, generalized (tonic/clonic); no neuro hx and normal exam; no special workup or tx needed. <u>Complex</u> : anything else.
Name most common midline and lateral congenital neck masses	<u>Midline</u> : thyroglossal duct cyst (vs. hemangioma). <u>Lateral</u> : brachial cleft cyst, asymptomatic. (vs. cystic hemangioma)
Dx of Peds Respiratory Foreign Body	Objects: coins (most common), peanuts, beans. SSx: high suspicion if sudden choking or coughing ± wheezing, stridor. CXR: obstructive emphysema (fb obstructs bronchus on expiration, hypodense), CT if in doubt, Rx: bronch (gold standard for Dx & Tx)
What is the approach to resuscitation in a choking child <1yr?	5 back blows, 5 chest compressions (no abd compressions)

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Dx and Tx of Croup	Age: 6mo-3yr. <i>Parainfluenza virus</i> . SSx: URI sx with barky, seal-like cough, inspiratory stridor, low grade fever, non-toxic appearance. XR: steeple sign. Rx: dexamethasone (0.6mg/kg), rac. epi. for stridor at rest (Rx before steroids if given, monitor for rebound x4hr); Dispo: admit if sick, hypoxic, or with persistent stridor. Consider bacterial superinfection
Dx and Tx of Epiglottitis	Age: 3-7 yr. <i>H. influenzae</i> (less since vaccine, now more adults), <i>Strep. spp.</i> (most common), <i>S. aureus</i> . SSx: toxic appearance, rapid progression of high fever, dysphagia. Exam: leaning forward/"tripod" position, dysphonia, drooling, inspir. stridor; XR: thumbprint sign. Rx: airway mgmt (OR for eval, BVM ok, avoid RSI), abx (CTX)
Dx and Tx of Bacterial Tracheitis	Age 3-5 yrs. <i>S. aureus</i> , mixed flora. SSx: URI prodrome similar to croup BUT intensifies to include high fever, inspir. & exp. stridor , mucopurulent sputum or cough. TOXIC appearing. XR: subglottic narrowing, hazy tracheal lumen. Rx: airway mgmt, abx. They do not respond to racemic epi or steroids. Pt's can breathe better when they lay flat.
Dx and Tx of Retropharyngeal Abscess	Age: 6m-4yrs. Staph/Strep/anaerobes. Common after trauma (e.g. popsicle stick), URI. SSx: fever, sore throat, dysphagia, drooling trismus, stridor; TOXIC appearing, limited neck extension, muffled voice; XR: widened pervertebral space 7mm at C2, CT (imaging study of choice). Rx: Clinda, ENT/OR. It's more insidious in onset when compared to epiglottitis.
Dx and Tx of Bronchiolitis	Age < 2yrs. RSV (most common). Lower airway inflammation. SSx: URI prodrome → fever, tachypnea, wheeze, apnea (<1mo) , lasts 1-2wks. CXR: diffuse infiltrates. Rx: mild- nasal suctioning, hydration; severe- trial of nebs (controversial), humidified HFNC. Dispo: admit if persistently hypoxemic or < 3mo for apnea monitoring.
Review the most common causes of pneumonia by age group: <3mo, 3mo-5yr, >5yr	<u>0-3wks</u> : GBS , <i>E. Coli</i> , <i>Listeria</i> , <i>Staph</i> ; <u>3wk-3mo</u> : C. trachomatis , RSV, pertussis; <u>1mo-5yrs</u> : RSV , numerous viruses, <i>S. Pneumo</i> , atypicals; <u>>5yrs</u> : M. pneumoniae , atypicals. SSx: tachypnea (best indicator of LRI), SaO2<92% (most sensitive). Rx: <u>Neonates</u> : septic w/u, Amp + Gent ± Cefotax, admit, <u>3wk-3mo</u> : Azithro ± Cefotax, <u>3mo-18yrs</u> : Vanc + CTX ± Azithro (ICU), CTX (inpt), Amox or Azithro (outpt)
Review the appropriate workup and treatment of fever in kids <4wks, 4-8wks, >8wks	<u><4wks</u> : (GBS, <i>E. coli</i> , <i>Listeria</i>) blood & urine cultures, XR, LP, admit, Rx: cefotaxime + ampicillin, add acyclovir & vanc depending on risk. <u>4-8wks</u> : more targeted, low threshold to treat like <4wks. <u>>8wks</u> : use clinical decision rules (Philadelphia, Rochester, Boston - generally well appearing, WBC <15, bands <1.5, CSF wnl, UA WBC <10); LP, abx. Dispo: home (low risk), admit (high risk). Always send UA.
Compare Dx and Tx of Myocarditis vs Pericarditis in kids	<u>Myo</u> : Most common cause of HF in kids. Viral infxn (Parvovirus); SSx: poor feeding & sweating, inc RR/HR, hepatomegaly (most specific finding or HF); Dx: nonspecific EKG, +trop; Rx: diuretics, ionotropes, IVIG, admit, consider ECMO. <u>Peri</u> : viral infxn (Coxsackievirus); SSx: fever + dyspnea + cp (worse lying flat); Dx: EKG diffuse STE or PR depression, trop (-), get TTE r/o effusion; Rx NSAIDs

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Compare Dx and Tx of PDA vs. ASD vs. VSD in kids	<u>PDA</u> : L→R shunt (Ao→PA); SSx: continuous machine murmur, wide PP, **may be worse with O2**; Rx: indomethacin, surgery. <u>ASD</u> : L→R shunt & R heart failure; SSx: asymptomatic unless large (often delayed dx), fixed split S2. <u>VSD</u> : most common congenital heart dz; L→R shunt R heart failure. SSx: loud, harsh holosystolic murmur at LLSB, usually presents @ 6wks; Rx: CHF tx, Peds cards, ± surgery
Name the 5 cyanotic congenital heart lesions	All R→L shunt. Truncus Arteriosus, Transposition of Great Arteries, Tricuspid Atresia, Tetralogy of Fallot, Total Anomalous Pulmonary Venous Return
Dx and Tx for ductal dependent congenital heart lesions	Rapid decompensation with cyanosis at d2-10 (when ductus closes). Lesions: coarctation of aorta, critical aortic stenosis, hypoplastic left heart, tricuspid atresia, tetralogy, transposition; Rx: PGE1 to reopen duct (side effects: hypotension, apnea, cardiac arrest), admit: PICU
Dx and Tx of Tetralogy of Fallot	Key features: RVH, RVOT, VSD, overriding Ao; SSx: cyanosis d2-10 with duct closure, shock, little improvement with O2; Dx: XR boot-shaped heart; Rx: PGE1, bicarb, IVF, blood, sat to 70s is ok
Describe a typical tet spell and appropriate treatment	Hypercyanosis associated with feeding, straining, crying, or exertion; Rx: inc. SVR (knee to chest, squatting), O2, morphine (dec PVR)
Dx and Tx of Coarctation of the Aorta	Most common cause of crashing neonate (healthy). SSx: <u>Neonate</u> - HF & shock @ d2-10 2/2 duct closure, <u>infant/child</u> - HTN UE>LE, UE pulse delay HF sx, <u>adults</u> -HTN, XR: rib notching Associations: Turner's syndromem intracranial aneurysms. Rx: PGE1, balloon angio+stent, surgery
What is the appropriate administration of glucose for hypoglycemia in neonates and kids?	"Rule of 50" or "5-2-1": <1yr: D10 (5cc/kg), 1-8 yrs: D25 (2cc/kg), >8 yrs: D50 (1cc/kg)
Dx and Tx of Congenital Adrenal Hyperplasia	Virilization ("girls look like boys, boys not much different") & salt wasting. 21-hydroxylase most common. SSx: adrenal crisis (↓ Na, ↑↑ K). Rx: glucose, IVF, IV steroids (Hydrocortisone, not dexamethasone, which only has glucocorticoid activity)
Dx and Tx of (Generic) Inborn Error of Metabolism	Ammonia & acid production. SSx: present at day 3-5 tachypnea, vomiting, AMS, sz's, odd smell. Labs: hypoglycemia, metabolic acidosis, hyperammonemia. Rx: stop protein breakdown (NPO, IVF), glucose (D10, no D5), remove ammonia (NH3 scavenging meds)
Dx painless abdominal mass, age 3-5yr	Wilms Tumor (nephroblastoma)
Risk factors for SUDI	Sudden Unexplained Death in Infancy; formerly SIDS. Unexpected death of infants without pathologic cause. Age <2yr (peak 2-4 mo). Risks: maternal smoking/drug use, prone sleeping (key!), loose bedding, soft sleeping surface, male, prematurity, +FHx; Protective: breast feeding, pacifier, supine sleeping

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Dx and Tx of BRUE	Briefly Unexplained Resolved Event; formerly ALTE. Age: 1wk-2mo. Sudden, brief, now resolved episode including: cyanosis or pallor, irregular breathing, change in tone, ALOC; Associations: Pertussis, RSV. Idiopathic = most common cause. Workup per h&p. Low threshold to admit for board exam.
Dx: Poor family, infant with seizure	Hyponatremia from dilute feeds. Rx: hypertonic saline (2ml/kg 3% NaCl)
Dx: AMS, kid with ETOH ingestion	Hypoglycemia (replete per rule of 50)
Dx: kid at grandma's house with AMS, pinpoint pupils	Clonidine ingestion, Rx: (lots of) narcan
Dx: kid with lethargy, intermittent crying	Intussusception
Dx: kid with bloody diarrhea, lethargy	EColi O157:H7 & possibly HUS (NO abx → increases chance of HUS)
Dx: Boyfriend babysits	Non-accidental trauma/abuse
Dx: Recurrent RML pna	Aspirated foreign body
Compare Dx and Tx of Phimosis vs. Paraphimosis	<u>Phimosis</u> : unable to retract foreskin, NOT an emergency if pt can urinate; Rx: topical steroid cream, gentle retraction; <u>Paraphimosis</u> : inability to reduce foreskin back to anatomic position, surgical emergency → causes ischemia, Rx: manual reduction, Urology consult (dorsal slit procedure, circumcision)
Describe the risks for UTI, criteria for sending Ucx, and dispo criteria for UTI in kids	Males: <1yr & uncircumcised < 2yrs, Females: ALL, esp < 2 yrs. Ucx analysis: suprapubic aspiration (rare but gold standard), even if udip normal, >50K CFU+; Fever + UTI = Pyelo requires admit for IV abx, otherwise home with abx
What is a common cause of UTI <1yr	50% with vesicouretral reflux or other structural abnormality
Dx and Tx of Reyes Syndrome	Kid takes aspirin for viral URI → AMS and fatty degeneration of the liver, cerebral edema. Rx: supportive.
What is the most common cause for meningitis in a neonate?	Group B Strep
Dx: Rectal prolapse in a kid	Cystic Fibrosis
Review neonatal conjunctivitis	Rule of 5's: <i>N. gonorrhoeae</i> (d0-5), tx with IV 3rd generation cephalosporin, <i>C. trachomatis</i> (5d-5wk), tx oral macrolide to prevent pneumonia, <i>Strep</i> or <i>H. flu</i> (5wk-5yr); eye = most common site of gonorrhea in newborns
Dx and Tx of Legg-Calve-Perthes disease	Idiopathic avascular necrosis of one/both femoral heads. Pt will be male 4-10yrs. SSx: limp + unilateral hip/thigh/knee pain, worse with activity. Limited hip abduction & internal rotation, ± limb length discrepancies. Dx: XR "moth eaten", "crescent sign". Rx: non-weight bearing, Ortho referral.

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Dx and Tx of SCFE	Slipped Capital Femoral Epiphysis. Most common hip disorder in teens. Obese males. Age 12-16yrs. SSx: limp (L>R), hip pain, ext. rotation deformity. Dx: XR pelvis ("ice cream falling off of cone"). Rx: non-weight bearing, surgery, admit. Complication: avascular necrosis.
Dx and Tx of Transient Synovitis	Inflammation + hypertrophy of hip synovium. Most common cause of hip pain ages 3-10. SSx: URI hx, limp with dec. ROM. Hip held in flexion, ABduction, and ext. rotation. Normal labs. XR: normal, US: ± joint effusion. ±Arthrocentesis (normal). Rx: NSAIDs. Association: Leg-Calve-Perthes.
Dx and Tx Septic Arthritis	Infection (most commonly <i>S. aureus</i>) of the joint space. Pt will be male <4yrs. Knee (most common joint) > hip. SSx: fever, irritability, pain, refusal to bear wt or move joint; if hip, held in flexion, ABduction and external rotation. Dx: WBC >12k, ESR >40, fever, refusal to bear wt (Kocher criteria). Synovial fluid will show WBC > 50,000 with > 75% PMNs. Rx: IV abx, surgery, admit.
Dx and Tx of Post-Streptococcal Glomerulonephritis	Follows a GAS infxn (pharyngitis > impetigo). SSx: HTN, hematuria, periorbital edema. Check UA: proteinuria, RBC casts. Others: +ASO titer, low C3 level. Rx: supportive. Abx do NOT prevent this dz.
What is biliary colic in pediatric populations most commonly associated with?	1. Hemolytic anemia (e.,g. sickle cell dx). Hemolysis → pigmented stones 2. Cystic fibrosis and 3. (less so) obesity
What is an apt test?	A way to determine if GI bleeding in a neonate is from the baby or swallowed from the mom. Basically, the bloody stool is exposed to an alkali solution and if it's fetal blood it will stay pinkish, red. Maternal blood will degrade.

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Respiratory	
Bizz	Buzz
What is the most common cause of infectious airway obstruction in children?	Croup, i.e. "laryngotracheobronchitis". <i>Parainfluenza virus</i> . SSx: barky, seal-like cough worse at night, inspiratory stridor
What is the characteristic XR finding in Croup?	"Steeple sign" (tapering of the upper airway on AP view)
Management of croup in the ER?	Dexamethasone for all. <u>Mild</u> : home tx, antipyretics, fluids, mist. <u>Moderate-Severe</u> : Rac Epi → if stridor goes away, observe for 3hrs and then okay to discharge home if stridor does not persist/return or if child is well appearing. Things that support admission for croup: age <6 months, stridor at rest, respiratory distress, hypoxemia, lack of good follow up.
Dx: Sore throat, normal posterior oropharynx, ill-appearing, in tripod position	Epiglottitis. <i>H.flu</i> (unvaccinated), <i>Staph/Strep</i> (vaccinated). More common in adults now thanks to the HiB vaccine
What is the characteristic XR finding in Epiglottitis?	"Thumbprint sign" (enlarged epiglottis on lateral view)
What is the appropriate treatment of Epiglottitis?	Emergent airway management. <u>Unstable/ill-appearing</u> : go to the OR with ENT for direct visualization/scope. Respiratory arrest = BVM. No RSI! <u>Well-appearing/stable</u> : consider XR. Rx: antibiotics, steroids with ENT consult.
Dx: Inspiratory whoop between violent coughing spells.	Pertussis. <i>Bordetella pertussis</i> . SSx (3 phases): <u>Catarrhal</u> (1-2wk): URI **HIGHLY CONTAGIOUS during this time**; <u>Paroxysmal</u> (2-3mo): violent "whooping" cough, inspiratory stridor; <u>Convalescent</u> (1-2wk): gradual reduction in sx. Dx: nasopharyngeal cx, PCR. Rx: macrolides (also cover close contacts). MCC of death: pneumonia. Associations: seizures. Infants can present ONLY with apnea . One third of adults do not have the "whooping" either
What is the appropriate treatment for Pertussis?	Azithromycin = first line. No Clarythro/Erythro for infants < 1mo (inc. risk of pyloric stenosis). Alt: TMP-SMX. Treat close contacts, update Tdap/DTaP
Dx: Bleeding from trach site weeks after placement	Tracheo-innominate fistula; SSx: often smaller sentinel bleed weeks after placement followed by massive hemorrhage. Rx: intubate to compress bleeding through trach site; can also hyperinflate cuff, compress with finger
Dx: Child alone in room starts coughing	Inhaled foreign body
What is the appropriate treatment for suspected hereditary angioedema?	FFP, Icatibant; normal allergic rxn meds don't work

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What is the general approach to treatment of patients with pneumonia and the following dispos: Outpatient, Inpatient, ICU	<u>Outpatient/CAP</u> : <i>S. Pneumo</i> or <i>atypicals</i> ; Rx: Doxy or Azithro (healthy), respiratory tract fluoroquinolone (RTF) (comorbidity). <u>Inpatient</u> : add Gram neg. coverage. Rx: Combo tx (CTX + Azithro) > RTF. <u>ICU</u> : antipneumococcal β -lactam (ceftriaxone or cefotaxime) + either azithromycin or an RTF \pm MRSA coverage (Vanc or Linezolid)
Most common cause of community acquired pneumonia? Tx?	<i>Strep pneumoniae</i> , Gram positive lancet-shaped encapsulated diplococcus; Key features: rusty sputum, lobar pneumonia. SSx: acute onset + rigors, follows URI or influenza. Rx: CTX + Azithro
Dx: Pneumonia + history of Cystic Fibrosis	<i>Pseudomonas</i> , Gram negative rod; SSx: green sputum, multilobar pneumonia. Rx: antipseudomonal antibiotics (e.g. cefepime)
MCC of pneumonia in young children with cystic fibrosis?	Staph aureus, Haemophilus influenzae. By the time they are 18 yo, 80% of pts with CF will be colonized by pseudomonas though.
Dx: Pneumonia + alcoholic + currant jelly sputum	<i>Klebsiella</i> , encapsulated Gram negative bacillus in pairs. Key features: higher risk in alcoholics, diabetics, nursing homes. Dx: XR \pm RUL infiltrate + "bulging" fissure, air-fluid level. Rx: cephalosporin (e.g. CTX) + Gent/Amikacin
Dx: Pneumonia after Influenza	<i>Staph aureus</i> , Gram positive cocci in clusters. Associations: IVDU, hospitalization. SSx: appear sick, follows influenza often. Dx: XR \pm patchy/multilobar/cavitary/abscess.
Dx: Infection in a patient with an indwelling urinary catheter	<i>Pseudomonas</i> ; Human + plastic (ETT/Foley/Trach) = <i>Pseudomonas</i>
What are risk factors for Health Care Associated Pneumonia?	Nursing homes, hospitalization in the last 90 days, HD, home IV abx; more likely to have drug-resistant bugs and thus require broad coverage including <i>Pseudomonas</i> & MRSA. Note : HCAP is now defunct, replaced by Hospital Acquired Pneumonia. Know HCAP criteria (test lags behind practice a few years)
Dx: Immunocompromised with marked dyspnea and hypoxemia	PCP pneumonia. HIV with CD4 < 200. Dx: \uparrow LDH, CXR: "bat wing" sign. Rx: TMP-SMX (first line), Alt: IV Pentamidine (\pm hypoglycemia, hypotension), PO Dapsone (\pm methemoglobinemia). Add steroids if PaO ₂ < 70 (~SaO ₂ < 93%) or A-a gradient > 35
Dx: Mild pneumonia symptoms and ear pain	<i>Mycoplasma pneumoniae</i> . Atypical "walking" pneumonia. Young adults. Extrapulmonary sx: bullous myringitis, Faget sign (fever + relative bradycardia). Dx: CXR shows diffuse interstitial pattern, +cold agglutinin test. Rx: Azithromycin. Note : bullous myringitis is most commonly caused by <i>S. Pneumo</i> , but the test often associates with Mycoplasma
What complications are associated with <i>Mycoplasma pneumoniae</i> ?	Aseptic meningitis, hemolytic anemia, Guillain-Barré, erythema multiforme
Dx: Pneumonia + Gram Positive Rods	Pulmonary anthrax. Flu like illness progressive to pneumonia. Widened mediastinum on xray. Rx: cipro.
Dx: Infant with staccato cough	<i>Chlamydia pneumoniae</i>
Dx: Pneumonia and headache in a bird owner	Psittacosis, <i>Chlamydia psittaci</i> . Tx: Doxycycline (or Tetracycline)
Most common viral pneumonia in adults?	Influenza

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Dx and Tx: ARDS after exposure to rodents	Hanta Virus; supportive care only
Dx and Tx: Pneumonia + diarrhea + hyponatremia	Legionnaire's Disease (<i>Legionella pneumophila</i> , a Gram negative rod). Associated with aerosolized water (e.g. nursing homes, hospitals) or air travel. SSx: high fever, relative bradycardia, GI sx (n/v, diarrhea), neuro sx (confusion, seizure). Labs: hypoNa. Dx: CXR shows patchy alveolar infiltrates, urinary antigen testing. Rx: Azithro or fluoroquinolone (severe dz)
Dx and Tx: Pneumonia + sheep	Q fever. <i>Coxiella burnetii</i> , obligate intracellular Gram negative bacterium. Labs: LFT abnormalities + proteinuria Rx: tetracycline or doxycycline
Dx and Tx: Pneumonia + high temp + hunter/butcher	Tularemia. <i>Francisella tularensis</i> , a Gram negative coccobacillus. Rx: Streptomycin.
Dx and Tx: Pneumonia in alcoholic who passed out/vomiting	Aspiration pneumonia, CXR: RLL or RUL. Rx: broad spectrum antibiotics (need Gram negative, anaerobes coverage)
What pathogen is associated with bullous myringitis accompanying pneumonia?	Classically <i>Mycoplasma pneumoniae</i> ; newer studies suggest it is actually caused by <i>Strep pneumoniae</i> .
What is the underlying pathologic process in emphysema?	Irreversible destruction of alveolar septae. Associated with smoking, certain jobs (e.g. ship-building), CF, <i>alpha</i> -1-antitrypsin
What are the typical CXR findings in a patient with COPD?	Hyperinflation, flat diaphragms, blebs/bullae
What is required for a diagnosis of chronic bronchitis?	A cough most days of the month, for 3 months each year, for at least 2 consecutive years
Differential for acute decompensation in COPD patient?	Pneumothorax (high risk), mucous plug, PE, MAT/arrhythmia, pneumonia
Review the approach to mechanical ventilation of a COPD patient	Avoid barotrauma, minimize auto-PEEP by using lower RR (8-10/min), lower TV (5cc/kg), prolong expiration time, and tolerate respiratory acidosis/permmissive hypercapnea
Review the approach to supplemental O2 in a COPD patient	They are chronically hypoxic and hypercapnic, so respiratory drive relies on hypoxemia. Tolerate sats to low 90s, limit supplemental O2 and target SpO2 to 88-92% unless you've made the decision to intubate and are preoxygenating
What are the typical pulmonary function test abnormalities in Asthma and COPD patients?	↓ FEV1, ↓FEV1/FVC, ↓ PEFr
Review the approach to treatment of COPD patient	Supplemental O2 prn for target SpO2 88-92%, antibiotics for change in sputum or obvious infection, steroids, albuterol/ipratropium, BiPAP, intubate if all else fails
What are the most likely causes/triggers for COPD and asthma?	<u>COPD</u> : infection (virus) = most common cause of exacerbation. <u>Asthma</u> : more likely 2/2 meds, exercise, allergens. Always get CXR for COPD eval on test.

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What is the underlying pathologic process in asthma?	Triad of lower airway inflammation + bronchoconstriction due to hyperreactivity + reversible airflow obstruction
Dx: Persistent cough in patient with atopic history	Cough-variant asthma
Management of exercise-induced asthma	Albuterol treatment before, during and after exercise
What pulmonary function test can be used to monitor asthma severity/treatment response?	Peak expiratory flow rate (PEFR)
Review the approach to treatment of asthma patient	Ok to supplement O2 (not as dependent on hypoxic drive as COPD pts), steroids, albuterol/ipratropium, Mg if sick, Epi if sick, BiPAP, intubate if all else fails
What is the mechanism of albuterol in treatment of asthma?	Beta-2 agonist. Causes bronchodilation by increasing cAMP → smooth muscle relaxation, affects smaller peripheral airways
What is the mechanism of ipratropium in treatment of asthma?	Anticholinergic. Causes bronchodilation by decreasing cGMP → inhibiting vagally-mediated bronchoconstriction in larger airways
What is the mechanism of systemic steroids in treatment of asthma?	Limits recruitment and activation of inflammatory cells, decreases leukotriene and prostaglandin production. note that these effects are delayed (onset 1-2hr, peak 24hr).
Review the approach to intubation and mechanical ventilation of an asthma patient	<u>Intubation indications</u> : cardiac or resp. arrest, physical exhaustion, AMS. <u>Intubation</u> : Rx IVF before prior (PPV decreases preload and may cause hypotension). RSI: consider Ketamine. <u>Ventilation</u> : Permissive hypercapnea (can tolerate acidosis). Goals: avoid barotrauma, minimize auto-PEEP. Settings: lower RR (8-12/min), lower TV (6cc/kg), PEEP (0-5), prolonged expiration time
What is the best measurement of airway pressure in pt on vent?	Plateau pressure (i.e. alveolar pressure). Peak pressure measures flow resistance in larger airways. Keep plateau pressure < 30 in asthma/COPD
Dx and Tx: PEA arrest after intubation of asthma patient	Tension pneumothorax; disconnect from vent, squeeze chest, place bilateral chest tubes, give IVF.
Dx and Tx of Bronchiectasis	Permanent destruction and dilatation of bronchi 2/2 recurrent infections, cystic fibrosis. SSx: chronic foul-smelling sputum, hemoptysis, recurrent pna. Dx: CXR ± honeycombing, "tram-track" markings, CT to dx (dilated, tortuous airways). Rx: abx (cover <i>Pseudomonas</i>), albuterol.
Dx: Child or teenager with pancreatitis	Suspect cystic fibrosis (GI variant)
What is the pathophysiology of cystic fibrosis?	Autosomal recessive. Leads to mutations affecting Na/Cl exchange channel. Results in abnormally viscous mucous secretions. Multiple organ systems affected: recurrent pulmonary infxn (<i>Pseudomonas</i>), meconium ileus & intussusception, pancreatic insufficiency & pancreatitis
How is cystic fibrosis diagnosed?	Elevated quantitative sweat chloride test or DNA testing

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Dx: Fever, sick, possible ruptured esophagus	Mediastinitis (2/2 Boerhaave syndrome)
Most common cause of pleural effusion in elderly patients	Malignant effusion
What is the pathophysiologic difference between exudative and transudative effusions, and how are each of these managed?	<u>Exudative</u> : damaged capillaries leak thick fluid usually 2/2 inflammation, effusion must be removed. <u>Transudative</u> : intact capillaries leak thin fluid 2/2 increased hydrostatic or decreased oncotic pressure, tx underlying cause.
What are common causes of exudative and transudative pleural effusions?	<u>Exudative</u> : pulm infxns (pneumonia) > malignancy (MCC of massive effusions), PE (MCC of isolated, unexplained effusion). <u>Transudative</u> : CHF (MCC in US, West), renal failure, liver failure.
Review Light's Criteria to distinguish exudative from transudative pleural effusion	Exudative if 1) fluid protein : serum protein >0.5, 2) fluid LDH : serum LDH >0.6, 3) fluid LDH > 2/3 upper limit of normal for serum LDH. In other words, exudative if high protein and high LDH.
Dx: PCP pneumonia with sudden worsening SOB	Pneumothorax (strong association PCP and PTX)
What is the most sensitive bedside test for possible pneumothorax?	Bedside ultrasound (better than CXR); look for "seashore sign" on M mode, "comet tails" with lung sliding on 2D. PTX will show "barcode sign" on M mode, and the absence of "comet tails" indicates no lung sliding.
What are possible CXR findings in a supine patient with pneumothorax?	"Deep sulcus" sign
Treatment of tension pneumothorax	Immediate needle decompression (2nd intercostal space at midclavicular line) followed by chest tube. DO NOT take the time to get CXR.
Dx and Tx for an empyema	Empyema = pus in pleural space. Dx: thoracentesis (pH < 7.20, WBC > 50k, glucose < 60, pus). Rx: ultimately requires fluid drainage (thoracentesis v. tube thoracostomy v. thoracotomy), long-term antibiotics
What is the most common cause of hemoptysis in the US and abroad?	US: bronchitis; Worldwide: TB
What defines massive hemoptysis?	≥ 50 mL single expectorant or ≥ 500 mL / 24-hr
What is the most common cause of death in massive hemoptysis?	Hypoxia/asphyxiation (not blood loss); early airway management is key
What is the appropriate treatment for an unstable patient with massive hemoptysis?	Early intubation, mainstem to ventilate good side if possible, and position patient with bleeding side down so the blood follows gravity and stays in the impaired lung; blood transfusion is less important. After initial stabilization the pt will need bronch or angio to ID source of bleeding.

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Dx and Tx: Young person, massive hemoptysis, bilateral whiteout on CXR	Diffuse Alveolar Hemorrhage. Cause is usually inflammatory or autoimmune. Rx: high dose steroids, supportive care
Dx and Tx: AMS + vomiting + patchy dependent consolidation	Aspiration pneumonitis, chemical pulmonary inflammation. Risk factors: ALOC (ETOH, sz) neuro d/o, dysphagia. Rx: supportive, monitor for development of aspiration pneumonia, no antibiotics unless true pna.
Dx and Tx: Alcoholic with foul breath, cough and CXR with air fluid level	Lung abscess. Polymicrobial: anaerobes = MCC (e.g. Peptostreptococcus), <i>Staph</i> . CXR: consolidation + cavity/air-fluid level; aerobes/TB (upper lobe), anaerobes (lower lobe). Rx: antibiotics (ampicillin+sulbactam, carbapenem), surgery if severe.
What are potential CXR findings in primary, reactivation and miliary TB?	<u>Primary</u> : lower lobes, looks like pna. children: pronounced hilar adenopathy / <u>elderly</u> : isolated pleural effusion. <u>Reactivation</u> : upper lobe granuloma ± cavitation. <u>Miliary</u> : scattered nodules (millet seeds) throughout lung fields
How is TB diagnosed?	<u>Sputum stain</u> (for AFB, suggestive, faster); <u>Sputum cx</u> (gold standard, confirmatory test, takes weeks). <u>Quantiferon Gold</u> (possible alternative, expensive). <u>Tuberculin skin testing</u> (i.e. PPD, used to screen, but positive tests require followup with CXR, Quant Gold, etc.).
What defines a positive TB skin test?	Assessed for induration (not erythema). <u>≥5mm</u> : HIV, immunosuppressed (e.g. organ transplant), close contact with active TB, abnormal CXR. <u>≥10mm</u> : h/o IVDA, exposure to high risk setting (immigrant from TB-endemic area, jail, healthcare worker), children <4yo. <u>≥15mm</u> : everyone else
What is the treatment for latent and active TB?	<u>Latent</u> : Isoniazid (+B6) x6-9 mo. <u>Active</u> : Rifampin, Isoniazid, Pyrazinamide, Ethambutol (alt Streptomycin) x 9-12 mo
What are potential side effects of RIPE therapy?	<u>Rifampin</u> : orange body fluids, hepatitis low platelets. <u>Isoniazid</u> : neuropathy (B6 deficiency), seizures (Rx B6 for refractory cases), hepatitis. <u>Pyrazinamide</u> : hepatitis, high uric acid → gout, teratogenic. <u>Ethambutol</u> : optic neuritis, red-green blindness. <u>Streptomycin</u> : vestibular nerve damage, renal injury (contraindicated in pregnancy → congenital deafness)
What are potential EKG changes seen with pulmonary embolism?	Sinus tach (most common), nonspecific ST-T changes, R heart strain (e.g. RAD, new RBBB, p-pulmonale, S1Q3T3 = classic but rare), precordial TW inversion
Echocardiographic signs of right heart strain?	RV dilation, RV hypokinesis, septal shift to the LEFT, tricuspid regurg, elevated pulmonary artery pressure, decreased LV filling, and impediment of LV output.
What is the most common symptom and sign of pulmonary embolism?	Dyspnea (73%) and tachypnea (54%). Other "classic" symptoms are less common, such as pleuritic pain (44%), cough (37%), hemoptysis (15%), tachycardia (24%), calf pain/swelling (44%).
What are potential CXR findings in pulmonary embolism?	CXR: nonspecific abnormalities, Hampton's hump (pleural-based wedge infarct), Westermark's sign (vascular cut-off sign)

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What is the appropriate workup for patients with clinical symptoms and multiple risk factors for DVT and PE?	<u>DVT</u> : need negative D-dimer & Doppler US (repeat Doppler US if high risk and initial study negative) to exclude. <u>PE</u> : need negative imaging (CTA or VQ scan) to exclude
When should thrombolytics be considered in a patient with PE?	<u>Hemodynamically unstable</u> : hypotension/shock, cardiac arrest. <u>Hemodynamically stable</u> : RV enlargement or dysfunction (EKG, TTE, or CT proven), extensive clot burden, severe hypoxemia, RA/RV thrombus, PFO
Dx: IVDA + multiple infiltrates on CXR	Septic pulmonary emboli (raising concern for endocarditis)
Dx and Tx of pulmonary arterial hypertension	SSx: SOB (most common), chest pain, hypoxia; Dx: TTE, cath, CT; XR with enlarged pulmonary arteries, EKG with R heart strain. Rx: vasodilators (prostacyclins like Remodulin)
Potential cause of acute decompensation in patient with pulmonary hypertension?	PE or IV pump failure (if on continuous PGA infusion and infusion stops)
Potential cause of acute decompensation in patient with pulmonary fibrosis?	Progression of disease vs. acute pneumonia
Dx and Tx: Non-caseating granulomas in lungs with bilateral hilar adenopathy	Sarcoidosis, associated with erythema nodosum. Rx: steroids
What are the different types of Pneumoconioses?	<u>Pneumoconiosis</u> : lung dz caused by inhalation of organic or inorganic dust. SSx: SOB, cough (non-productive), hypoxia. Dx: CXR shows interstitial fibrosis. <u>Asbestosis</u> : shipping, roofing, plumbing (from the roof, but affects the base [lower lobes]); <u>Berylliosis</u> : aerospace, fluorescent bulbs; <u>Byssinosis</u> : cotton; <u>Silicosis</u> : foundries, sandblasting, mines; <u>Coal worker's lung</u> : coal, Silica & coal: from the base (earth), but affect the roof (upper lobes); <u>Siderosis</u> : arc welding (iron); <u>Stannosis</u> : tin welding
What labs are classically abnormal in patients with sarcoidosis?	Hypercalcemia, high ACE
What are the criteria for diagnosis of ARDS?	1) Acute onset (sx within 1wk of causative insult), 2) bilateral opacities on CXR/CT (pulm edema), 3) no cardiac cause, 4) impaired O2 exchange (PaO2/FiO2 < 300)
What are the clinical features of ARDS?	Poor lung compliance, pulmonary edema, severe hypoxemia unresponsive to supplemental O2; Causes: shock states (gram neg. sepsis = MC), trauma, almost anything EXCEPT heart failure.
What is the approach to "lung protection" in ventilated ARDS patients?	Low tidal volume (4-6cc/kg), high PEEP (5-20), permissive hypercapnea but can increase RR as needed, supplemental O2 (PEEP & FiO2 should be titrated up together [ARDSnet trial])
What is the expected PCWP in ARDS vs CHF?	ARDS: low/normal PCWP; CHF: high PCWP

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Dx: Intermittent cough and episodic diaphoresis, XR with lung mass	Pulmonary carcinoid
Dx: Cough and ulnar neuropathy	Pancoast tumor, XR with mass at lung apex, causes brachial plexus compression
What size pneumothorax can be managed with O2 and observation alone?	20% or less. Pneumothoraces resorb 1-2% per 24hrs in healthy lungs.
What is the most common airway location for foreign bodies to lodge?	SSx: cough, wheezing, dyspnea, asymptomatic (20%). <u>Adults</u> : proximal airways (75%; larynx, trachea, main bronchi: R mainstem bronchus most common). <u>Children</u> : <50% are proximal (main bronchi branch from the trachea at more equal angles, lower airway foreign bodies are equally likely to affect the right and left lung fields in children)
What mechanism of hypoxemia will not improve with O2 supplementation?	Right to left shunt
What mechanisms of hypoxemia cause an increase in the A-a gradient (>15)?	Right to left shunt, Diffusion impairment, V-Q mismatch
Treatment of refractory hiccups?	Thorazine, always investigate for serious cause of hiccups prior to symptomatic treatment.

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HEENT	
Bizz	Buzz
Dx and Treatment of blepharitis	Inflammation of the eye lid. Tx: wash with gentle soap, topical antibiotics (<i>Strep/Staph</i>)
Dx and Tx: hordeolum vs. chalazion	<u>Hordeolum</u> : acute painful blocked gland of Zeis at lid margin (at or near eyelash follicle). Rx: warm compresses, abx if concurrent with preseptal cellulitis. <u>Chalazion</u> : chronic or gradual-onset nontender granuloma due to blockage of meibomian gland, found above eyelash on upper lid. Rx: warm compresses, ophtho excision
Hordeolum vs. stye	They are the same thing
Dx and Tx: Iritis, Uveitis, Choroiditis	Inflammation of iris, ciliary body and choroid, respectively. SSx: painful red eye, photophobia; exam shows decreased visual acuity, cell and flare, ciliary flush. Tx: Ophtho consult, dilate, steroids, pain meds.
Dx and Tx: Dacrocystitis	Infection of the nasolacrimal (tear duct; inframedial) system (<i>S. aureus</i>). SSx: purulent discharge, possible adjacent cellulitis. Rx: warm compresses, antibiotics.
Dx and Tx: Corneal dendrites	HSV keratitis; Rx: Ophtho consult, topical trifluridine unless complicated
Dx and Tx: Bacterial conjunctivitis + contact lens wearer	Risk for <i>Pseudomonas</i> infection, Rx: tobramycin drops (↑ resistance to cipro)
Dx: Metal worker + eye pain	Intraocular foreign body (look for teardrop pupil, Seidel's sign to r/o globe rupture); Rust rings removed in 24-48 hours by Ophtho
Dx: skier or welder + eye pain	UV keratitis. bilateral decreased visual acuity, pain, and redness. Exam shows multiple punctate lesions. Rx: cycloplegics
Dx: hyphema?	Hyphema: bleeding in the anterior chamber of the eye. Can be traumatic. Tx: consult ophtho, bedrest, HOB elevation, pain meds, cycloplegics. More urgent consult for sickle cell disease.
Primary concern after hyphema? Treatment?	Rebleed (most common complication), risk for glaucoma.
Dx and Tx of endophthalmitis	Infection of anterior, posterior and vitreous chambers of the eye. Iatrogenic (cataract surgery = MCC in US), trauma. SSx: severe pain & visual impairment. Dx: exam with decreased visual acuity, injected conjunctiva, chemosis, possible hypopyon. Tx: ophtho consult, intraocular + systemic antibiotics
Laceration close to the medial canthus?	High risk for lacrimal duct injury. Fluorescein staining will show stain extruding from the laceration. This repair should be done by oculoplastics.
Treatment of acute angle closure glaucoma	First decreased the intraocular pressure, next constrict to the pupil to promote drainage. ↓ <u>aqueous production</u> : α-agonist (apraclonidine), β-blocker (timolol), carbonic anhydrase inhibitor (acetazolamide); ↑ <u>outflow</u> : pilocarpine (miotic).

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Dx: Sudden painless unilateral vision loss, retina with "box-cars" or "cherry-red spot"	Central retinal artery occlusion
Dx: Sudden painless unilateral vision loss, retina with "blood and thunder" appearance (dilated retinal veins, diffuse hemorrhage, cotton wool spots)	Central retinal vein occlusion, increased risk with chronic glaucoma
Dx: painless unilateral loss of vision associated with floaters at first and then visual field cuts. Visual acuity is normal (or at their baseline). Nothing seen on slit lamp.	Retinal detachment - clinical diagnosis based on hx. Pt should see ophtho in the ER or w/in 24hr. Classic descriptor: "curtain being drawn over my eye" or "floaters, flashes of light"
Dx: painful red eye, decreased visual acuity, ciliary flush, "cell and flare" on slit lamp.	(Traumatic) iritis or anterior uveitis. Tx: Ophtho consult or follow up w/in 24hrs, dilate (cycloplegics e.g. cyclopentolate, tropicamide), pain meds
Dx: Dizzy + vertical, multidirectional, or non-fatigable nystagmus	Central vertigo
Ddx for gingival hyperplasia	Acute necrotizing ulcerative gingivitis, HIV, phenytoin toxicity, acute leukemia
White plaques on oral mucosa, can be scraped off	Candidia, associated with immunocompromise, abx use
Name Centor criteria for acute bacterial pharyngitis	Fever, tender anterior LAD, no cough, tonsillar exudates; 4/4 → empiric abx, 3/4 → culture/rapid strep
What medication is most likely to improve symptoms of viral pharyngitis?	Dexamethasone, NSAIDs are also a mainstay of tx
Dx and Tx of PTA?	Common complication of acute tonsillitis. <i>Strep</i> (GAS) = most common. Most common deep facial infxn in adults. SSx: sore throat, odynophagia, muffled voice, referred otalgia. Exam: trismus, deviation of soft palate + uvula. Rx: needle aspiration, abx (PCN + metronidazole v clinda), ENT followup
Dx and Tx: fever, sore throat, brawny neck edema, tongue elevation, dysphagia, drooling?	Ludwig's Angina. Dental infxn = MCC, immunodeficiency. Rx: airway mgmt, ENT consult, broad spectrum abx. Sudden asphyxiation (laryngospasm) = MCC of death
Identify classifications of dental fractures and appropriate management	<u>Ellis I</u> : fx of enamel . Exam: white, painless. Rx: smooth rough edges, dental follow-up. <u>Ellis II</u> : fx thru dentin & enamel. Exam: yellow, painful. Rx: smooth rough edges, apply calcium hydroxy paste, dental follow-up. <u>Ellis III</u> : fx thru pulp , dentin & enamel. Exam: pink/red, painful. Rx: dental emergency, cover with moist cotton & dental foil, abx (PCN), and get dental consultation or arrange dental f/u within 24hr for root canal & pulpectomy

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Dx and Tx of dry socket (alveolar osteitis)	Localized osteomyelitis. Due to loss of protective clot. SSx: severe pain 3-5d after dental extraction. Exam exposed bone w/o clot. Rx: irrigate with saline, iodoform guaze, eugenol (oil of clove), abx if needed (signs of infection), PAIN CONTROL and oral surgery referral
Dx and Tx of CMV retinitis	CD4 <50 = AIDS defining illness. SSx: ↓ visual acuity, floaters/visual field cuts, photophobia. Exam: white fluffy perivascular lesions with hemorrhage. Rx: IV gancyclovir
Dx of vitreous v. retinal detachment on ultrasound	BOTH vitreous & retinal detachments show serpiginous structure within the globe. <u>Vitreous detachment</u> : can cross over the optic nerve; <u>retinal detachment</u> : will NEVER cross the optic nerve since it is made of nerve fibers, which converge into the optic nerve
What is an APD? (Afferent pupillary defect?)	A sign that indicates a lesion of the retina or optic nerve. It is detected by moving a light from the better functioning eye to the eye with poor vision and observing dilation of the pupil
4 causes of afferent pupillary defect (APD)	CRAO, CRVO, optic neuritis, and retrobulbar neuritis.
Dx and Tx: Monocular vision loss, worse centrally, afferent pupillary defect, pain with EOM, diminished color vision	Optic neuritis. Causes: Idiopathic > MS. Also toxicological and infectious. Rx: Consult neurology & ophtho, IV steroids, MR to eval for MS. Seen with: methanol toxicity and ethambutol toxicity. Herpes Zoster.
Treatment of CRAO	Orbital massage to disrupt clot, ↓ IOP (acetazolamide, mannitol, timolol), vasodilators (nitro)
Pinguecula vs. pterygium	<u>Pinguecula</u> : degenerative eye lesion 2/2 chronic inflammation from wind and UV light. Exam: yellow, raised, fleshy conjunctival mass (lateral). Rx: none. <u>Pterygium</u> : slow growing thickening of conjunctiva 2/2 wind, sand, dust. Exam: vascular triangular mass in "bat wing" shape (medial). Rx: surgery if interferes with vision.
What should be the approach to stopping anterior epistaxis?	Direct pressure, vasoconstrictors (oxymetaxoline/Afrin, pheynelephrine, cocaine), balloon/tampon devices (be sure to soak with water not saline), Note: use of abx is controversial. Send home with ENT f/u in 2-3 days.
What are the most common sources of anterior and posterior epistaxis?	Kiesselbach plexus (anterior) and sphenopalatine artery (posterior)
What is the appropriate treatment and disposition for patients with posterior nasal packing?	Prophylactic antibiotics + admit to ICU/monitored bed due to risk of vagally-mediated bradycardia and airway compromise
Most common site of sialoadenitis? Tx?	Submandibular gland (Wharton duct). Rx: milk stone, sialogogues
Dx and Tx: sudden onset vertigo with head position	Benign Paroxysmal Positional Vertigo (BPPV). Rx: Epley, meclizine (only for continuous sx, not episodic sx)
Recurrent bouts of vertigo + hearing loss + tinnitus?	Meniere disease; aka "Idiopathic endolymphatic hydrops" due to increased endolymph w/in the inner ear. Rx: treat vertigo, refer to ENT. Hearing loss and tinnitus are often refractory to treatment. **peripheral vertigo**

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Severe vertigo + URI	Vestibular neuritis - Rx: supportive. Resolves on it's own.
Severe vertigo + AOM (plus or minus hearing loss)	Labrynthitis - caused by an infection of the inner ear. It can be caused by measles or mumps viral infection or can be due to an extension of acute bacterial otitis media, which causes suppurative labyrinthitis. Suppurative labyrinthitis should be treated with intravenous antibiotics and ENT referral.
Differentiate labrynthitis from Ménière dz	<u>Labrynthitis</u> : vestibular neuritis + unilateral hearing loss, assoc. URI. SSx: vertigo (NOT recurrent) + hearing loss + unstable gait. Exam: nystagmus, +head impulse. Rx: self limited. <u>Ménière</u> : due to inc. endolymph. SSx: episodic vertigo + hearing loss + tinnitus. Rx: avoid triggers, antihistamines, diuretics, benzos, surgery (refractory cases)
Dx if positive head impulse test (i.e. presence of corrective saccade) in a pt with continuous vertigo	Vestibular neuritis / labyrinthitis
Dx and Tx of perichondritis	Infection of the cartilage. Presents: Swollen, warm, tender, erythematous auricle. No TM involvement, no earlobe involvement. Presents after ear surgery, trauma or piercing the upper ear. <i>Pseudomonas</i> , Staph, Strep spp. Rx: fluoroquinolone (e.g. cipro), if severe admit for IV coverage.
Dx and Tx of acute otitis media	Infant or young child. Viral (RSV) > bacterial (<i>Strep. Pneumo</i>). SSx: ear pain, fever, URI symptoms. Exam: TM bulging/erythema & decreased mobility of TM (most sensitive). Rx: amoxicillin; recurrent/persistent: Augmentin, others. Complications: hearing loss, perforation (add abx in suspension), facial nerve paralysis (needs myringotomy)
Dx and Tx of acute mastoiditis	Bacterial infection of mastoid air cells. Direct extension from AOM = MC mechanism. <i>Strep. Pneumo</i> = MCC. SSx: postauricular erythema & tenderness, protrusion of the auricle. Dx: CT temporal bone. Rx: abx (CTX), surgical drainage. Complications: osteo, intracranial infxns, venous sinus thrombosis
Dx and Tx of necrotizing (malignant) otitis externa	Necrotizing infection of auditory canal + skull base. Elderly + DM2. <i>Pseudomonas</i> = MCC. SSx: otorrhea, otalgia, severe/persistent pain, CN VII palsy. Dx: CT temporal bone. Rx: cipro (outpt), antipseudomonal β -lactam + aminoglycoside (inpt) if toxic, CN palsies. Complication: CNVII palsy, intracranial infxns.
Sudden pain + decreased hearing after ear irrigation	TM perforation. Rx: pain meds, keep ear dry, antibiotics only if concurrent infection, ENT outpt follow-up 1-2wks. Overall, infxn = MCC of TM perf
Dx and Tx: pain/swelling/ttp over parotid gland, fever, trismus, dysphagia	Suppurative (bacterial) parotitis. <i>Staph aureus</i> = MCC. Rx: abx (Ampicillin-sulbactam)
Dx and Tx of preseptal cellulitis	Infection of the anterior portion of the eye (NOT orbital structures). <i>Staph/Strep</i> spp. SSx: Eyelid swelling \pm eye pain. LACKS pain with EOM or proptosis. Dx: clinical, CT if concerned for orbital cellulitis. Rx: amoxicillin-clavulanate, outpt ophtho follow-up

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Dx and Tx of orbital cellulitis	Infection of the contents of the orbit (posterior to orbital septum). Children > adults. Bacterial rhinosinusitis = MCC. SSx: eyelid swelling, deep eye pain, pain + limitation with eye movements, proptosis. Rx: broad spectrum abx (Vanc + Zosyn), ophtho consult, admit. Complications: vision loss, cavernous sinus thrombosis, meningitis
Name the structures that run through the cavernous sinus	CN III, IV, VI, V1, V2, internal carotid artery. Isolated CN VI (abducens) palsy = MC CN palsy.
Dx and Tx of cavernous sinus thrombosis	Classically follows acute bacterial sinusitis. <i>Staph</i> = MCC. SSx: headache (MC sx) + fever + CN palsies (abducens palsy is classic which means the eye looks in towards the nose) + periorbital edema. Dx: MRV. Rx: IV abx (nafcillin, ceftriaxone, and metronidazole), ENT consult, ICU admit. Anticoagulation is controversial.
Cause of brisk bleeding after recent tracheostomy placement	Tracheoinnominate artery fistula (TIF)
Recurrent bouts of vertigo + hearing loss + tinnitus?	Meniere disease; aka "Idiopathic endolymphatic hydrops" due to increased endolymph w/in the inner ear. Rx: treat vertigo, refer to ENT. Hearing loss and tinnitus are often refractory to treatment. **peripheral vertigo**
Chemical Injury to the eye?	Alkali injury is worse (leads to liquefaction necrosis). Tx: check pH, irrigate, check pH, normal pH 7.4
Globe Rupture?	Teardrop pupil, flattened anterior chamber, Seidel's sign is present. Rx: DO NOT CHECK PRESSURES, give antibiotics, CT scan, cover with eye patch and call ophtho in.

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Neurology	
Bizz	Buzz
Dx: Eye down and out	CN III palsy; consider CVA, uncal herniation if with blown pupil
Dx: Bilateral internuclear ophthalmoplegia	Combined 3rd & 6th nerve palsy; usually multiple sclerosis
Dx: Urinary incontinence, AMS, ataxia	Normal Pressure Hydrocephalus; "wet, wacky, wobbly"; also may be shunt malfunction
Dx: Young obese woman, headaches, vision changes, CN VI palsy	Idiopathic intracranial hypertension (pseudotumor cerebri); Dx: CT normal, LP: diagnostic (↑ opening pressure) & therapeutic (drain to <20cm H2O); Complication: permanent vision loss. Note, they may report pressure in mm so elevated would be 200mm.
Dx and Tx: Neuroleptic Malignant Syndrome	Neuroleptic use (dopaminergic/antipsychotic). SSx: autonomic instability (hyperthermia), "LEAD PIPE" MUSCLE RIGIDITY, AMS; no clonus/DTR changes. Rx: supportive (IVF, benzos, cooling), ± dantrolene (direct skeletal-muscle relaxant), ± bromocriptine (dopamine agonist)
Dx and Tx: Serotonin Syndrome	Serotonergic agent use (combo 2+ SSRIs) or multi-drug overdose. SSx: autonomic instability (hyperthermia), CLONUS & HYPERREFLEXIA (LE>UE), AMS. Rx: supportive (IVF, benzos, cooling), ± cyproheptadine (antihistamine w/ antiserotonergic properties)
Dx: CNS mass lesion in AIDS	1) Toxo (multiple ring-enhancing lesions w/ edema), 2) CNS lymphoma (hyperdense, round enhancing lesions)
Treatment of intraparenchymal hemorrhage	BRAINS': BP control (~160/90), R verse coagulopathy, A irway mgmt, I CP control if herniating (hyperventilate, HOB 30, mannitol; N eurosurg consult (craniotomy for cerebellar vs. ventriculostomy), S eizure ppx (controversial)
Dx: Contralateral hemiparesis/hemiplegia, Contralateral sensory loss, Homonymous hemianopia	Putamen hemorrhage. Most common type of ICH.
Dx: Ataxia, headache/vomiting, gaze palsy, facial weakness	Cerebellar hemorrhage
Dx: hemiparesis or hemisensory loss, upward gaze palsy, miotic pupils	Thalamic hemorrhage
Dx: deep coma, total paralysis, pinpoint pupils	Pontine hemorrhage
Classic symptoms for ACA vs. MCA vs. PCA strokes	<u>ACA</u> : frontal lobe dysfxn, apraxia, contralat paralysis (lower > upper). <u>MCA</u> : contralat paralysis (upper > lower), ipsilat hemianopsia, aphasia. <u>PCA</u> : LOC, nausea/vomiting, CN dysfct, ataxia, visual agnosia. Rule out hypoglycemia! Dx: CT shows loss of grey-white interface, acute hypodensity

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Pt w/ unilateral CN deficits, contralateral hemiparesis and hemisensory loss?	This brain stem stroke. This presentation is known as “Crossed signs.”
Treatment of sickle cell pt with CVA	Exchange transfusion
Indications for tPA for CVA	Age ≥18, dx of ischemic stroke + neuro deficits, symptoms < 4.5hr, CTH negative for bleed, no clear reversible cause
Absolute contraindications for tPA for CVA	Ischemic stroke, neurosurgery, or head trauma within 3mo; ANY ICH (current or previously); possible SAH; known intracranial neoplasm/AVM/aneurysm, BP >185/110 after reduction attempted; possible reversible cause; active bleeding or coagulopathy (Plt <100k, INR >1.7, PT>15s), CT shows multilobar infarcts, glucose <50
Relative contraindications to tPA	Pregnancy, sz at onset with postictal period, major surgery in last 14d, GI or GU bleeding in last 21d, MI in last 3mo
What is the dosing for tPA in ischemic stroke?	0.9 mg/kg (up to 90 mg) with 10% of the dose given as a bolus and the rest of the dose given as an infusion over 1 hour
Major difference between lacunar and cortical infarcts	<u>Cortical</u> : large artery, cortical dysfxn (aphasia, neglect, ALOC), motor AND sensory sx, deficits contralateral side. <u>Lacunar</u> : small artery, pure motor OR sensory sx
Neglect, or “hemi-inattention” typically indicates stroke where?	Results from infarction in the parietal lobe in the non-dominant hemisphere. This is the right hemisphere for most people.
Dx and Tx: transient episode of slurred speech and unilateral arm weakness, now resolved	Transient Ischemic Attack (TIA). Transient episode of neurological dysfunction without acute infarction. 10% of TIA patients will have a stroke within 90 days. Rx: Aspirin + dipyridamole or clopidogrel monotherapy
How to use ABCD2 for TIA dispo	Predicts likelihood of subsequent stroke within 2 days. <u>A</u> ge >60 (1), <u>B</u> P >140/90 (1), <u>C</u> linical features: unilateral focal weakness (2) speech disturbance w/o weakness (1), <u>D</u> uration of symptoms: >60min (2), 10-59min (1), <10 (0), <u>D</u> iabetes (1). Hospitalize for score >2. Workup includes MRI/MRA head&neck, EKG/TTE. 2 Tx that saves lives: carotid endarterectomy >70% stenosis, anticoagulation for Afib or LV thrombus
Best study to diagnose venous sinus thrombosis	MR venography = gold standard
Appropriate treatment for suspected bacterial meningitis	Airway control pm, Antibiotics: CTX + Vanc ± Amp (age>50, alcoholics) ± Acyclovir, Steroids, THEN CTH (for boards don’t delay abx) followed by LP, supportive (MAP/temp control, control sz pm)
Dx and Tx: bloody and necrotizing encephalitis	HSV; Rx: IV acyclovir
Findings with UMN lesion	Spastic paralysis, ↑ DTRs, positive (upgoing) Babinski, ↑ tone
Findings with LMN lesion	Flaccid paralysis, ↓ DTRs, muscle wasting, fasciculations
What distinguishes Conus Medullaris Syndrome from Cauda Equina Syndrome?	CMS is similar to CES except it is ABOVE L1 & is an UMN lesion; CMS will ALWAYS will have positive (upgoing) Babinski (UMN lesion finding)

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Dx and Tx: Guillain-Barré Syndrome (GBS)	Most common peripheral neuropathy caused by autoimmune demyelination. SSx: preceding resp. (Mycoplasma) or GI illness (Campylobacter = MCC), progressive, ascending, symmetric motor weakness, sensory loss, ↓ DTRs, normal rectal tone. Dx: LP shows ↑ CSF protein + mild pleocytosis. Rx: plasmapheresis, IVIG, measure NIF, vital capacity (resp. monitoring)
What is the Miller Fisher variant of GBS?	Descending GBS + ataxia + CN abnormalities (bulbar sx)
Difference between GBS and tick paralysis	<u>GBS</u> : paresthesias/pain AND motor loss, post-infectious autoimmune dz. <u>TP</u> : symmetric ascending flaccid paralysis + ataxia ONLY; caused by neurotoxin in tick saliva so must remove tick
Dx and Tx: Botulism	Paralytic illness. Cause: <i>Clostridium botulinum</i> neurotoxins → blocks ACh release at neuromuscular junction → flaccid paralysis. Forms: food borne (canned foods, honey), wound, infantile (most common). SSx: floppy baby, constipation, weak cry (infants); CN/bulbar sx , dilated pupils (differentiates from myasthenia), symmetric, descending flaccid paralysis / weakness (MC finding) “botulism bottoms out”, parasympathetic blockade (dry mouth/sore throat, urinary retention). Tx: supportive care, resp. monitoring (resp. failure = MCC death), antitoxin (Rx infants: BabyBIG, Rx age>1: antoxin, abx (for wounds))
Dx and Tx: Syringomyelia	Cyst or cavity in upper spinal cord (cervical spine = MC location). SSx: headache (2/2 Chiari malformation), "cape-like" loss of pain/temp sensation to UE, preservd proprio/vib. Dx: MRI. Rx: neurosurgery consultation, monitor vs. drain.
Dx and Tx: Myasthenia gravis	Autoantibodies to nicotinic ACh receptors (25% with associated thymoma). SSx: repeated muscle use = ↑ weakness/fatigue (HALLMARK), proximal muscle weakness, ocular sx (CNIII palsy, ptosis, dysphagia, diplopia). Dx: Tensilon Test (edrophonium) or Ice Pack test: ↓ sx. Rx: Pyridostigmine. <u>Myasthenic crisis</u> : triggered by infxn, meds; resp. failure → mechanical ventilation (follow vital capacity, NIF); Rx: IVIG, plasmapheresis.
Difference between Myasthenia Gravis and Lambert-Eaton Myasthenic Syndrome?	<u>MG</u> : fatigue with repeated movement. <u>LEMS</u> : improves with repetition. Note that LEMS is often paraneoplastic, so look for underlying cancer if not already diagnosed.
Dx and Tx: Young man presents with LE paralysis, high thyroid, low K	Thyrotoxic Periodic Paralysis. Hallmark of periodic paralysis = painless weakness precipitated by heavy exercise, high carb meals. Caused by transmembrane shift of K into cells. Rx: K repletion (not aggressive) and beta blockers.
Dx and Tx of status epilepticus	Seizure lasting > 5 minutes or > 2 discrete seizures without recovery of consciousness. Causes: AED discontinuation, medication noncompliance. Rx: BZDs, phenytoin (second line), phenobarbital (third line)
Treatment of seizures related to eclampsia	Magnesium sulfate. Side effects: loss of DTRs, dysrhythmia, resp. failure

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Treatment of seizures related to INH overdose	Vitamin B6/Pyridoxine (1gm per 1gm of INH toxicity)
Describe the tests for meningismus	<u>Jolt accentuation</u> : baseline HA ↑ when the patient turns head horizontally 2-3 rotations/sec. <u>Brudzinski's sign</u> (B end the brain): flexing the neck causes the hips and knees to flex. <u>Kernig's sign</u> (extend k nees): knees/hips flexed to 90 degrees, knee extension causes pain.
What is appropriate chemoprophylaxis for a healthcare worker exposed to <i>N. meningitidis</i> ?	Rifampin 600mg BID x2d, CTX 250 mg IM x1, Cipro 500 mg PO x1
Dx and Tx: HSV Encephalitis	SSx: fever + headache + AMS; neuro deficits, psych sx. Dx: CSF with ↑ RBCs and no bacteria, usually focal at temporal lobes (may see hemorrhage on CT). Rx: IV acyclovir.
Dx and Tx: Neurocysticercosis	Pt will be immigrant from tropical areas, Mexico. Acquired by eating pork containing larval cysts of <i>Taenia solium</i> (tapeworm). SSx: new onset sz in adult. Dx: CT shows multiple ring-enhancing lesions. Rx: Albendazole. Complication: obstructive hydrocephalus
What is the most common cause of meningitis in an adult?	<i>Streptococcus pneumoniae</i>
CSF findings for viral meningitis	Opening pressure: normal. WBCs: ↑ (< 300; lymphocyte predominance), protein: ↑ (not typically > 200), glucose: normal, culture: negative
CSF findings for bacterial meningitis	Opening pressure: ↑, WBCs: ↑↑ (>1000; neutrophil predominance), protein: ↑, glucose: ↓, culture: positive
CSF findings for fungal meningitis	Opening pressure: ↑, WBCs: ↑ (but < 500, lymphocyte predominance), protein: ↑↑, glucose: normal to slightly ↓, culture: positive (fungal)
Rx for brain abscess	3rd generation cephalosporin (e.g. CTX) + anaerobic bacterial coverage (e.g. Metronidazole), Neurosurgical consultation.
In what direction would you expect nystagmus with normal caloric testing?	Tests vestibulo-ocular reflex. Intact brainstem elicits nystagmus on test. Direction of movement depends on temperature of H2O used. 'COWS': C old- O pposite, W arm- S ame.
Dx and Tx: Posterior Reversible Encephalopathy Syndrome (PRES)	SSx: HTN ER w/ neuro sx (headache, AMS, seizures, vision loss); associated with vasogenic edema of the brain, most often in BILATERAL occipital and posterior parietal lobes; associated with preeclampsia/eclampsia. Dx: mainly clinical, CT may show edema, MR is more specific. Tx: supportive
Dx: Diplopia with lateral gaze	Internuclear ophthalmoplegia, associated with MS
What diseases typically present with ascending vs descending weakness?	<u>Ascending</u> : GBS, tick paralysis. <u>Descending</u> : Botulism, myasthenia gravis, Miller Fisher variant GBS, Lambert-Eaton myasthenic syndrome.

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Compare peripheral v. central vertigo	<u>Peripheral vertigo</u> : sudden onset, short duration, severe intensity, worsened by position, unilateral nystagmus (never vertical; and it is fatigable), no neuro sx, ± auditory sx; Causes: BPPV, AOM, labyrinthitis, Meniere's dz, vestibular neuronitis, trauma. <u>Central vertigo</u> : gradual, constant sx, not positional, mild intensity, nystagmus (multidirectional, non-fatigable, non-inhibited), +neuro findings, no auditory findings; Causes: brainstem, cerebellum; admit
Dx and Tx: woman describing unilateral lower face, electric, shock-like pain, worse with chewing or burshing teeth?	Trigeminal neuralgia. Rx: Carbamazepine.
Dx and Tx of temporal arteritis	Pt will be woman >50 years old. SSx: monocular vision loss, unilateral headache, jaw claudication. Exam: temporal artery tenderness. Labs: ↑ ESR , ↑ CRP. Dx: temporal artery bx. Rx: high dose steroids ASAP (don't wait or refer if visual sx). Associated with polymyalgia rheumatica.
Dx and Tx of Bell's Palsy	CN VII Palsy. SSx: ipsilateral facial weakness (CANNOT wrinkle forehead → indicates peripheral lesion), lip droop/drooling, hyperacusis, retroauricular pain, ipsilateral tongue numbness, loss of taste sensation. Causes: idiopathic, infection (HSV = MCC), trauma, malignancy. Rx: prednisone, eye care. Complication: keratitis (cannot close eyelids). *Note: bilateral palsy: think Lyme, HIV, botulism, infectious mono.*
Recurrent episodes of severe unilateral headaches w/ ipsilateral lacrimation, rhinorrhea, nasal congestion, and conjunctival injection	Cluster headaches. Tx: oxygen via NRB (NC not effective). More common in men in their late 20s early 30s. V1 distribution. Attacks last 45-90 minutes. Happen 1-3x a day. Cluster period is 6-12 wks w/ remission for 12 months. Pt's may also have Homer's Syndrome.
Who needs a CT head prior to an LP?	1. Evidence of head trauma 2. AMS 3. Seizure 4. Focal neurological deficits 5. Papilledema and 6. Immunocompromised pts
5 yo boy w/ wide based gait, poor tone, horizontal nystagmus. Rapid onset. Happened 2 weeks after a URI.	Acute post infectious cerebellar ataxia. Must rule out other causes of ataxia.
Transverse myelitis	Young person with acute or subacute presentation of back pain and rapid progression to sensory loss and then motor loss at a certain transverse level. Typically happens after a viral infection but can be idiopathic. Get imaging and manage ABCs.

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Infectious Disease	
Bizz	Buzz
Dx and Tx: Fish tank granuloma	<i>Mycobacterium marinum</i> , Rx: Clarithromycin + Ethambutol OR Rifampin
Dx: Rose thorn injury and rash	<i>Sporothrix schenckii</i> . Rx: Itraconazole (amphotericin B if systemic)
Dx and Tx: Dog and cat bite with rapid infection	<i>Pasteurella multocida</i> . Rx: Amoxicillin-Clavulanate (augmentin → “dog-mentin”)
Dx: Reptile bites and infection	<i>Salmonella</i>
Dx: Sickle cell disease and joint pain	<i>Salmonella osteomyelitis</i>
Dx: Cat scratch fever	<i>Bartonella henselae</i> . Tx: doxy; azithromycin in pregnancy
Dx and Tx: Human bite and infection	<i>Eikenella corrodens</i> . Rx: amoxicillin-clavulanate
Dx: Gastroenteris on a cruise ship	Norwalk virus
Dx: Gastroenteritis at a daycare	Rotavirus, (remember, there is a vaccine now).
Compare general Dx and Tx of toxin-mediated vs. invasive bacterial diarrheal illness	<u>Toxin</u> : abrupt onset, watery, non-bloody; Rx IVF, ± loperamide, ± Cipro (prolonged/severe ssx). <u>Invasive</u> : gradual onset, bloody, systemic sx; Rx: IVF, ± Cipro UNLESS kids or elderly patients with possible <i>E. coli</i> O157:H7 (can increase risk of HUS)
Dx: Watery diarrhea + eggs/mayo	<i>Staph. aureus</i> (toxin)
Dx: Watery diarrhea + fried rice	<i>Bacillus cereus</i> (toxin)
Diarrhea + flatulence + recent hiking and drinkingsg from a fresh water stream	<i>Giardia lamblia</i> . Parasitic infection. Test for it w/ a stool antigen (not ova and parasite). Rx: Metronidazole / tinidazole. People who are risk: hikers, children at daycare, and oral-anal sexual conduct.
Dx: Watery diarrhea + travel	Enterotoxigenic <i>E. coli</i> (toxin); if no blood in diarrhea, give 1 dose of Cipro 750mg. If pt traveled to southeast asia this is likely campylobacter and give 1,000mg of azithromycin,
Dx: Watery diarrhea + meat/poultry	<i>Clostridium perfringens</i> (toxin)
Dx: Watery diarrhea + dark meat fish + rash/itching	Scombroid (histamine fish toxicity). Dark fleshed, peppery tasting fish: tuna, mahi-mahi, mackerel. Excess histidine on fish broken down by bacteria to histamine. SSx: Histamine ingestion leads to anxiety, flushing, headache, palpitations, vomiting. Rx: antihistamines
Dx: Watery diarrhea + carnivorous fish + neuro ssx	Ciguatera (toxin causes neuro ssx) - “hot cold reversal”
Dx: Bloody diarrhea + undercooked eggs/chicken + relative bradycardia	<i>Salmonella typhi</i> (invasive), cafeteria outbreaks, classically with high fever and relative bradycardia; can cause osteomyelitis in sickle cell patients.
Dx: Bloody diarrhea (severe) + high fever + institutionalized	<i>Shigella</i> (invasive); can cause seizures in kids
Dx and Tx: Bloody diarrhea + followed by weakness	<i>Campylobacter</i> (invasive). Associated: appendicitis mimic & Guillain-Barré; Rx: Azithro/Erythro (resistance to cipro)

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Dx: Bloody diarrhea + farm animals + appendicitis ssx	<i>Yersinia</i> (invasive); pseudoappendicitis (appy mimic) can cause terminal ileitis
Dx: Bloody diarrhea + undercooked seafood + alcoholic who gets very sick	<i>Vibrio parahaemolyticus</i> (invasive)
Dx: Bloody diarrhea + poorly cooked ground beef/raw milk	<i>E. coli</i> O157:H7, associated with TTP (adults) and HUS (kids); NO ANTIBIOTICS
Dx: Rice-water stools + contaminated water	<i>Vibrio cholerae</i> (toxin). Oral rehydration tablets.
Dx and Tx: Profuse diarrhea after recent antibiotics	<i>Clostridium difficile</i> (invasive). Rx: metronidazole or PO Vanco
Dx: Diarrhea + AKI ± low platelets	<i>E. coli</i> O157:H7 causing TTP/HUS
Dx: Food-borne illness associated with premature delivery in pregnant patients	<i>Listeria monocytogenes</i>
What is the most common cause of bacterial diarrhea in the US?	<i>Salmonella</i> ; 2nd <i>Campylobacter</i>
What factors on hx prompt you to get stools studies for patients w/ diarrhea?	1. Fever > 38.5C 2. Symptoms for greater than 1 week 3. Concerns for C.diff 4. Immunocompromised or advanced age 5. Underlying IBD.
Dx and Tx: Botulism	Paralytic illness. Cause: <i>Clostridium botulinum</i> neurotoxins → blocks ACh release at neuromuscular junction → flaccid paralysis. Forms: food borne (canned foods, honey), wound, infantile (most common). SSx: floppy baby, constipation, weak cry (infants); CN/bulbar sx , dilated pupils (differentiates from myasthenia), symmetric, descending flaccid paralysis / weakness (MC finding), parasympathetic blockade (dry mouth/sore throat, urinary retention). Tx: supportive care, resp. monitoring (resp. failure = MCC death), antitoxin (Rx infants: BabyBIG, Rx age>1: antoxin, abx (for wounds)
Leading cause of infertility?	<i>Chlamydia trachomatis</i>
Dx and Tx: Painless vesicular lesions or ulcers to groin + buboes (huge LNs)	Lymphogranuloma venereum (LGV). <i>Chlamydia trachomatis</i> . Rx: Doxy or Azithro (and treat partners), drain abscesses
Dx and Tx: Painful ulcer with irregular borders to groin + buboes (huge LNs)	Chancroid. <i>Haemophilus ducreyi</i> . SSx: looks like syphilis but the lesion is painful. Rx: CTX, Azithro or cipro (ANY one), drain abscesses
Dx: Neonate with copious purulent discharge from eyes	<i>Neisseria gonorrhoeae</i> conjunctivitis. Tx: systemic cefotaxime. Topical tx not enough.
Review the timing of the various causes for neonatal conjunctivitis	<u>Chemical</u> : first 24 hours. <u>Gonococcal</u> : first 2-5 days. <u>Chlamydial</u> : 5 days to 2 weeks.
Tx for neonatal chlamydial conjunctivas?	Systemic tx with azithromycin or erythromycin. Pt's must be admitted to look for pneumonia as well.

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Dx: "Gunmetal grey" pustules to hands/skin, septic arthritis ± tenosynovitis	Disseminated gonococcus. Arthritis-dermatitis syndrome. Gram-negative intracellular diplococci. Rx: IV ceftriaxone
Dx: Contact with armadillos	Leprosy. <i>Mycobacterium leprae</i> . Rx: dapsone + rifampin (+clofazimine for lepromatous disease)
Dx and Tx: Contact with prairie dogs	Bubonic plague. <i>Yersinia pestis</i> . Rx: streptomycin, tetracycline, doxycycline (alt. fluoroquinolones)
Most common cause of viral pneumonia in adults?	Influenza
Dx and Tx: HIV + Lung Disease + Pancytopenia	<i>Mycobacterium avium intracellulare</i> (MAI). CD4 < 50. Rx: Rifampin + Ethambutol + Azithro/Erythro
Dx and Tx: Primary tuberculosis	<i>Mycobacterium tuberculosis</i> . Transmission via inhalation of droplets. SSx: often asymptomatic. Can progress to latent or reactivation TB. Rx: Isoniazid (INH) + Pridoxine (Vit B6) x9mo.
Most common CXR finding in primary TB?	<u>Most common overall</u> : a single lobar infiltrate associated w/ hilar adenopathy / <u>children</u> : pronounced hilar adenopathy / <u>elderly</u> : isolated pleural effusion. <u>Ghon complex</u> : calcified lung lesion, ± calcified LNs [a.k.a. Ranke complex] representing HEALED infection. Immunocompromised pts often cannot form a Ghon complex.
Dx and Tx: Reactivation TB	SSx: often occurs if immunocompromised, other stressor; cough, fever, night sweats, weight loss, hemoptysis. Dx: XR with <u>upper lobe</u> apical lesions ± cavitation. Dx: MTB culture/PCR of sputum (takes weeks), AFB smear (suggestive but not diagnostic, need culture to confirm). Tx: RIPE (Rifampin, Isoniazid, Pyrazinamide, Ethambutol), **respiratory isolation** (airborne precautions), test/treat contacts
Immunocompetent person undergoes a high risk exposure to TB, what should you do?	1. Test them now w/ PPD or interferon gamma and test them again in 3 months for conversion. 2) if either of these tests are positive obtain a CXR and call an ID specialist.
What are the common side effects of TB treatment with RIPE?	<u>Rifampin</u> : orange body fluids, hepatotoxicity. <u>Isoniazid</u> : neuropathy, hepatotoxicity, seizures (in overdose, Rx: Vit B6). <u>Pyrazinamide</u> : hepatotoxicity, teratogenic (pregnancy). <u>Ethambutol</u> : optic neuritis (red-green color blindness)
Dx and Tx: Rapidly progressive skin infection, unusually high HR, and indifferent patient	Gas gangrene/myonecrosis. Clostridial myonecrosis (usually <i>C. perfringens</i>). SSx: similar presentation to necrotizing fasciitis, tachy out of proportion to fever, la belle indifference. Dx: subQ/intramuscular gas, incision with foul-smelling "dishwater" fluid and dead muscle. Rx: abx (amp + gent + clinda) + wide surgical debridement (dont delay abx)
Review the definitions of SIRS, sepsis, severe sepsis, septic shock	<u>SIRS</u> : Temp <36 (96.8) or >38 (100.4), Tachy >90, RR>20, WBC <4k or >12k or >10% bands. <u>Sepsis</u> : SIRS + infection. <u>Severe Sepsis</u> : sepsis + end organ damage. <u>Septic Shock</u> : sepsis + <u>refractory hypotension</u> . Note: ITE/boards still test these concepts.

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Review the key components of Early Goal-Directed Therapy for sepsis	Early IVF (30 cc/kg), early empiric antibiotics, MAP >65 (IVF or pressors), SvO ₂ >70%, CVP 8-12, transfuse pRBCs if Hct <30%. Note: strict adherence to this regimen has been debunked by several recent trials, but the necessity of adequate fluid resuscitation (30cc/kg) and early antibiotics remains well-supported.
Empiric abx for sepsis	<u>CAP</u> : CTX + Azithro. <u>HAP</u> : Vanc+Zosyn. <u>Urinary</u> : Amp+Gent. <u>Intra-abdominal</u> : Amp+Gent+Metronidazole. <u>Biliary</u> : pip+tazo. <u>Device related</u> : Vanc+Gent. <u>Skin/Soft tissue</u> : Vanco
Dx and Tx: Young woman with high fever + rash + shock and organ failure	Toxic Shock Syndrome. Cause: tampon, surgical or nasal packing or other foreign body; bacterial superantigen. <u>Staph</u> (TSS): more common; erythematous rash w/ desquamation, hypotension, fever, assoc. w/ fb; <u>Strep</u> (STSS): fever, but less rash often with existing wound. Rx: remove foreign bodies FIRST, supportive care, and antibiotics (clinda first to reduce protein production, then empiric broad-spectrum for sepsis coverage), IVIG for refractory cases
Dx and Tx: Primary syphilis	<i>Treponema pallidum</i> (spirochete). SSx: painless genital ulcer (<u>chancre</u>), regional LAD. Dx: VDRL/RPR are nonspecific and often negative at this stage. Tx: PCN G benzathine 2.4 million U IM x1
Dx and Tx: Secondary syphilis	Onset 5-8wks after primary. SSx: rash (papulosquamous) trunk → palms/soles, condyloma lata. Dx: VDRL or RPR, confirm with FTA-ABS. Tx: PCN G benzathine 2.4 million U IM x1 (if late disease three weekly doses)
Dx and Tx: Tertiary syphilis	Onset: years after primary. SSx: gummatous lesions throughout body, neurosyphilis (meningitis, dementia, Argyll-Robertson pupils [accommodate but don't react to bright light], tabes dorsalis [dorsal column demyelination causing impaired proprioception and vibratory sense [ataxia]). Dx: CSF-VDRL, confirm with FTA-ABS. Tx: admit for IV PCN q4h x2wks
Dx: Worsened rash and toxicity shortly after treatment of syphilis	Jarisch-Herxheimer reaction (2/2 endotoxin release from dying spirochetes). Tx: Supportive.
Care plan if syphilis pt is allergic to penicillin?	Admit for desensitization, they need PCN
Pathophysiology, Dx, and Tx: Tetanus	<i>Clostridium tetani</i> spores which enter into wounds. Sources: dust, soil, feces. Tetanospasmin = neurotoxin (blocks inhibitory [GABA] firing, leads to unopposed excitatory firing). SSx: <u>muscle spasticity</u> (lockjaw, painful tonic convulsions), but NORMAL mental status. Tx: supportive (benzos, opioids, ± paralytics), wound care, abx (metronidazole > PCN), Tdap vaccine (prevention) + tetanus IG (unimmunized + high risk wounds)
Review indications for tetanus prophylaxis	<u>≥3 vaccine doses + low risk wound</u> : dT >10 yrs since last dose. <u>≥3 vaccine doses + high risk wound</u> : dT >5 yrs since last dose. <u>Uncertain or < 3 vaccine doses + low risk wound</u> : dT. <u>Uncertain or <3 vaccine doses + high risk wound</u> : dT & TIG . High risk wound : >6hrs old, contaminated (dirt, saliva, feces), puncture/crush/avulsion wounds, fb, frostbite

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Dx and Tx: Red rash to diaper area with satellite lesions	<i>Candida</i> ; Rx: topical antifungals (also occurs in moist areas/skin folds esp. on diabetics)
Dx and Tx: Immunocompromised + odynophasia/dysphasia	<i>Candida</i> esophagitis; Rx: oral fluconazole, low threshold for IV for those who can't tolerate PO.
Dx and Tx: Indwelling catheter + yeast on blood cultures	<i>Candida</i> fungemia; Rx: Amphotericin B
Dx and Tx: AIDS + Diarrhea	<i>Cryptosporidium</i> , <i>Isospora</i> , CMV, <i>M. avium</i> ; often unclear cause. Rx: symptomatic care
Dx and Tx: Immunocompromised + Painless brown/black skin lesions	Kaposi sarcoma (classically on face, chest, oral cavity), caused by HHV-8; Rx: cryo or radiation. AIDS defining illness.
Dx and Tx: AIDS + white plaque on oropharynx	<u>Candida/Thrush</u> : plaques scrape off (Rx: clotrimazole) vs. <u>Oral Hairy Leukoplakia</u> : lateral tongue, can't scrape off, caused by EBV, very specific for HIV, NOT precancerous (DON'T confuse with oral leukoplakia which is PRECANCEROUS, tobacco)
Dx and Tx: Meningitis and focal neuro findings in AIDS pt	<i>Cryptococcus neoformans</i> (encapsulated yeast in soil with pigeon poop), SSx: HA, neck pain, fever, AMS or CN abnormalities. Dx: CSF cryptococcal antigen, LP with high opening pressure, + india ink stain (send the CSF for cyptococcal antigen for confirmatory testing). Rx: amphotericin B + flucytosine. MCC meningitis in AIDS.
Dx and Tx: Histoplasmosis	Dimorphic fungus. Found in spelunkers, caves, bird/bat droppings; can cause epidemics if soil upturned. Endemic to Ohio and Mississippi River valleys. SSx: flu-like sx, disseminated disease or chronic progressive pulmonary disease (diffuse infiltrates and calcified nodes). Rx: itraconazole, amphotericin B.
Dx and Tx: Immunocompromised + encephalitis + ring-enhancing lesions on CT	<i>Toxoplasmosis gondii</i> (protozoan). Associated with cat poop, bad for fetus if infection occurs during pregnancy (TORCH). Tx: Pyrimethamine, sulfadiazine, folinic acid.
Dx: Travel + cyclical fever	Malaria. <i>Plasmodium</i> protozoan transmitted by female <i>Anopheles</i> mosquito. Infects RBCs & hepatocytes. SSx: cyclical fevers (febrile during periods of RBC rupture and merozoite spread), splenomegaly, thrombocytopenia. Complicated dz: profound hypoglycemia, hemolytic anemia, sz, coma. Dx: thick+thin blood smears (ring forms) ± Giemsa or Wright stain.
Malaria Tx	<u>Uncomplicated+chloroquine-sensitive</u> (central america, Caribbean): chloroquine. <u>Uncomplicated+chloroquine resistant</u> (South America, South Asia, Africa): Quinine (hypoglycemia) + Doxy. <u>Complicated or P. Falciparum</u> : Quinidine IV (QT prolongation)
What is the most dangerous/severe strain that causes malaria?	<i>P. falciparum</i> : cerebral malaria, "Blackwater" fever, death; Rx: IV quinidine

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Dx and Tx: traveler + myalgias and fever	Dengue fever. Dengue virus + transmitted <i>Aedes aegypti</i> mosquito. Common in Caribbean (Puerto Rico). SSx: high fever, dramatic myalgias ("break-bone fever"), morbilliform rash. Dx: serology or ELISA, leukopenia + thrombocytopenia. Rx: supportive
What is the cause, vector, and treatment of Lyme disease?	<i>Ixodes</i> tick (deer tick) carrying <i>Borrelia burgdorferi</i> , primarily in northeast US & Wisconsin; tick bite history is often absent. Rx: doxycycline, CNS/cardiac involvement: IV CTX. Pregnant, children <8 Rx: Amoxicillin
What are the typical stages of Lyme disease?	<u>Stage 1</u> : erythema migrans ("bull's eye") rash (1 wk). <u>Stage 2</u> : neuro changes (meningoencephalitis = MC neuro finding with CN palsies - bilateral Bell's palsy), cardiac changes (variable AV block = MC cards finding); (days to weeks). <u>Stage 3</u> : arthritis, neuro sx (months to years). Tx: Doxy for everything other than severe carditis or neurological manifestations, use IV ceftriaxone
Describe prophylactic tx of Lyme disease	Criteria: tick attached for ≥ 36hrs, ppx started within 72 hours of tick removal. Other factors: tx if tick engorged. Rx: single dose Doxy
Dx, vector, and Tx: Recent hiking or camping, rash spreading inwards ("centripetal", palms/soles)	Rocky Mountain Spotted Fever. <i>Rickettsia rickettsii</i> . Transmission: wood tick (<i>Dermacentor andersoni</i>), eastern US (Carolinas, Oklahoma). SSx: fever (MC sx), centripetal (towards trunk) rash (palms+soles), calf tenderness. Labs: low platelets, hypoNa. Rx: ALWAYS doxycycline (even children)
Dx and Tx of Ehrlichiosis	<i>Ehrlichia</i> spp. with tick vector. South central & south Atlantic US. SSx: abrupt onset fevers/chills/myalgias/rigors (differentiates from other tick borne illnesses) ± rash. Labs: leukopenia, thrombocytopenia, and elevated LFTs. Rx: Doxy, alt. Rifampin
Dx and Tx: Fever, exudative pharyngitis, posterior lymphadenopathy	Infectious mononucleosis. Epstein-Barr virus (EBV); Exam: ± splenomegaly (risk rupture, limit contact/activity). Rx: supportive.
Dx: Mono + amoxicillin for presumed strep	90% develop maculopapular rash (NOT an allergy)
What are associated lab abnormalities found in patients with mono?	Atypical lymphocytes, +heterophile antibodies (monospot test), hemolytic anemia, thrombocytopenia, elevated LFTs, false positive RPR or VDRL
What is the difference between genetic drift and genetic shift?	<u>Antigenic drift</u> : minor mutation. <u>Antigenic shift</u> : major mutation; often used in context of influenza (orthomyxovirus) and HA/NA surface antigens
What defines a pandemic flu?	When antigenic shift takes place and a new strain of the influenza virus emerges.
Who is at high risk for death with influenza and what is the usual cause of death?	Extremes of age and pregnant women are highest risk; most common cause of death is secondary pneumonia.
What is the appropriate tx regimen and complications of tx for influenza?	Oseltamivir/Tamiflu (GI sx) or Zanamivir/Relenta (bronchospasm, wheezing) = Neuraminidase inhibitors if within 48hr of sx onset or hospitalized; No Amantadine/Rimantadine (both CNS sx) 2/2 resistance.

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Dx and Tx: Rat poop and ARDS	Hantavirus. Transmitted via aerosolized rodent excretions. SSx: Hantavirus pulmonary syndrome - ARDS, thrombocytopenia. Starts with a flulike syndrome and is then rapidly progressive (and therefore many patients are discharged and return critically ill). Rx: supportive
What is the location of dormant herpes simplex?	Dorsal root ganglion, reactivated with stress/immunocompromise
What are the classic locations/presentations for HSV 1 and 2 and how are they diagnosed?	<u>HSV-1</u> : mouth, stomatitis, keratitis (possible corneal ulcer), vesicles on digits (Whitlow); <u>HSV-2</u> : anus, genital, & neonatal (C-section if pregnant and in labor). Dx: Tzanck smear (multi-nucleated giant cells), viral culture. Rx: acyclovir
Compare the presentation and treatment of chickenpox vs. shingles (Varicella Zoster Virus)	<u>Primary varicella</u> (chickenpox): highly contagious, incubation ~2wks. SSx: crops of vesicles in various stages of healing. Rash: "dew drop rash on a rose petal", starts on hairline → chest, palms/soles (+mucous membranes). Rx: <u>healthy/age <12</u> : supportive, monitor for bacterial superinfection; <u>immunocompromised, age >12</u> = acyclovir. <u>Shingles</u> : reactivation of dormant VZV. SSx: prodrome (itching, burning), rash-painful vesicular eruption (usually unilaterally in a single dermatome), Rx: acyclovir, steroids (controversial), pain control. Complication: post-herpetic neuralgia
Dx: Bell's palsy and vesicle on ear	Ramsay-Hunt syndrome/Zoster Oticus (VZV of CN VIII)
Dx: Vesicle on tip of nose	Hutchinson's sign, V1 zoster, predicts corneal involvement/ulceration (Zoster Ophthalmicus)
Treatment of herpes zoster ophthalmicus?	Systemic oral anti-virals and topical steroids. This happens when the nasociliary branch of CN V is infected with VZV. Emergent ophthalmology follow up is needed.
Tx of pregnant or immunocompromised pt after exposure to VZV?	Send titers to check for immunity, and if negative give varicella zoster IG.
Pregnant woman presents with SOB and wide spread itchy vesicular rash in various stages of development.	Varicella pneumonia. Tx: Admit and tx with IV acyclovir. VZIG IS NOT ENOUGH treatment.
what is the definition of disseminated zoster?	Involvement of 3 or more dermatomes.
What animals are high risk for rabies transmission?	<u>Domestic animals</u> : cats > dogs (dogs in developing countries are HIGH risk). <u>Wild animals</u> (account for ~90% cases in US): bats (MCC in US) > raccoons > skunks > foxes > coyotes, mongooses. NOT rabbits or rodents.
What are symptoms of rabies infection?	Incubation 3-7wks, pain/paresthesia at bite site, hydrophobia (drinking water causes painful spasm), seizure, encephalitis, death
What is the treatment for rabies?	There is no treatment for active disease. PEP: HRIG (at wound site), HDCV (rabies vaccine): 5 injections over a month. PEP (vaccine + RIG) for close proximity with a bat and exposure cannot be ruled out (e.g. awakening to find bat in room, unattended children)

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What defines AIDS?	HIV with CD4 < 200 OR AIDS defining illness (esophageal candidiasis, cryptococcus, CMV, Kaposi, PCP, toxoplasmosis, TB (in an non-endemic area))
What is the usual presentation of acute HIV infection?	Acute HIV (Seroconversion) Syndrome. Often missed, 2-4wk post-exposure. SSx: non-specific viral syndrome (fever, rash, LAD, myalgias). Most infectious stage of HIV (high viral load + shedding), but antibody testing will be negative as seroconversion takes 3-12 wk post-exposure.
How is HIV diagnosed	<u>ELISA</u> : to screen (sensitive), oral swab, delayed (+) weeks to months. <u>HIV-1/2 immunoassay & Western blot</u> : to confirm; (sensitive + specific), blood test.
What opportunistic infections are more likely below the following CD4 counts: <500, <200, <100, <50	<500: TB, HSV, VZV, Kaposi's sarcoma. <200: PCP, HIV encephalopathy, candidiasis, PML. <100: toxoplasmosis, histoplasmosis, cryptococcus. <50: CMV (GI, pulm, retina), MAC avium, CNS lymphoma. **NOTE: HIV pts get all usual infections as well, but have increased risk of opportunistic as CD4 drops.**
What common lab test can be used as a surrogate to determine CD4 count?	Absolute lymphocyte count (ALC). ALC < 1000 → suggests CD4 < 200
What is the time range for starting post-exposure prophylaxis after HIV exposure?	Should start within 72hr
Dx and Tx: Immunocompromised + pneumonia with severe dyspnea/hypoxia + high LDH	PJP pneumonia. <i>Pneumocystis jirovecii</i> (formerly PCP). Most common opportunistic infection in AIDS. SSx: fever, cough, desat on exertion. Dx: CXR shows interstitial perihilar infiltrates ("bat wing" pattern). Rx: TMP-SMX, steroids (indications: children, PO2 < 70 mm Hg [~SaO2 ~93%], A-a gradient > 35)
What are possible side effects with pentamidine (Tx for PCP pna)	Hypoglycemia, hypotension, pneumothorax
what are the appropriate outpatient tx for PCP pneumonia?	TMP-SMX; For sulfa allergic people: Primaquine + clindamycin, or TIM-dapsone. Pentamidine can be given but it's only available in IV and inhalation forms making outpatient therapy complicated. It also has many side effects. v
What are typical CT (non-contrast and contrast) findings with <i>Toxoplasmosma</i> encephalitis?	<u>Non-contrast CT</u> : multiple subcortical lesions in basal ganglia. <u>Contrast CT</u> : ring-enhancing lesions with surrounding edema. Rx: Pyrimethamine, sulfadiazine and leucovorin
Dx: Ring-enhancing intracranial lesions with 1) focal neuro deficit or 2) generalized AMS	<u>Focal deficits</u> : <i>Toxoplasma</i> . <u>Generalized AMS</u> : CNS lymphoma (hyperdense focal lesions, CD4 < 50)
Dx: HIV + CD4 <200, focal neurologic deficits with nonenhancing white matter lesions	PML (JC virus)

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Dx and Tx: Progressive blindness in AIDS patient	CMV retinitis. Exam shows "fluffy white perivascular lesions (cotton wool spots) with areas of hemorrhage." Rx: IV Gancyclovir
What factors increase the risk of transmission after occupational exposure to HIV?	Deep injury, visible blood, hollow bore needle from vein or artery, late stage HIV/AIDS or high viral load; transmission risk is 0.3% with needlestick, 0.1% with mucous membrane exposure
What are the guidelines for post-exposure prophylaxis for HIV?	HIV+ and <72hr: HAART for 28d; if low risk and >72hr no treatment is necessary. All others per clinical judgement.
What are potential oral antibiotic options for community acquired MRSA?	Clindamycin, TMP-SMX, doxycycline (requires IV vanco if hospital-acquired)
Dx: Skin lesion, Gram positive rod	Anthrax
Dx and Tx: Cutaneous vs. pulmonary anthrax	<i>B. anthraxis</i> (Gram positive rod). <u>Cutaneous</u> : pruritic, black eschar + painful LAD over 1-2wks. <u>Pulmonary</u> : due to inhaled spores (not contagious), flu-like sx. CXR shows wide mediastinum ; rapid progression to sepsis + death. Rx: cipro
Dx and Tx: Pneumonic and bubonic plague	<i>Yersinia pestis</i> . <u>Pulmonic</u> : inhaled aerosolized rat droppings, very contagious, severe pna, bio-terrorism agent. <u>Bubonic</u> : transmitted via flea bite, causes buboes + acral necrosis (black/dead distal extremities), may travel to lungs (contagious at this stage). Rx: streptomycin, gentamicin, doxycycline.
What risk factor is most strongly associated with cellulitis?	Lymphedema
What is the appropriate management of a patient with a tick bite, target rash, and Bell's palsy?	CT and LP followed by ceftriaxone with concern for disseminated Lyme/CNS Lyme
What is the most infectious blood-borne pathogen?	Hep B, followed by Hep C and HIV
Dx and Tx: military recruit or college student with fever, HA, petechial rash	Meningococcemia. <i>Neisseria meningitidis</i> (aerobic, gram-negative diplococcus). Rx: CTX + Vanco. Most common complication: Myocarditis with CHF or conduction abnormalities. Comments: <u>Waterhouse-Friderichsen syndrome</u> : bilateral adrenal hemorrhage + meningococcemia

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<p>Classic opportunistic infections in AIDS</p>	<p>Chronic watery diarrhea: <i>Cryptosporidium</i> White cottage cheese lesions: <i>Candida</i> Irremovable white lesions on lateral tongue: hairy leukoplakia (EBV) Pneumonia, CD4 <200: PCP TB: CD4 <200, may have negative CXR/PPD Ring-enhancing intracranial lesions + focal neuro deficits: <i>Toxoplasma gondii</i> Ring-enhancing intracranial lesions + AMS: primary CNS lymphoma Meningitis, CD4 <100: <i>Cryptococcus</i> Focal neurologic deficits, non-enhancing white matter lesions, CD4 <50: PML (JC virus) Retinitis, cotton-wool spots: CMV, CD4 <50 Dark purple skin/mouth nodules: Kaposi's sarcoma Cutaneous: HSV, zoster reactivation</p>
<p>HIV medication side effects</p>	<p>Didanosine: pancreatitis Efavirenz: vivid dreams, headache, severe rash, dizziness Indinavir: nephrolithiasis (radiolucent), hyperbilirubinemia, hepatitis Lopinavir: nausea, vomiting, diarrhea, hepatitis NRTIs: lactic acidosis Ritonavir: paresthesias Zidovudine: bone marrow suppression</p>
<p>Relative bradycardia in the setting for a fever after travel to SE Asia when pt presents w/ fever, malaise and relative anemia and elevated LFTs.</p>	<p>Typhoid fever. Caused by salmonella typhi. Oral ingestion of contaminated food or water. Sxs: Fever, malaise, abdominal pain, fatigue. Diarrhea in children, constipation in adults. Rx: fluoroquinolones in adults, third generation cephalosporin in children. Vaccines only gives 55% immunity.</p>
<p>Nail puncture wound to the plantar surface of the foot?</p>	<p>Barefoot: lower risk for infection, if infection occurs typically staph or strep. W/ shoes: pseudomonas is a risk. Obviously high risk pts are still high risk: DM, PVD, incredibly contaminated environment, etc ...</p>

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Immunology	
Bizz	Buzz
What is the difference between anaphylaxis and anaphylactoid reactions?	<u>Anaphylaxis</u> : IgE mediated; <u>Anaphylactoid</u> : histamine release independent of IgE. However they look the same and should be treated the same
What is the typical presentation and appropriate treatment of anaphylaxis?	Criteria: skin sx (e.g. urticaria, itching or flushing = MC) + resp. compromise OR 2+ organ system involvement (bronchospasm, hypotension, urticaria, GI sx). Usually occurs within 60 min of exposure. Rx: ABCs (intubate PRN), IV, supplemental O2, cardiac monitor, Epinephrine (1:1000) 0.3mg IM (not subQ), H1/H2 blockers, steroids. Refractory Rx: epi drip, glucagon, vasopressors
What medication should be given for a patient on beta blockers who develops anaphylaxis?	Glucagon (pts on BB may not respond to Epi)
Dx and Tx angioedema	Edema of cutaneous & subQ tissue 2/2 capillary dilation. SSx: painless, non-pruritic (NO urticaria), non-pitting edema of skin (NO rash). May affect abdominal organs & upper airway. <u>Hereditary</u> : deficiency or dysfxn of C1-esterase inhibitor; Rx: FFP (replace C1 esterase inhibitor). <u>Drug-induced</u> : ACE-I/ARB; ↑ bradykinin; supportive. General Rx: supportive, intubate pm, give standard anaphylaxis Rx (likely wont work), FFP (contains C1 esterase)
Review Type I-IV hypersensitivity reactions and give an example of each	<u>I</u> : IgE-mediated, 2 separate exposures; e.g. anaphylaxis/urticaria. <u>II</u> : cytotoxic antibody (IgG & IgM-mediated) reacton; e.g. hemolytic transfusion reactions. <u>III</u> : IgG-immune complex deposition, e.g. serum sickness & vasculitis. <u>IV</u> : T-cell mediated (no antibodies), e.g. Stevens-Johnson syndrome, TB skin test, contact dermatitis
Dx and Tx: Serum sickness	Type III rxn. Clasically occurs after meds (PCN, sulfas). SSx: fever, rash (fingers/toes → morbilliform), arthralgias. Labs: ↓ C3/C4. Rx: supportive.
Most likely infection after renal transplant w/in 1-6months?	CMV
Management: s/p transplant + sick	Assume infection AND rejection (they look the same); less likely to have fever with infection.
Dx and Tx: Graft versus host disease	Acute is <100 days since transplant; SSx: fever, rash (most common), hypoxemia, multi-organ failure. Tx: steroids!, empiric antibiotics; avoid ASA and NSAIDs
What is the best prognostic marker for graft function after renal transplant?	Creatinine - must calculate GFR

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What are the most likely sources of infection in transplant patients in the following periods post-transplant: <1mo, 1-6mos, 6+mos?	<1mo: infection related to procedure and hospitalization such as wound infections (Strep, Staph/MRSA, <i>Pseudomonas</i>). 1-6mos: Viruses (CMV, EBV). >6mos: chronic viral infections (CMV, EBV, HSV, VZV, Hep B and C) and community acquired infections.
What is the timeline for hyperacute vs. acute vs. chronic rejection after transplant?	<u>Hyperacute</u> : minutes to hours after transplant, 2/2 preformed antibodies causing irreversible graft destruction (esp. ABO mismatch). <u>Acute</u> : 1-2wks, humoral/T-cell mediated. <u>Chronic</u> : months-years.
Dx and Tx: pruritis and erythematous rash while receiving Vanco	Red man syndrome. AnaphylactOID reaction. Rx: stop infusion, Rx diphenhydramine; may restart if sx resolve (to r/o anaphylaxis) at a slower rate
Dx and Tx of Systemic Lupus Erythematosus (SLE)	Pt will be an African-American or a woman. SSx: fever, LAD, weight loss, general malaise, or arthritis, malar rash ("butterfly rash"). Labs: +ANA, +anti-dsDNA Ab, anti-smith Ab, anti-histone Ab. Rx: NSAIDs, steroids, immunosuppressants, hydroxychloroquine. <u>Drug-induced</u> : Hydralazine, INH, Procainamide, Phenytoin, Sulfonamides (HIPPS). False-positive test for syphilis
Dx and Tx: elderly woman with monocular vision loss, unilateral HA, jaw claudication, tender temple	Temporal Arteritis (Giant Cell Arteritis). Dx: Labs show ESR > 50, temporal artery biopsy. Rx: high dose steroids ASAP (don't wait for biopsy results. Associated with polymyalgia rheumatica (PMR)
Classic organ transplant presentations	<u>Renal transplant rejection</u> : ↑ Cr, tenderness, ↓ urine output <u>Lung transplant rejection</u> : cough, chest tightness <u>Heart transplant rejection</u> : fatigue, HF, no angina/CP <u>Liver transplant rejection</u> : fever, abnormal LFTs, RUQ pain <u>Transplant rejection Rx</u> : steroids
Common lab findings of sarcoidosis	↑↑ ACE levels, ↑ Calcitriol production (1, 25-dihydroxyvitamin D, active metabolite of Vitamin D), Hypercalcemia (MCC of ARF in sarcoidosis), Hypercalciuria, ↓ PTH levels
Classic vasculitides	<u>Temporal arteritis</u> : PMR, carotid artery branches affected, vision loss, Rx: immediate steroids <u>Takayasu's arteritis</u> : Asian, decreased pulses <u>PAN</u> : generalized without lung involvement, HBV <u>Buerger's disease</u> : smokers, claudication of hands/feet <u>Granulomatosis with polyangiitis (GPA)</u> : Upper & lower respiratory sx + renal sx, c-anca <u>Microscopic polyangiitis</u> : similar to GPA but without nasopharyngeal involvement, p-ANCA <u>Churg-Strauss syndrome</u> : vasculitis + eosinophilia + asthma <u>Cryoglobulinemia</u> : HCV, malaise, skin lesions, joint pain <u>Behçet's disease</u> : oral and genital ulcers, hyperreactivity to needle sticks

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Toxicology	
Bizz	Buzz
How does activated charcoal work for decontamination and how should it be administered?	High surface area binding to toxin and preventing systemic absorption. Dose: 10:1 (10g AC per 1g drug)
When is activated charcoal contraindicated or ineffective?	Contraindicated in AMS/obtunded patient, risk of seizure or aspiration, ileus. Ineffective for pesticides, lithium, hydrocarbons, heavy metals, alcohols, caustics.
How does whole bowel irrigation work for decontamination and how should it be administered?	Iso-osmotic agent (e.g. Go-Lytely) taken in large volume will hasten progress through intestines and prevent absorption; Dose: 1-2L/hr (adults) or 500mL/hr (kids), consider giving by NGT, and continue until clear rectal effluent is produced. Think about using this for “drug packers” and also for ER beta-blockers.
For what types of ingestion is whole bowel irrigation most effective?	Iron, lithium, sustained-release formulations, enteric-coated meds, body packers
What toxins are cleared by HD?	"I STUMBLE": <u>I</u> sopropyl alcohol, <u>S</u> alicylate (aspirin), <u>T</u> heophylline, <u>U</u> ric acid, <u>M</u> ethanol, <u>B</u> arbiturates/ <u>B</u> eta-blockers, <u>L</u> ithium, <u>E</u> thylene glycol
Review the general pathophysiology, Dx, and Tx of Acetaminophen (APAP) overdose	APAP normally broken down via sulfation & glucuronide conjugation. Small amount is broken down by CYP450 enzymes → toxic NAPQI metabolite. <u>APAP OD</u> : overloads glutathione inactivation/metabolism → excess NAPQI accumulates → liver toxicity. Dx: can use acetaminophen nomogram for SINGLE, ACUTE ingestions; get APAP level at ≥ 4 hours (< 4 hours NOT useful, unless ZERO). High risk of toxicity: >150mg/kg (acute) or >4g/day (chronic). Rx: N-acetylcysteine (NAC) → restores glutathione, acts as an antioxidant; best if given w/in 8 hours of ingestion; Dose PO (140mg/kg load, 70mg/kg q4hr) or IV (150mg/kg load, 50mg/kg over 4hr, 100mg/kg over 16hr). Safe for pregnant woman and children. Side effect: anaphylactoid reaction.
When can you use the Rumack-Matthew nomogram for acetaminophen toxicity?	For single acute ingestions , not for chronic ingestions. For chronic you treat based on acetaminophen level or signs of acute toxicity
Review the general pathophysiology, Dx, and Tx of NSAID overdose	COX inhibitor decreases prostaglandin production. SSx: minimal toxicity but can cause GI upset (rarely GI Bleed); large doses: AMS/ataxia, coma, metabolic acidosis, seizure. Rx: supportive.
Review the general pathophysiology, Dx, and Tx of Aspirin (ASA) overdose	Causes primary respiratory alkalosis (stimulates resp. center, EARLY, uncouples oxidative phosphorylation (AG metabolic acidosis, hyperthermia). SSx: ↑ RR, ↑ temp, ↑ HR (sinus tach = MC sign), tinnitus, vertigo, AMS, seizure. Rx: GI decon, urine alkalization with bicarb (+K, +Mg) infusion (enhances urinary excretion of salicylate, also prevents CNS distribution), HD (acute level >100, chronic level >60, OR there is a presence of renal failure, severe acidemia, or pulmonary/cerebral edema. **If you intubate, you must set high RR or the acidemia will worsen and the pt will arrest.**

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Review the general pathophysiology, Dx, and Tx of opioid overdose	Analgesic causing respiratory depression and impaired consciousness. SSx (TRIAD): CNS depression + respiratory depression + miosis (pinpoint pupils). Rx: naloxone (start low dose to avoid withdrawal & vomiting, uncomfortable but not life-threatening except in neonates)
Review unique clinical complications for meperidine, tramadol and methadone	<u>Meperidine</u> : seizures, serotonin syndrome, often dilated pupils . <u>Tramadol</u> : seizures, serotonin syndrome, anticholinergic effects (mydriasis). <u>Methadone</u> : QT prolongation (& TdP), hypoglycemia.
What types of opioids are NOT seen on urine tox screen?	Synthetics. Includes fentanyl, hydromorphone, buprenorphine, methadone, meperidine. Natural derivatives will show up (heroin, morphine, codeine, hydrocodone, oxycodone).
What is the potential risk of using meperidine, tramadol, or dextromethorphan in the setting of antidepressant use?	Serotonin syndrome
Review the general pathophysiology, Dx, and Tx of clonidine intoxication	Alpha-2 agonist. In OD appears similar to opioid toxidrome but causes bradycardia. SSx: AMS + miosis + respiratory depression. Others: bradycardia, hypotension . Rx: supportive, atropine, pressors, naloxone.
What common substances are associated with methanol, ethylene glycol, and isopropyl alcohol ingestions?	<u>Methanol</u> : wood alcohol, moonshine, windshield washer fluid, paint solvent, perfume, antifreeze. <u>Ethylene glycol</u> : antifreeze & other automotive fluids, radiator coolant, aircraft de-icing. <u>Isopropyl alcohol</u> : rubbing alcohol, hand sanitizer.
Clinical manifestations of an ethylene glycol and methanol intoxication?	Comatose pt with slight hypothermia, tachypnea and a blood gas that shows metabolic acidosis with normal respiratory compensation. **remember metabolic acidosis w/ respiratory compensation = last two numbers of pH roughly equal the PCO2 (pH 7.23 → PCO ~ 23)
What are the potential clinical consequences and clues to diagnosis of methanol, ethylene glycol, and isopropyl alcohol ingestions?	<u>Methanol</u> : metabolized to formic acid → optic neuropathy ("snowy" field of vision, blindness) and basal ganglia injury; Labs: + AG metabolic acidosis, ↑ osmolar gap. <u>Ethylene glycol</u> : metabolized to glycolic acid & oxalic acid (binds Ca → low Ca + renal failure); Labs: hypoCa, calcium oxalate crystals in urine, +AG metabolic acidosis, ↑ osmolar gap. <u>Isopropyl alcohol</u> : metabolized to acetone (uncharged ketone), no significant pathology other than CNS depression; similar to ethanol overdose, ketosis WITHOUT acidosis, NO anion gap.
Biggest lab difference btw ethylene glycol and methanol ingestions when compared to isopropanol (isopropyl alcohol) ingestion?	Ethylene glycol and methanol will lead to an AG metabolic acidosis with ↑ osmolar gap. Isopropanol will lead to ketosis, no acidosis
How do you calculate osmolar gap?	Serum Osmolality: $2 \times \text{Na} + \text{Glucose}/18 + \text{BUN}/2.8 + \text{EtOH}/4.6$; Gap = Calculated - Measured; normal is less than 10

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What is the treatment for methanol, ethylene glycol and isopropyl alcohol ingestions?	Fomepizole (& ethanol) inhibits alcohol dehydrogenase + prevents formation of toxic metabolites for methanol/ethanol/ethylene glycol. <u>Methanol</u> : fomepizole, ethanol, folinic acid, HD. <u>Ethylene glycol</u> : fomepizole, ethanol, thiamine, HD is ultimate treatment. <u>Isopropyl alcohol</u> : supportive care.
What cofactors are required with treatment of ethylene glycol and methanol ingestions?	Ethylene glycol: thiamine & pyridoxine. <u>Methanol</u> : folinic acid.
What are potential adverse effects of ethanol intoxication?	"4 Hs": <u>Hypotension</u> , <u>Hypoventilation</u> , <u>Hypothermia</u> , <u>Hypoglycemia</u> ; atrial tachycardias ("holiday heart")
What is the time course of symptoms in alcohol withdrawal?	Alcohol withdrawal syndrome: cessation/reduction of etoh + etoh w/d sx. SSx: begin within 6-24 hours after the last drink; Tremulousness (6-12hrs) → hallucinations (12-48hrs) → seizures (12-48hrs) → delirium tremens (>48hrs).
Review the general pathophysiology, Dx, and Tx of lidocaine toxicity	Blocks Na channels → blocks/slows nerve conduction. Toxic dose: >4mg/kg plain or >7mg/kg with epi. SSx: CNS toxicity (perioral numbness, slurred speech, seizure), CV toxicity (VT/VF, AV block, AVNRT), methemoglobinemia, allergic rxn. Rx: benzo (seizure), bicarb (arrhythmia), methylene blue (MetHgb), epi (allergic rxn), consider intralipid for cardiovascular collapse.
What is the difference between ester and amide anesthetics?	<u>Esthers</u> (1 I): cocaine, procaine, benzocaine; shorter acting, higher risk of allergic reaction 2/2 preservative or PABA. <u>Amides</u> (2 I's): lidocaine, mepivacaine, bupivacaine; longer acting, OK to use if allergy to ester. If pt has allergy to lido can use crash cart lido (preservative free) or IV diphenhydramine
Review the general pathophysiology, Dx, and Tx of anticholinergic toxidrome	Causes: tricyclic antidepressants, atropine, antihistamines (MCC), belladonna (nightshade), jimsonweed, phenothiazines. Clinically DRY : "Blind as a bat (mydriasis), mad as a hatter (agitation/AMS), red as a beet (flushing), hot as a hare (hyperthermia), dry as a bone (hot/dry skin), bloated as a toad (urinary and stool retention), and the heart runs alone (tachycardia)." Rx: supportive, benzos, sodium bicarb (wide complex dysrhythmias) ± physostigmine (cholinesterase inhibitor; avoid if known TCA OD, wide QRS, seizure)
How might you identify TCA overdose in a patient with anticholinergic toxidrome?	Get an EKG! TCA overdose is suggested by wide QRS or terminal R wave in aVR.
How can you distinguish an anticholinergic toxidrome from a sympathomimetic toxidrome?	The skin (not the pupils)! Anticholinergic will have dry skin. Sympathomimetic toxidrome will have diaphoresis . Just think about sweaty people after taking cocaine (sympathomimetic) and partying so they are sweaty.
Review the general pathophysiology, Dx, and Tx of cholinergic toxidrome	Causes: insecticides, organophosphates, chemical warfare. Clinically WET : "DUMBBBELLS" (Diarrhea, Urination, Miosis, Bradycardia, Bronchorrhea, Bronchospasm, Emesis, Lacrimation, Lethargy, Salivation). Rx: atropine (often high doses, titrate to dry secretions), 2PAM (pralidoxime). Do not intubate w/ succinylcholine as it will cause prolonged paralysis.

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What is the mechanism of and approach to reversal of warfarin?	Inhibits vitamin K activation, blocking VitK-dependent synthesis of clotting factors (II, VII, IX, X), monitored by INR. Rx: asymptomatic → hold doses & follow INR; active bleeding → FFP (or prothrombin complex concentrates) + vitamin K
What is the mechanism of and approach to reversal of heparin and LMWH?	Potentiates antithrombin III (ATIII) enzyme → inactivates thrombin (monitored by PTT) and activated factor Xa (LMWH). Reversal agent: protamine sulfate (only partially effective for LMWH); may cause allergic reaction.
Dx and Tx: Heparin-induced thrombocytopenia (HIT)	Antibodies that inactivate platelets usually at 5d if naive and only min/hours if prior exposure. Dx (4 T's): <u>t</u> hrombocytopenia, <u>t</u> ime of onset (5-10d), <u>t</u> hrombosis, no <u>o</u> ther cause. Labs: +HIT antibody. Rx: STOP heparin or LMWH, can change to DTI (argatroban).
What is the mechanism of and approach to reversal of clopidogrel?	Anti-platelet agent monitored with P2Y12 level. SSx: hemorrhage, TTP, neutropenia. Rx: platelet transfusion.
What is the mechanism of and approach to reversal of dabigatran (Pradaxa)?	Direct thrombin inhibitor. Reversal: HD, albumin, prothrombin complex concentrates (NB: makes pt transiently hypercoagulable), idarucizumab (Praxibind).
What is the mechanism of and approach to reversal of rivaroxaban (Xarelto) and apixaban (Eliquis)?	Direct factor Xa inhibitors. Rx: supportive, no known reversal agent, consider PCC or TXA if life-threatening bleeding is present.
Review the general pathophysiology, Dx, and Tx of phenothiazines (e.g Compazine, Phergan, Thorazine) toxicity.	Block dopamine receptors (also ACh receptors, ion channels). SSx: CNS effects: sedation, seizures; EPS effects: dystonia, parkinsonism, TCA-like ion channel effects, CV: long QT/TdP, hypotension; others: miosis NMS. Rx: supportive (IVF, benzos, Mg)
Review the general pathophysiology, Dx, and Tx of 5HT3 (serotonin) antagonists.	Ondansetron (Zofran). Prolonged QT & TdP. Rx: Mg 2g IVP.
What drugs are at risk for causing prolonged QT/torsades and what is the treatment?	Class Ia&Ic antidysrhythmics, TCAs, antipsychotics, abx (macrolides, fluoroquinolones), antiemetics. Rx: Mg IVP, (alt. overdrive pacing with isoproterenol), cardioversion (unstable).
Review the general pathophysiology, Dx, and Tx of cocaine intoxication	Inhibits neuronal uptake of catecholamines (e.g. norepinephrine), Na ⁺ channel blockade. ↑ catecholamines → HTN, hyperthermia, rhabdo, MI 2/2 coronary vasospasm, seizure, VT. Rx: benzos, cooling, nitrates, nicardipine, phentolamine for tachycardia (on exam). AVOID β-blockers as these might cause unopposed α.
Review the general pathophysiology, Dx, and Tx of amphetamine intoxication	↑ catecholamine release → HTN, tachycardia, hyperthermia, rhabdo, hypertensive intracranial hemorrhage. Rx: benzos, cooling, nitrates, nicardipine (avoid beta blockers).
Review the general pathophysiology, Dx, and Tx of synthetic cannabinoid ("K2," "Spice," "Herbal Marijuana") intoxication	Causes a variety of effects on neuromodulation. SSx: anxiety, paranoia, tachycardia, diaphoresis, psychosis, seizures. Labs: ± marked metabolic derangements. Rx: supportive (IVF, benzos for agitation & seizures)

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Review the general pathophysiology, Dx, and Tx of hallucinogenic amphetamines (e.g. MDMA/ecstasy).	↑ catecholamine, serotonin, and dopamine release. SSx: appears like amphetamine OD with serotonergic properties (hyperthermia, rhabdo, bruxism, hyponatremia). Rx: supportive (IVF, benzos, cooling, intubation/paralysis PRN)
Review the general pathophysiology, Dx, and Tx of GHB intoxication	"Date rape drug". Rapid onset, acts on specific inhibitory neuroreceptors. SSx: bradycardia, ↓ RR, poor coordination, hypotension, coma, rapid awakening after metabolism of drug. Testing not useful (in blood 6hr, urine 12hr). Rx: supportive, intubate PRN.
Review the general pathophysiology, Dx, and Tx of "bath salts" intoxication	Similar to sympathomimetics with adrenergic effects, users redose often. SSx: hallucinations, tremor, hyperreflexia, VS abnl (↑ HR, HTN, ↑ temp), CNS effects (agitation, psychosis, delirium), bruxism. Rx: supportive with benzos (large doses), nitro, nicardipine (avoid beta blockers), cooling, RSI with rocuronium (avoid succinylcholine given possibility of rhabdo).
Review the general pathophysiology, Dx, and Tx of irritant gas (e.g. chlorine, ammonia, hydrogen chloride) exposure	Causes respiratory tract and mucosal irritation. SSx: cough, SOB, pulmonary edema, and conjunctivitis. Rx: ABCs, O2, bronchodilators, bicarb.
Review the general pathophysiology, Dx, and Tx of alkaline vs. acidic ingestions	<u>Alkaline</u> : liquefactive necrosis → deep injuries, perforation (4-7d), and subsequent stricture. <u>Acidic</u> : coagulative necrosis → limited injury, perforation risk (3-4d), gastric outlet obstruction (2-4w). Rx: NPO, do NOT attempt to induce emesis or neutralize, endoscopy, supportive care.
Dx and Tx: Hydrofluoric acid burns	Common associations: glass etching, electronic manufacturing, rust removal, metal cleaning. SSX: burns cause severe pain, severe skin burns leading to eschar formation; also binds Ca & Mg → severe hypocalcemia & hyperkalemia (muscle spasms, arrhythmia). Rx: copious low-pressure irrigation, calcium gluconate gel and injection. **can cause systemic effects**
Review the general pathophysiology, Dx, and Tx of button battery ingestions	Can generate electrical current against mucosal surface and cause burns; high perforation risk. If found in nose, ear, or esophagus it requires emergent removal; do not use vasoconstrictive agents in the nose (e.g. oxymetazoline) to aid in removal as this accelerates burn. if passed to stomach and asymptomatic can monitor.
Dx and Tx: Beta blocker overdose	SSx: Bradycardia (most common), hypotension, HYPOglycemia (or normoglycemia), HYPERkalemia, AV blockade, QT prolongation, seziures (propranolol). Rx: GI decontamination, atropine, calcium, glucagon, high dose insulin (1U/kg/hr) + glucose, intralipid (if crashing), pacing/pressors (minimally effective).

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Dx and Tx: Calcium channel blocker overdose	SSx: Bradycardia, hypotension (most common), HYPERglycemia (often refractory to even high-dose insulin), AV blockade, any bradydysrhythmia, warm extremities. Rx: GI decontamination, atropine (minimally effective), calcium, high-dose insulin (1U/kg/hr) + glucose (may not be necessary given refractory hyperglycemia), pressors (will need high dose), glucagon, intralipid (if crashing), pacing (minimally effective).
Review the general pathophysiology, Dx, and Tx of cardiac glycoside intoxication (e.g. digoxin, floglove, oleander)	Blocks Na-K-ATPase → ↑ Ca in cell → ↑ contractility. SSx: ANY dysrhythmia possible (PVCs MC, bidirectional VT = pathognomonic, slow Afib), agitation, yellow-tinted vision, hyperkalemia (marker of degree of toxicity). Rx: activated charcoal, Fab fragments (e.g. Digifab, Digibind), IVF, atropine, transcutaneous pacing, AVOID Ca for HyperK (stone heart - occurs on the test but not in real life).
When do you give Dig-Fab in a chronic vs acute digoxin overdose?	chronic: clinical status of the patient, e.g. do they have an arrhythmia? NOT HYPERKALEMIA. Acute: hyperkalemia (which precedes arrhythmias) and an elevated Dig level of about > 10ng/dl
What characteristic EKG changes may be seen with digoxin effect vs digoxin toxicity?	<u>Dig effect:</u> short QT, downsloping ST ("Salvador Dali mustache"), biphasic T wave. <u>Dig toxicity:</u> can do literally anything to the EKG including PVCs, AV block, atrial tachycardia with block, polymorphic and bidirectional VT (pathognomonic but rare).
Name 4 common medications that cause bradycardia and bypotension ("Brady Bunch") in overdose	Beta blockers, calcium channel blockers, digoxin, clonidine
Dx: Depression + seizure + wide QRS	TCA Overdose
Review the general pathophysiology, Dx, and Tx of TCA overdose (amitriptyline, nortriptyline, doxepin)	Mechanism: inhibits reuptake of bioamines (serotonin, norepinephrine, and dopamine), anticholinergic effects, sodium channel blockade (wide QRS), α-1 blockade (vasodilation, hypotension), antihistamine effects (sedation), GABA antagonism (seizures). Rx: supportive, sodium bicarb for wide QRS or dysrhythmia.
What common medications are associated with Na channel blockade?	TCAs (most common), diphenhydramine, propranolol, procainamide, cocaine
Review the general pathophysiology, Dx, and Tx of MAOI toxicity (phenelzine, selegiline)	Blocks breakdown of catecholamines, dopamine, serotonin. Interact with many foods (tyramine - salami, red wine, aged cheese) and drugs (meperidine, cocaine, dextromethorphan, SSRIs, lithium). SSx: sympathomimetic toxidrome. Rx: supportive, IVF, nitroprusside PRN.
<i>What are primary concerns in SSRI overdose?</i>	<i>Serotonin syndrome, delayed seizures, arrhythmia (QTc prolongation), tele monitoring even if asymptomatic</i>
Review the general pathophysiology, Dx, and Tx of carbon monoxide toxicity	Sources: closed space fires, gas heaters/stoves. Path: lefts shit O2 dissociation curve (poor O2 delivery) 2/2 greater binding affinity to Hgb than O2. SSx: flu-like symptoms (multiple people with same ssx), cherry-red skin (with severe toxicity or when dead), O2 sat & ABG PaO2 not accurate. Dx: co-oximetry. Rx: supplemental O2, hyperbaric oxygen therapy (HBOT).

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What are the indications for HBOT due to CO poisoning?	End-organ damage at any COHb level, LOC/coma/seizure, CNS effects (neuro findings, AMS), MI/malignant dysrhythmia, COHb >25% (15% in pregnant women) regardless of sx
What is the half-life of carboxyhemoglobin on 1) room air, 2) 100% NRB, and 3) hyperbaric O2?	Room air: 4-6 hours. NRB: 60-90 minutes. Hyperbaric O2: 30 minutes.
What are the expected O2 sat, PCO2, and PO2 values in CO toxicity?	O2 sat will likely be normal (most pulse oximeters cannot distinguish COHb from oxyHb; PCO2 is unaffected, PO2 is dissolved O2 (not bound) and is unaffected; Co-oximetry will be abnormal.
Review the general pathophysiology, Dx, and Tx of cyanide toxicity	Sources: burning of wool, silk, plastics, smoke inhalation. CN inhibits oxidative phosphorylation, blocks ATP production. SSx: "bitter almond" smell, CV: bradycardia, hypotension, CV collapse, CNS: confusion, seizure, coma, false high O2 sat, cherry red skin. Labs: SEVERE lactic acidosis. Rx: hydroxycobalamine (forms carboxy-B12) OR amyl nitrate → MetHb → binds CN, Na thiosulfate (converts CN → thiocyanate).
What is the appropriate treatment for combined CO and CN toxicity?	O2, ONLY Na thiosulfate or hydroxycobalamine (amyl nitrate & Na nitrate cause methemoglobinemia, which worsens symptoms).
Review the general pathophysiology, Dx, and Tx of methemoglobinemia	State in which Hgb exists in ferric form (Fe3+) → can't transport O2 (Hgb only binds in its Fe2+ form) → low O2 sat (classically 85% regardless of degree of toxicity). SSx: "chocolate" brown blood, central cyanosis. Rx: oxygen, methylene blue (except if G6PD→ hemolysis).
When to give methylene blue for methemoglobinemia?	Cyanotic but otherwise asymptomatic pt w/ methemoglobin levels above 20% OR symptomatic pt w/ methemoglobin levels above 10%
What are potential causes of methemoglobinemia?	Dapsone, nitrates/nitrites, antimalarials, local anesthetics (**teething baby acting normally but has cyanosis bc parent's put benzoicaine containing gel on gums **), analine dyes, phenazopyridine (Pyridium), benzos, well water
how to tell if a baby is cyanotic bc of a congenital heart defect or acquired methemoglobinemia?	-congenital heart defect: on 100% O2 ABG still has low PO2 and the baby becomes cyanotic when they cry are upset. -acquired methemoglobinemia: on 100% O2 pt has high PO2 and is always cyanotic
Review the general pathophysiology, Dx, and Tx of hydrogen sulfide toxicity	Sources: decay of sulfur material (industrial sources, volcanoes, sulfur springs, septic tanks). Path: similar to CN. SSx: "rotten egg smell", industrial worker with unknown cause of LOC. Rx: remove from source, Hydroxycobalamin, amyl nitrate (induce MetHgb), HBOT
Review the general pathophysiology, Dx, and Tx of "metal fume fever"	Association: welder with flu-like illness, SOB worse on Monday and decreased effect with repeat exposure through the week (tachyphylaxis), aka "Monday morning fever." Dx: normal CXR. Rx: supportive.

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Review the general pathophysiology, Dx, and Tx of arsenic toxicity	Associations: wood preservatives, garlic taste/smell after ingestion. Path: decouples oxidative phosphorylation, interfering with ATP production and leading to multisystem organ failure. SSx: GI sx (TRIAD- abd pain, hematuria, jaundice), heme (massive RBC hemolysis), renal failure, shock, arrhythmia, CNS (seizure, ascending flaccid paralysis). Rx: dimercaprol (preferred) or dimercaptosuccinic acid (DMSA).
Review the SSx and Tx of hydrocarbon intoxication (paint thinners, gasoline, chloral hydrate, lighter fluid)	Commonly sniffed, huffed, or bagged; ingestion is usually not toxic but can cause ARDS if aspirated. May cause VF/VT (sudden sniffing death). Rx: beta blockers, supportive care.
What is the difference between sniffing, huffing, and bagging?	<u>Sniffing</u> : from container into nose. <u>Huffing</u> : from impregnated cloth into mouth/nose. <u>Bagging</u> : from plastic bag into nose/mouth.
Review the general pathophysiology, Dx, and Tx of iron overdose	Ferrous sulfate is most common (20% elemental). Path: acts as mucosal corrosive, inhibits oxidative phosphorylation and ATP synthesis. Toxicity: >20mg/kg is toxic, >60mg/kg is lethal. Stages of toxicity: <u>I</u> : GI ssx (0-6 hrs), <u>II</u> : latent; asymptomatic (6-24hrs), <u>III</u> : shock & lactic acidosis (6-72hrs) <u>IV</u> : hepatotoxicity/necrosis (12-96hrs) <u>V</u> : GI scarring & gastric outlet obstruction (2-8wks). Rx: whole bowel irrigation, IVF, deferoxamine (indications: level >500 mcg/dL OR >300 mcg/dL and symptomatic). Note: vomiting predicts toxicity (<4: local, >4: systemic)
Review the general pathophysiology, Dx, and Tx of lead poisoning	Sources: paint, old batteries, occupational exposure. SSx: microcytic anemia with basophilic stippling, abd pain, AMS, seizure, encephalopathy. Dx: peripheral smear, send whole blood lead level, XR: shows lead lines. Rx: chelation tx. PO succimer (DMSA) or IV EDTA (Calcium disodium edetate, given after Dimercaprol)
How and when to treat a child w/ lead toxicity?	No level is safe. ASYMPTOMATIC child w/ lead level of: - 5 to 44mcg/dl: find source and tx it. Get public health involved. -45 to 69 mcg/dl: oral chelation therapy only hospitalize if they don't have a lead free place to go -70mcg/dl: hospitalized for chelation therapy Symptomatic child at any level: hospitalize for chelation tx chelators: -oral: succimer (dimercaptosuccinic acid or DMSA) -IV or IM: calcium disodium EDTA or british anti-lewisite (dimercaprol). **kids w/ encephalopathy get treated w/ both EDTA and BAL** ICU level care is only for encephalopathy/seizures
Review the general pathophysiology, Dx, and Tx of isoniazid (INH) overdose	Inhibits pyridoxine (B6). SSx: hepatotoxicity, metabolic acidosis, seizures/status epilepticus. Rx: IV Pyridoxine
Review the general pathophysiology, Dx, and Tx of lithium toxicity	Usually interactions with drugs that affect renal function (NSAIDs, diuretics, ACE). SSx: GI ssx (acute), neuro ssx (chronic; tremors, AMS), TWI on EKG. Rx: IVF, whole bowel irrigation, HD (renal failure, level >5, neuro sx)

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What can be precipitated by abruptly stopping lithium?	Thyroid storm. Random but testable. Lithium inhibits thyroid hormone release from the thyroid gland. Thus, stopping it abruptly can uncover thyroid pathology that was previously present but "managed" with the lithium.
How does the timing of symptoms in mushroom ingestions impact clinical management?	In general, if pt has symptoms (n/v/d) within 6hr of ingestion it is likely non-toxic. If symptoms start after 6hr hepatotoxicity may occur.
Review the SSx and Tx of cyclopeptide mushroom ingestion (<i>Amanita, Galerina, Lepiota</i>)	SSx: delayed GI sx >6 hrs followed by liver failure , renal failure, AMS, and death. Rx: supportive care, GI decontamination only.
Review the SSx and Tx of monomethylhydrazine mushroom ingestion (<i>Gyromitra</i> - "false morel")	SSx: delayed GI sx (>6 hrs) followed by seizures (think 'gyri' like brain), hepatorenal failure. Most have full recovery. Rx: supportive, ? activated charcoal, benzos and B6 for seizures (can be refractory)
Review the general pathophysiology, Dx, and Tx of muscarine mushroom ingestion (<i>Inocybe, Clitocybe</i>)	Muscarinic effect (cholinergic/"DUMBBBELLS"). Rx: atropine, 2PAM.
What is the key clinical effect (and treatment) of Psilybin mushrooms ("magic mushrooms" <i>Psilocybe, Conocybe, Gymnophilus, Panaeolus</i>)?	Hallucinations, euphoria, agitation. Rx: benzos.
What is the key clinical effect (and treatment) of Coprine mushrooms (inky caps)?	Disulfiram-like reaction. Rx: supportive.
Review the SSx of phenytoin (Dilantin) toxicity	PO: gingival hyperplasia, seizure uncommonly, no cards effect with PO. IV: hypotension (2/2 propylene glycol). Give fosphenytoin instead.
Review the general pathophysiology, Dx, and Tx of carbamazepine (Tegretol) toxicity	Na channel blockade, anticholinergic effect. SSx: ataxia, GI sx, QRS widening, seizure at high doses. Rx: supportive, sodium bicarb if wide QRS
Review the general pathophysiology, Dx, and Tx of benzodiazepine overdose	GABA agonist. SSx: ataxia (MC sign), lethargy, respiratory depression. Rx: supportive, intubate PRN, don't use Flumazenil except in kids (can precipitate withdrawal in pts who use benzos or EtOH normally)
Review the general pathophysiology, Dx, and Tx of barbiturate overdose	GABA agonist. SSx: hypotension, bradycardia, respiratory depression, rhabdo ("barb blisters"). Rx: supportive, intubate PRN.
What meds/ingestions are radiopaque on X-ray?	CHIPES: C hloral hydrate, H heavy metals, I ron/Iodine, P henothiazine, E nteric-coated, S olvents

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Dx and Tx: Neuroleptic Malignant Syndrome	Antipsychotic use. SSx: AMS, "LEAD PIPE" MUSCLE RIGIDITY, hyperthermia, autonomic instability. Rx: supportive (IVF, benzos, cooling), Bromocriptine (dopamine agonist)
Dx and Tx: Serotonin Syndrome	Serotonergic agent use (e.g. SSRI, dextromethorphan) or multi-drug overdose. SSx: AMS, CLONUS/HYPERREFLEXIA, hyperthermia. Rx: supportive (IVF, benzos, cooling), ± Cyproheptadine
Review the general pathophysiology, Dx, and Tx of strychnine poisoning	Associations: gopher poison, adulterant in heroin. Inhibits glycine (similar to tetanus). SSx: agitation, myoclonus, severe and painful muscle contractions, rhabdo, seizures, "awake seizures" Rx: IVF, benzos, paralysis PRN.
Review the general pathophysiology, Dx, and Tx of sulfonylurea overdose (Glipizide, Glyburide)	Stimulates insulin release. SSx: sulfonylureas are long-acting → severe recurrent hypoglycemia → admit for monitoring. Rx: dextrose (IVP ± drip), octreotide (inhibits release of insulin).
Review the general SSx and Tx of insulin overdose	SSx: hypoglycemia. The length of effect and need for monitoring is determined by the half-life of the type of insulin. Rx: glucagon, dextrose PRN.
What are common toxic causes of hypoglycemia?	Ethanol (especially in kids), insulin/hypoglycemics (NOT metformin), beta blockers, salicylates, quinine
Review the general pathophysiology, Dx, and Tx of metformin overdose	Inhibits gluconeogenesis → reduces hepatic glucose output → converts glucose to lactic acid → lactic acidosis. Rx: bicarb, lasix (increasing excretion), HD PRN.
Review the general pathophysiology, Dx, and Tx of theophylline toxicity	Methylxanthine derivative (like caffeine) & beta agonist. metabolized by hepatic CYP450 enzymes (meaning that there are many drug interactions). SSx: hypotension, dysrhythmia (MAT = classic), seizures. Rx: IVF, beta blocker, consider HD in severe intoxication.
Review the general pathophysiology, Dx, and Tx of hydrogen peroxide ingestion	Toxicity occurs with industrial strength concentrations; SSx: stroke-like sx (cerebral gas embolism). Rx: hyperbaric oxygen.
Antidote for acetaminophen toxicity?	N-acetylcysteine (NAC)
Antidote for aspirin toxicity?	Bicarb, HD
Antidote for beta blocker toxicity?	Glucagon, Insulin
Antidote for calcium channel blocker toxicity?	Calcium, glucagon, insulin
Antidote for carbon monoxide toxicity?	Oxygen (or hyperbaric O2 if severe and available)
Antidote for cyanide toxicity?	Hydroxycobalamine OR triple therapy (sodium nitrite, amyl nitrate, sodium thiosulfate)
Antidote for digoxin toxicity?	Digoxin Fab (e.g. Digibind, Digifab)
Antidote for ethylene glycol and methanol toxicity?	Ethanol or fomepizole
Antidote for benzodiazepine toxicity?	Flumazenil (but can cause seizures in pts otherwise taking benzos)
Antidote for opioid toxicity?	Naloxone

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Antidote for malignant hyperthermia?	Dantrolene
Antidote for serotonin syndrome?	Cyproheptadine, benzos
Antidote for neuroleptic malignant syndrome?	Dantrolene, Bromocriptine, benzos
Antidote for anticholinergic syndrome?	Physostigmine, benzos
Antidote for iron toxicity?	Deferoxamine
Antidote for mercury toxicity?	Dimercaprol or dimercaprosuccinic acid (Succimer)
Antidote for lead toxicity?	Dimercaprol, EDTA, or dimercaprosuccinic acid (Succimer)
Antidote for isoniazid toxicity?	Vitamin B6 (pyridoxine)
Antidote for organophosphate toxicity?	Atropine, 2PAM
Antidote for valproic acid overdose?	L-Carnitine
What treatments for hyperkalemia and bradycardia are classically contraindicated in digoxin toxicity?	Hyperkalemia: don't give calcium - rare risk of "stone heart." Bradycardia: don't do transvenous pacing - associated with increased ventricular arrhythmias 2/2 irritable myocardium.
What marker predicts mortality in digoxin toxicity?	Hyperkalemia > 5.0
Dx and Tx: Opioid withdrawal	Reverse opioid effects: tachycardia, HTN, abd pain, N/V/D, sweating, agitation, dilated pupils , piloerection , yawning. Opioid withdrawal is NOT life-threatening except in neonates. Rx: antiemetics and clonidine.
Dx and Tx: Valproic acid toxicity?	SSx: GI sx, AMS. Dx: high VA level, high ammonia. Rx: activated charcoal, L-carnitine, hemodialysis (if renal failure, severe cases).
How do you treat body packers?	Packers - people who intentionally swallowed prepared packets of recreational drugs. Tx: if they are stable, cardiac monitoring and then give them miralax to help the packets come out sooner. Tx: if they are unstable, call surgery.
Toxin smells like garlic?	Arsenic or organophosphates
Toxin smells like almonds?	Cyanide
Toxin smells like fish?	Zinc
Toxin smells like fruit?	Isopropanol, ethanol

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Environmental	
Bizz	Buzz
Differentiate Dx and Tx of chillblains (pernio) from trench foot (immersion foot)	Both are NON-freezing cold injuries. <u>Chillblains</u> : inflammatory lesions resulting from exposure to cold (DRY or damp) SSx: red/blue edematous plaques with itchy, burning pain; Rx: warming, drying, topical steroids, ? nifedipine. <u>Trench foot</u> : nerve and tissue injury resulting from repetitive exposure to WET cold (non-freezing) temperatures → vasoconstriction → ischemia/gangrene; Rx: warming, drying, prevention with dry footwear.
Differentiate Dx and Tx of frostnip from frostbite	Both are FREEZING cold injuries. <u>Frostnip</u> : no ice formation or tissue loss, Rx: rewarming. Frostbite: MC freezing injury. Intracellular ice forms + causes tissue loss (can't initially distinguish the two); Rx: rewarming in warm H2O immersion in a circulating bath (37°C - 39°C), NO DRY HEAT EVER, pain control, Tdap pm, delayed debridement. <u>Blister Rx</u> : debride clear blisters, leave hemorrhagic blisters (simple wound care).
What are the phases of frostbite? How is severity graded?	I : vasoconstriction/ice formation. II : reperfusion of warmed tissues → edema/blisters & dry gangrene. <u>Severity</u> : graded by tissue death. 1st : Epidermis; erythema & edema; minimal pain with rewarming. 2nd : epidermis/dermis; hard edema + clear blisters; mild-mod pain with rewarming. 3rd : hypodermis; hemorrhagic blisters, pale grey ext; severe pain with rewarming. 4th : skin/muscle/tendon/bone; insensate, black/grey painless during rewarming.
Distinguish stages of hypothermia: mild, moderate, and severe	<u>Mild</u> (33-35°C): amnesia, dysarthria, shivering. <u>Moderate</u> (29-32°C): stupor, dysrhythmias, AMS, <u>NO shivering</u> . <u>Severe</u> (22-28°C): dysrhythmias (VFib, susceptibility), loss of DTRs, pulmonary edema, cold diuresis, obtunded. <u>Profound</u> (9-20°C): flat EEG, asystole.
Review common physiologic changes in hypothermia	Hyperglycemia (don't treat initially), functional coagulopathy, irritable myocardium, OxyHb curve left shifted (↑ O2 affinity + ↓O2 delivery)
Review appropriate treatment of severe hypothermia	ABCs: CPR/intubation PRN, 1 round of ACLS/shock, then just rewarm passively (insulating blanket) and actively (Bair Hugger, warm O2, and IVF to 107 F, bladder/stomach/peritoneal/chest tube lavage; ECMO is most effective). Goal rewarming to 86 degrees in arrest. "Not dead until they're warm and dead."
Pathophysiology and prevention: Altitude sickness	Unclear but likely related to hypoxia causes pulmonary vasoconstriction & pulmonary HTN → pulmonary edema; cerebral vasodilation → headaches/acute mountain sickness & ultimately AMS/cerebral edema. Acclimatization causes hyperventilation → respiratory alkalosis + bicarb diuresis. <u>Prevention</u> : acetazolamide causes bicarb diuresis & metabolic acidosis → triggers hyperventilation + speeding acclimatization. Note: young and healthy people are NOT protected from altitude sickness (and in fact are more likely to get it).

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Dx and Tx: Acute mountain sickness	MC high altitude illness. When: >2000m (6500ft). SSx: headache, n/v, anorexia, insomnia. Rx: stop ascent, supportive (fluids, analgesia, antiemetics), acetazolamide/steroids, portable HBO chamber, descent
Dx and Tx: High altitude pulmonary edema (HAPE)	MC lethal altitude illness. When: >3000m (9500ft). SSx: Initial (2-4d at new altitude): cough, dyspnea on exertion; progression: dyspnea at rest (classic), pink-frothy sputum, ↑HR, ↑RR, fever, hypoxia. CXR: patchy alveolar infiltrates (ARDS like). Rx: O2 (first line), nifedipine (for pulm HTN), portable HBO chamber, descent (immediately is not necessary but is only definitively effective treatment). **Acetazolamide is NOT helpful in acute illness.**
Dx and Tx: High altitude cerebral edema (HACE)	Most severe altitude sickness but uncommon. When: >4500m (14000ft)Cerebral edema. SSx: ataxia (early+most sensitive finding), encephalopathy, severe lassitude, AMS, seizure. Rx : requires immediate Rx, descent = definitive (best when pt is still ambulatory), O2, steroids (rescue Rx), portable HBO chamber (e.g. Gamow bag).
What causes barotrauma (diving) related illness?	Illness 2/2 descent/ascent. Explained by <u>Boyle's Law</u> (gas volume = 1/pressure). Volume change is greater closer to surface (rapid change of 30ft near surface worse than deeper down)
Review injuries related to descent (localized "squeeze") and appropriate treatment	<u>Barotitis Externa</u> : edema and hemorrhage to external auditory canal 2/2 blockage; Tx: corticosteroid, dry ear precautions. <u>Barotitis Media</u> : MC diving related disorder. SSx: pain, vertigo 2/2 TM rupture; Rx: decongestants, antibiotics, dry ear precautions. <u>Barotitis Interna</u> : rupture/bleeding of round window. SSx: decreased hearing , vertigo, nystagmus; Rx: ENT consult/eval. <u>Sinus squeeze</u> : frontal sinus = MC. Sinuses = 2nd MCC "squeeze" injuries. SSx: edema, pain, epistaxis; Rx: decongestants. <u>Mask squeeze</u> : petechiae and subQ hemorrhages.
Review injuries related to ascent (localized "reverse squeeze") and appropriate treatment	<u>Barodontalgia</u> : air in dental cavity/filling expands with ascent and causes pain, tooth may fall out. <u>GI barotrauma</u> : excess intraluminal gas causes burping/flatulence. <u>Pulmonary Over-Pressurization Syndrome</u> ("POPS"): pulmonary alveolar rupture → pneumomediastinum, ± pneumothorax. SSx: crepitus, SOB, chest pain. Rx: O2, supportive (needle pm). <u>Arterial Gas Embolism</u> ("AGE"): rupture of air or nitrogen into pulmonary vein + left heart → enter circulation → systemic emboli. SSx: LOC on ascent OR within 10 min of surfacing; can cause ACS, CVA, etc. Rx: 100% O2, supine positioning, hyperbaric O2 therapy. Note: POPS & AGE are caused by ascending without exhaling .
What causes dissolved gas (diving) related illness?	Illnesses related to gas in tissue. Explained by <u>Henry's law</u> (↑ pressure → ↑ gas pushed into solution).

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Review illnesses related to dissolved gas at while diving and appropriate treatment	<u>Nitrogen narcosis</u> : "rapture of the deep" (>30m/100ft). Breathing nitrogen at high partial pressures leads to ↑ nitrogen in CNS with anesthetic. SSx: acts drunk & dumb, may drown 2/2 confusion/behavior. <u>O2 toxicity</u> : ↑ pO2 with depth causes toxicity, usually with deep diving or using Nitrox; SSx: muscle spasm, nausea, vision changes, seizure . Rx: ascent or can prevent by decreasing %O2 in tank.
Review illnesses related to dissolved gas after ascent and appropriate treatment	<u>Decompression sickness</u> (DCS): results from nitrogen dissolved in tissue under pressure precipitating out of solution (joints, lungs vessels) and forming bubbles during decompression. Risk factors: depth of dive, rapidity of ascent, multiple dives, air flight soon after dive. Type I : MSK sx (most common: arthralgia/myalias; most common affected shoulders and elbows ("The Bends"), cutis marmorata rash 2/2 lymphatic obstruction. Type II : pulmonary: dyspnea, cp, cough ("The Chokes"); neuro: vertigo, tinnitus, ataxia ("The Stagers"); spinal cord: paralysis, paresthesias; dermat: pruritus, burning ("Skin Bends"). Rx: supportive (IVF, O2, ASA), hyperbaric O2 (must do this quickly). Prevention: slow ascent wit frequent stops (Navy dive tables)
What is the key to diagnosis of arterial gas embolism vs decompression sickness?	Timing - arterial gas embolism SSx occur within minutes of surfacing, decompression sickness within hours
Dx and Tx: Heat syncope	Standing in heat with peripheral pooling 2/2 vasodilation, and decreased preload causes syncope. Tx: passive cooling, fluids.
Dx and Tx: Heat cramps	Muscle spasms 2/2 dehydration and electrolyte depletion. Rx: rest, passive cooling, fluid replacement, salt replacement.
Dx and Tx: Heat exhaustion	Flu-like symptoms. No CNS changes . Core temp usually <104°F. Rx: passive cooling, rest, IVF, replete electrolytes.
Dx and Tx: Heat stroke	Due to failure of thermoregulatory mechanisms, mortality 30-80%. SSx: CNS dysfunction (AMS, seizure, ataxia) + temp usually >104°F. Labs: LFTs universally abnormal, renal failure, DIC, rhabdo, ATN, pulm edema. Types: " Classic ": nonexertional, minor dehydration, higher mortality, due to high ambient temperature and poor thermoregulatory function, usually in elderly; SSx: dry skin, AMS. " Exertional ": young athletes strenuous exercise in hot environment, higher morbidity; SSx: sweaty, profound dehydration, hypoglycemia. Rx: rapid evaporative cooling is BEST (spray lukewarm water on body and use fans to help evaporate - cold water can cause shivering, Rx: low-dose benzos or thiorazine), ice packs, cold water GI lavage if intubated, IVF (small if "Classic," more if "Exertional"), AVOID pressors, STOP COOLING at 102°F (39°C) to avoid hypothermia overshoot. Note: ASA & tylenol do NOT help - the problem is hyperthermia, not fever.
What are the key differences between heat exhaustion and heat stroke?	<u>HE</u> : temp <104, flu-like ssx, NO neuro ssx. <u>HS</u> : temp >104 + neuro ssx

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Differentiate the SSx and Tx of the different degrees of thermal burn injury	<u>1st degree</u> : superficial, epidermis. SSx: sunburn, redness, blanching, pain, no blisters (NOT counted in TBSA). Rx: NSAIDs. <u>2nd degree</u> : superficial partial, upper dermis. SSx: blistering with pain, intact sensation. Rx: NSAIDs, topical antibiotics. <u>3rd degree</u> : full thickness, hypodermis; charred insensate, eschar formation; Rx: skin grafting. <u>4th degree</u> : deep tissue, muscle/tendon/bone; painless; Rx: skin grafting.
Review how to use Rule of 9s (in adults) to calculate TBSA	9%: head, each arm. 18%: entire leg, front of torso, back of torso; 1% adult palm, child's entire hand, perineum. **Only counts 2nd-4th degree burns**
Review appropriate fluid resuscitation in thermal burns using the Parkland Formula	Resuscitation volume = 4 cc/kg x TBSA% x wt (kg). Rx: LR, 50% in first 8hr since time of burn, 50% over next 16hr. UOP goal >1cc/kg/hr. Children: include maintenance IVF for age <5
What additional injuries are commonly associated with thermal burn injuries?	<u>Inhalation injury</u> : higher risk if enclosed space; inhaled toxins cause edema & loss of surfactant; SSx: cough, stridor, hypoxia, carbonaceous sputum but sx are often delayed; Rx: intubate early due to anticipated airway edema. Consider CN & CO exposure, traumatic injuries, rhabdo (esp. with electrical burns or 4th degree thermal burns, DIC).
Review key components of treatment of thermal burns	Intubate early pm (e.g. inhalational injury), IVF: time starts from burn Parkland formula, check for associated traumatic injuries, update tetanus, keep warm, provide wound care but no need for prophylactic antibiotics
Appropriate management of restrictive full thickness burns (respiratory compromise or decreased peripheral perfusion)	Immediate escharotomy
What are indications for referral to a burn center?	2nd degree burns > 10% TBSA or ANY 3rd/4th degree burn; burns involving hands, feet, genitals/perineum, face, major joints; electrical/chemical/inhalational burns; all circumferential burns; pts with significant comorbidities
What is the appropriate treatment for tar burns?	Cool tar and remove with emulsifier (return to rinse in 24hr), do not peel off
Review the key differences between high vs. low voltage electrical injuries	Voltage = Current x Resistance. Household circuits are low voltage (110V) but more accessible; high voltage/industrial (1000V) is more dangerous. Electricity will travel the path of least resistance (preference for nerves, blood vessels, wet skin). Damage is therefore usually deep with little evidence of surface damage. Most damage is 2/2 heat produced by resistance (bone, tendon).
Review the key differences between AC and DC current	<u>AC</u> : more lethal; 1k-10k volts; back-and-forth current; electrical outlets. <u>MSK</u> : sustained contractions (tetany): flexor>extensor → draws person to source, posterior shoulder dislocation; <u>CV</u> : Vfib; Burns: mre extensive; Myoglobinuria/fasciotomy: common. <u>DC</u> : 10mil-2bil volts; single direction; batteries/lightning; single powerful muscle spasm → traumatic fx as ot is thrown from source; <u>CV</u> : asystole (acts like defibrillator); Myoglobinuria/fasciotomy: rare

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Review primary clinical concerns with electrical injuries	Arrhythmia/asystole, deep burns, rhabdo, associated trauma, vascular spasm and thrombosis, AMS/seizure, delayed peripheral neuropathies
Review appropriate management of electrical injuries	<u>Low voltage</u> : asymptomatic → home without testing; mild symptoms → cards monitor, EKG and urine dip (r/o rhabdo) → d/c if normal. <u>High voltage</u> : ALL pts: labs, urine dip, CPK, CTb if AMS, tetanus for burns, admit ALL.
Review clinical concerns and appropriate treatment for pediatric commissure burns of the mouth	Association: Kid chews on cord and gets burns at corner(s) of mouth. Concern for delayed bleeding of labial artery (day 5); ok for d/c if no LOC, no other injury, normal EKG, tolerating PO and reliable parents - instruct parents to hold pressure and return if delayed bleed occurs. Pt will need outpatient f/u with plastics/OMFS for wound check and further care.
Review the general pathophysiology of lightning injuries	Large DC voltage → asystole + apnea. Other SSx: steam burns, TM rupture (classic), superficial fern-shaped/branching burns, associated trauma, delayed cataracts. **Note: apparently dead lightning strike victims are an exception to mass casualty triage rules, can survive with rescue breathing. → attend to pts with no signs of life FIRST**
Dx and Tx: Leg numbness/weakness and cyanosis after lightening strike	<u>Keraunoparalysis</u> : current goes up one leg and down the other causing vasospasm and neuroparalysis; spontaneously resolves after 6hr
What skin finding is pathognomonic for lightening injury?	Lichtenberg figure: superficial burn or feather pattern from "electrical shower"; usually resolves within 6hr
What is the usual cause of death in submersion injury?	Breath holding → involuntary gasp → aspiration / laryngospasm / LOC → active aspiration of fluid leading → loss of surfactant / hypoxia / ARDS → death. Also causes airway obstruction and metabolic acidosis (MC abnormality), delayed pneumonia. Consider associated trauma.
What is the mammalian diving reflex?	More common in children. Sudden submersion in cold water causes bradycardia, blood shunting to CNS, and slowed metabolism.
What is the appropriate treatment for submersion injury?	<u>Apneic, unconscious, severe respiratory distress</u> : apply BiPAP or intubate, consider ECMO, warm → admit to ICU. <u>Asymptomatic</u> : monitor 2-3hr then home; <u>Mild sx</u> : normal pulse ox & CXR → home after 4hr obs; <u>moderate sx</u> : hypoxia → admit for obs.
What is the difference between a Gray, a Rad, and alpha, beta and gamma rays?	The amount of energy deposited into tissues is measured in Rads. The principle unit of radiation is the gray (Gy); 1 Gy = 100 Rad. <u>α- particles</u> = subatomic particles emitted during radioactive decay. <u>α- particles</u> : larger, do not penetrate clothing or skin, dangerous if inhaled or ingested. <u>β- particles</u> : smaller, can penetrate superficial skin layers. <u>Gamma rays</u> : high energy electromagnetic radiation, dangerous in any form of exposure because they can penetrate tissues very deeply.

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What clinical syndromes are caused by whole body radiation exposure?	<u>Hematopoietic syndrome</u> : >2Gy, first to show injury, most sensitive organ system; SSx: pancytopenia & infection; onset <2 days. GI syndrome: 2nd most sensitive system, >6Gy: n/v, GI bleed; onset hours. CV/CNS syndrome: >10Gy, shock, CNS damage, onset: minutes. CV system primarily affected at high doses. Lymphopenia develops first in all exposures.
How does the absolute lymphocyte count (ALC) predict outcomes?	ALC at 48hrs is key to prognosis; if >1500 good prognosis, if <300 lethal. ALC is also the earliest indicator of acute radiation syndrome.
What medication can be taken before inhaled/ingested radioactive iodine to prevent thyroid cancer?	Potassium iodide
What is the correct approach to decontamination of pt exposed to radiation?	Remove clothing (90%), wash with soap/water, don't abrade skin.
What bacteria cause infection in human, dog, and cat bites and what is the correct antibiotic therapy?	<u>Human</u> : <i>Eikenella corrodens</i> ; <u>Dog & cat</u> : <i>Pasturella multocida</i> . Rx: amoxicillin-clavulanate or ampicillin-sulbactam; alt levofloxacin or metronidazole. Dog bites cause crush injuries; cat bites cause puncture wounds (caution near joints), but have equal rates of infection.
Specific concern and appropriate management of primate bite?	Primates carry herpes simiae ("B virus"). Fatal in humans if not treated early. SSx: paresthesias (early) followed by vesicular rash and encephalitis. Rx: IV acyclovir
Appropriate management of dog bites?	Primary repair for uncomplicated injuries (not contaminated, not crush injury, time<12hrs [face<24hrs], no hands or feet). Rx: abx if bite is on the hand or below the knee (or the victim is high risk).
What type of arthropod bites/stings are most commonly concerning?	Hymenoptera (bees, wasps, hornets, ants): venom contains histamine and proteins potentially leading to anaphylaxis.
Review the following sting reactions: local, toxic, anaphylactic, delayed	<u>Local</u> : redness, swelling, pain (e.g. typical bee sting). <u>Toxic</u> : >10 stings (killer bees, fire ants), ssx: syncope, headache, n/v; can resemble anaphylaxis but WITHOUT generalized hives/edema or bronchospasm. <u>Anaphylaxis</u> : onset within minutes, includes bronchospasm, hypotension, urticarial rash. <u>Delayed</u> : like serum sickness with arthralgias, fever, malaise, occurring 1-2wks later.
What is the appropriate treatment for Hymenoptera stings?	Remove stinger (with tweezers - all venom is already injected), wound care, diphenhydramine, NSAIDs, steroids, and epinephrine (0.3-0.5 mg 1:1000 IM) for anaphylaxis or systemic symptoms. Infection is uncommon so empiric abx are not necessary (though initially stings may look infected).

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<p>Distinguish Dx and Tx of brown recluse vs black widow spider bites</p>	<p><u>Brown recluse</u> (<i>Loxosceles</i>): "violin" on back, warm/dry places in southern American Midwest; they tend not to be aggressive (hence the name); SSx: painless bite, venom is cytotoxic → local tissue destruction (necrotic ulcer is classic), rarely heme abnormalities (hemolysis, DIC); Rx: supportive (wound care), tetanus, NO antivenin. <u>Black widow</u> (<i>Lactrodectus</i>): yellow/red "hourglass" on belly, warm/dry places across the US; they tend to be more aggressive. SSx: painful bite, venom is neurotoxic → ACh + NE release → painful muscle cramping (can mimic acute abdomen), CNS excitation, sweating. Rx: supportive (opioids, benzos), antivenin only for severe pain (risk of anaphylaxis).</p>
<p>Dx and Tx: Scorpion stings</p>	<p>Bark scorpion (<i>Centruroides</i>): most venomous, resides mostly in Arizona. Small scorpions are worse, stings usually occur at night. Venom is neurotoxic (ACh + NE release). SSx: local sx (heightened sensitivity to touch = pathognomonic), sympathomimetic sx (HTN, tachy, hyper salivation, bronchoconstriction), CNS sx (roving eye movements, bulbar neuropathies), somatic effects (fasciculations, muscle spasm); Rx: supportive (opioids, benzos), intubate as needed, atropine for secretions, antivenom prn. Most likely fatal in children.</p>
<p>Distinguish Dx of Crotalid vs. Elapid snake bites</p>	<p><u>Crotalidae</u> ("pit vipers" = rattlesnakes, copperheads, cottonmouths): most US envenomations, ~25% are dry bites. Venom types: cytotoxic & hemolytic. SSx: <u>local</u>- painful, edema/erythema/bullae; <u>systemic</u>- paresthesias, metallic taste; <u>heme</u>- coagulopathy, DIC, thrombocytopenia. <u>Elapidae</u> (coral snakes, sea snakes): "Red on yellow, kill a fellow" is true in US only. Snake must hang on and "chew" to inject venom but you don't usually see bite marks. Venom type: neurotoxic (inds ACh receptors). SSx: broadly think neuro symptoms: bulbar palsies, resp. paralysis.</p>
<p>Distinguish Tx of Crotalid vs Elapid snake bites</p>	<p><u>Crotalid Rx</u>: local wound care, supportive care, q2 coags, update tetanus, consider antivenom for mod-severe envenomations (CroFab-from sheep, allergic rxns not common. <u>Elapid Rx</u>: admit ALL for monitoring of delayed neuro sx, antivenin for eastern coral snakes, intubate and give supportive care PRN.</p>
<p>Dx and Tx: Crotalid compartment syndrome</p>	<p>Rare. Only occurs with bite into deep compartment, causing classic "5 Ps" Rx: antivenin and more antivenin, NOT surgery (unless progressive despite antivenin).</p>
<p>What is the correct advice for initial first aid (prior to ED) for snake bites?</p>	<p>Get away from snake (don't try to catch it), immobilize extremity, minimize movement, compression (air splint or elastic bandage). NO tourniquet unless it is possibly a coral snake bite with neurotoxic venom. Don't try to suck the venom out of the wound either.</p>
<p>When should CroFab be given and what is the appropriate administration?</p>	<p>Severe symptoms after suspected Crotalid bite. Antivenin causes less anaphylaxis than previous versions. Dosing is per venom amountbased on amount of venom, not patient weight; Rx 4-6 vials (slow over 10 min to monitor for anaphylaxis then give the rest over 1 hour), titrate doses to arrest of symptoms (mark skin, repeat labs), monitor for possible rebound.</p>

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<p>Review Dx and Tx of infection by marine microorganisms including <i>Erysipelothrix</i>, <i>Mycobacterium marinum</i> and <i>Vibrio vulnificus</i></p>	<p><i>Erysipelothrix</i>: Gram positive rod in salt water, SSx: "erysipeloid" cellulitis; Rx: cipro or pcn/cephalosporin. <i>M. marinum</i>: acid-fast bacillus in salt water, SSx: "fish tank granuloma" nodules/papules in areas exposed to H2O weeks after cleaning a fish tank; Rx: clarithromycin + ethambutol (TB meds). <i>V. vulnificus</i>: Gram negative rod in salt water, ingestion of oysters or exposure to salt water. SSx: hemorrhagic bullae, necrotizing fasciitis, primary septicemia (e.g. cirrhotic patient eats raw shellfish); Rx: ceftriaxone + doxycycline.</p>
<p>What is the appropriate Tx of jellyfish envenomations (stings)</p>	<p><i>Cnidaria</i>: sea anemones, fire coral, jellyfish, box jellyfish, portuguese man-of-war. Contain nematocysts: venom causes localized pain (MC ssx), erythema, pruritis, arrhythmia (systemic). Rx: deactivating with 5% acetic acid/vinegar x30min OR saltwater rinse (NOT freshwater), scrape off nematocysts, give antivenom if box jellyfish. Portugese man-of war: saltwater then hot water, avoid vinegar/cold water</p>
<p>What is the appropriate Tx of marine vertebrate wounds (e.g. stingrays, lionfish, stonefish)?</p>	<p>Provide local wound care, give antibiotics (cephalexin/doxycycline), r/o retained foreign body, HOT water immersion (>113°F (45°C) deactivates heat labile toxin), no acetic acid; do NOT close wounds.</p>
<p>Distinguish ciguatera vs. scrombroid vs. paralytic shellfish intoxications</p>	<p><u>Ciguatera</u>: small fish feed on coral reef dinoflagellates (contain heat stable toxin) → larger, carnivorous fish (barracuda, grouper, red sapper, parrotfish, amberjack) feed on smaller fish; seen Hawaii, Florida, Caribbean; SSx: onset within 6 hrs; GI sx: n/v/d, abd pain; neuro sx: hot/cold reversal (cold alloynia), sensation of tooth looseness, ataxia, coma; CV: bradycardia, hypotension, pulmonary edema; Rx: supportive care, IVF (hypotension), atropine (bradycardia), mannitol (neuro ssx). <u>Scombroid</u>: histamine-like toxin (heat stable) from poorly refrigerated dark-meat fish (tuna, mackerel, yellowtail); caused by bacterial breakdown of histidine → histamine; SSx: tastes "peppery," causes rapid onset (10-30 min of meal) with flushing HA, abd cramps, n/v/d, often occurs in multiple people with same symptoms (as opposed to allergic reaction); Rx: IVF, antihistamines. <u>Paralytic shellfish reaction</u>: toxin from dinoflagellates in "red tide" summer, blocks Na conduction; SSx: rapid onset n/v/d, paresthesias and paralysis; Rx: supportive</p>
<p>Tarantula bites?</p>	<p>Mostly pain at the bite site. Usually very little swelling at the bite site. They have "urticating hairs" which can cause localized allergic reactions.</p>

Obstetrics & Gynecology	
Bizz	Buzz
What size ovarian cyst is high risk for torsion?	>3cm and <10cm
What is the cause of injury in ovarian torsion?	Twisting leading to obstruction of VENOUS and lymphatic flow, leading to congestion, edema and ischemia. Arterial obstruction is rare 2/2 dual blood supply.
What is the most common finding on US with ovarian torsion?	Ovarian enlargement
Most common type of ovarian cyst?	Simple follicular cysts. Thin walled and fluid filled. Present in 1st 2 weeks of the cycle.
Most likely type of ovarian cyst most likely to bleed?	Corpus luteal cyst. Present in the last 2 weeks of cycle. Can cause significant hemorrhage.
What is the next step in management if you have high suspicion for ovarian torsion but a normal ultrasound?	OB/Gyn consult for laparoscopy (gold standard)
Dx: Vaginal bleeding in post-menopausal woman	Gynecologic cancer until proven otherwise
What type of cancer is CA-125 a marker for?	Ovarian
Review the classic presentations for ovarian, endometrial, and cervical cancers	<u>Ovarian</u> : age 50s-60s, gradual subacute abd pain, ascites, CA-125+. <u>Endometrial</u> : vaginal bleeding in post-menopausal women. <u>Cervical</u> : h/o HPV or HIV, postcoital bleeding, abnormal cervix on pelvic
What is the age range for administering the HPV vaccine?	9-26yrs
What are the most common causes of vaginal bleeding in prepubertal female?	Vaginitis, anovulation, trauma or foreign body (malodorous and slightly bloody); consider vaginal foreign body (usually toilet paper) in young girl who just started school and has bloody foul smelling discharge.
What are the most common causes of vaginal bleeding in reproductive female?	Menses, pregnancy, anovulation; less likely fibroids, exogenous hormones
What are the most common causes of vaginal bleeding in perimenopausal women?	Anovulation, fibroids, cervical and endometrial polyps, thyroid dysfunction
What are the most common causes of vaginal bleeding in postmenopausal women?	Endometrial cancer, exogenous hormones, atrophic vaginitis
What is a potential medication for severe non-pregnant vaginal bleeding?	IV premarin (estrogen)

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Middle aged female with "ball coming out of vagina"?	Uterine prolapse or cystocele; worse with valsalva. Rx: pessary or surgery.
What patient population has a higher risk of uterine fibroids?	African-American women
What is the difference between menorrhagia and metrorrhagia?	Menorrhagia is excessive flow (heavy periods), metrorrhagia is irregular cycles.
Dx: Abdominal pain and tenderness, vaginal discharge, cervical motion tenderness	Pelvic inflammatory disease (PID). C. trachomatis = MCC, N. gonorrhoeae, polymicrobial. Dx: consider pelvic US to r/o TOA. **Note that they won't always have CMT, but may just have focal uterine or adnexal pain.** Tx: Ceftriaxone IM, 14 days of doxycycline and metronidazole.
PID and RUQ shoulder pain	Fitz-Hugh-Curtis syndrome (perihepatitis): infection to perihepatic space causing liver capsule inflammation and adhesions. SSx: RUQ + PID ssx. Chlamydia = MCC. CT: ±"violin-string" adhesions, usually normal LFTs. Rx: just like PID, 250mg ceftriaxone IM and 14 days of doxycycline (plus or minus metronidazole). Make sure no concerns for TOA.
Most common GYN problem in children	Vulvovaginitis
Dx and Tx: Vaginal discharge and clue cells	Bacterial vaginosis. <i>Gardnerella/anaerobes</i> . <u>Amsel criteria (3 out of 4 means positive test)</u> : thin, white d/c, "clue cells", vaginal pH >4.5, fishy odor (+whiff test). Rx: metronidazole.
Dx and Tx: Vaginal discharge and pseudohyphae on wet mount	Candidal vaginitis: overgrowth of normal flora, causing pruritis, "cottage cheese" discharge; Rx: fluconazole.
What other conditions are associated with frequent yeast infections?	Diabetes, HIV, pregnancy, antibiotics, steroids
Dx and Tx: Vaginal discharge and "strawberry cervix"	<u>Trichomoniasis</u> : protozoal infection. SSx: "frothy yellow-green" discharge. Dx: pH >5, WBCs, motile trichomonads. Rx: PO metronidazole.
What is the definitive treatment for Bartholin's cyst/abscess?	Marsupialization; in ED do I&D of abscess, place Word catheter
Dx and Tx: Early pregnancy with big uterus and high hCG	<u>Hydatidiform mole</u> : painless vaginal bleeding, uterus bigger than dates, hyperemesis, preeclampsia; Dx: hCG >100,000, US: "grape-like vesicles", "snowstorm"; Rx: chemo, radiation, or surgery. Complication: high risk of malignancy (choriocarcinoma).
What is the ultrasonographic and prognostic difference between partial and complete hydatidiform moles?	<u>Partial</u> : nonviable fetus, <5% become malignant. triploid karyotype. <u>Complete</u> : "snowstorm" appearance on US, 20% become malignant. Diploid karyotype.
Asymptomatic bacteruria in pregnancy	Treat it.
Most common side for pyelonephritis in pregnancy?	Right sided. 70-80% are right sided. Pregnant women are much more likely to have pyelonephritis than non-pregnant women,

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Describe the meaning of the following components of labor progression: dilation, effacement, station	<u>Dilation</u> : opening of cervical os, up to 10cm. <u>Effacement</u> : thinning of the cervix, up to 100%. <u>Station</u> : fetal presenting part location, ranges neg (above) to positive (below) cm relative to ischial spines (0).
Review the stages of labor 1-4	1: regular contractions to full cervical dilation (10cm). 2: full dilation to delivery of infant. 3: delivery of infant to delivery of placenta. 4: recovery/treatment of lacs/tears/hemorrhage.
How long does stage 1 of labor last? Second stage? Third stage?	First time: 6-20hrs; multiparous: 2-14 hrs / Second stage: primps: 30mins-3hrs; multips: 5-60mins/ third stage: everyone: 0-30mins.
What is the concern with late decelerations on tocodynamometer monitoring during labor?	Uteroplacental insufficiency
What is the concern with variable decelerations on tocodynamometer monitoring during labor?	Cord compression
Review the initial management of low FHR on toco monitoring	Change mom's position (left lateral is best), give oxygen, stop any supplemental oxytocin
What are signs of placental separation during Stage 3 of labor	Cord lengthening, fresh flow of blood, uterus becomes firm/globular, fundus rises
What vessels are present in a normal placenta?	3 vessels total: 2 arteries and 1 vein
Review the protocol for Apgar scoring	Max 10pts measured , 0-2pts for each: Appearance (pink, acral cyanosis, central cyanosis), Pulse (>100bpm, <100bpm, absent), Grimace (crying, grimace on suctioning, no response), Activity (flexing BUE and BLE, weak tone, flaccid), Respirations (robust cry, weak cry or irregular/gasping, not breathing)
What are management options for dystocia?	(Dystocia = abnormal labor, full dilation but can't deliver fetus); C-section, oxytocin, forceps/vacuum delivery, maneuvers
What is the appropriate management for shoulder dystocia?	Inability to deliver anterior shoulder 2/2 impaction against mother's pubic symphysis. "HELPER": H elp (call OB, neonatology, anesthesia), E pisiotomy, L egs flexed (McRoberts maneuver - hyper flexing and abducting the mother's hips), P ressure (suprapubic), E nter vagina (Rubin/Woods screw maneuvers - push anterior shoulder into infant's chest, grab the posterior scapula and rotate shoulder 180 deg), R emove posterior arm. Final option is to break infant's clavicle; can also place Foley to empty the mother's bladder.
What risks are associated with c-section compared to vaginal delivery?	Higher risk of thromboembolism, bleeding, infection, longer hospital stay/recovery
What is the appropriate management of a nuchal cord?	Prevent compression of cord by gently reducing it over the head (loose) or clamping and cutting the cord (tight) with rapid delivery of the fetus

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What is the appropriate management of a cord prolapse?	Obstetrical emergency. Elevate presenting part to reduce cord compression (mother in knee-chest position or Trendelenburg), perform immediate C-section. Overall high fetal morbidity/mortality.
What risks are associated with breech presentation?	Higher risk of cord prolapse, premature rupture of membranes, dystocia
What defines postpartum hemorrhage?	10% drop in Hct or blood loss requiring transfusion; typically 500cc for vaginal birth, 1L for c-section
What are the most common causes of postpartum hemorrhage based on timing of presentation (<24h or >24h)?	Early (<24hr): **uterine atony** (most common), lacerations, retained POC; Late (>24hr): retained POC, lacerations
What is the appropriate management of postpartum bleeding 2/2 uterine atony, lacerations, and retained products, respectively?	<u>Atony</u> : MCC < 24hrs; Rx: fundal massage, oxytocin, IVF, NO MAG. <u>Lacerations</u> : birth trauma = 2nd MCC. Rx: surgical repair. <u>Retained products</u> : early or late bleeding; Dx: US; Rx: surgical removal. All: transfuse as needed.
What is the incidence of postpartum depression?	Up to 50%, overall underdiagnosed
Dx and Tx: Uterine rupture	Higher risk: trauma, previous c-section or trauma. SSx: fetal distress, palpation of fetal parts, shock. Dx: US, nonreassuring FHT = most reliable sign. Rx: emergency C-section (and likely hysterectomy).
Dx and Tx: Fever and abdominal pain 2-3d post-partum	Endometritis (infxn of decidua). Polymicrobial. MC post-partum infxn; others: PROM>24hrs, multiple pelvic exams. SSx: foul-smelling lochia, uterine ttp, leukocytosis. Tx: IV abx, admission.
What are risk factors for endometritis?	C-section (MCC), PROM, prolonged labor, internal monitoring, absence of prenatal care, high number of cervical checks
Most common cause of third trimester vaginal bleeding?	Abruption placentae - premature separation of placenta
What can increase the risk of placental abruption?	HTN (MCC), preeclampsia (most significant risk factor) sympathomimetic (cocaine , meth), trauma, high parity, smoking, heavy EtOH, advanced maternal age
Dx and Tx: Placental abruption	SSx (TRIAD): vaginal bleeding (but **may be concealed = painless**) + painful uterine contractions + fetal distress . Dx: US to rule out placenta previa before pelvic examination; Labs: thrombocytopenia, hypofibrinogenemia (DIC = MC complication). Rx: fetal monitoring, Rho(D) immunoglobulin, <u>stabilize mother</u> (IVF, blood), <u>non-reassuring mother/fetus</u> = c-section, <u>term+stable</u> = delivery (vaginal or OR), <u>preterm+stable</u> = inpt conservative mgmt
What is the most sensitive test for predicting placental abruption? Most specific?	most sensitive: toco monitoring // most specific: ultrasound but US misses a lot of them especially if there is a retroplacental clot. MRI is a good test but it's only for stable patients. IF YOU THINK SOMONE IS ABRUPTING: CALL OB AND START TOCO.
What's the best way to determine fetal distress after trauma?	cardiotocography. Monitor all viable women for 4 hrs. If there are less than 3 contractions per hour for four hours, then it's okay to go home. Also, no late decelerations or baby bradycardia.

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Most common cause of painless vaginal bleeding during pregnancy?	<u>Placenta previa</u> : placenta partially or completely covering cervical os, which causes bleeding when the os starts to dilate
What are risk factors for placenta previa?	Prior c-section, high parity, multiple induced abortions, advanced maternal age
What percentage of placenta previa diagnosed on US before 20wks will resolve spontaneously?	50%
What distinguishes PROM and PPROM?	Premature rupture of membranes (PROM): rupture of membranes before onset of labor >37wks. Preterm PROM (PPROM): PROM <37wks
What are potential complications of PROM?	Infection (chorioamnionitis), cord prolapse
What are methods to confirm rupture of membranes?	<u>Clinical</u> : **least specific** "gush" of fluid, pooling of amniotic fluid in vaginal fornix. <u>Nitrazine paper</u> : pH > 7 turns paper blue, has high false positive rate from using lubricant on speculum, sperm, trichomonas infection, etc ...); <u>Ferning test</u> : **MOST SPECIFIC** dried secretions will show branching pattern of crystallization (ferming = amniotic fluid)
For what OB conditions are digital pelvic exams in ED contraindicated?	Placenta previa, suspected premature rupture of membranes (requires sterile speculum)
What is the treatment for PROM and PPROM?	If full term or late preterm (34-37 weeks): admit, continuous fetal monitoring, induce labor. <27 weeks: expectant management (if no infxn). 24-34wks: corticosteroids (hasten lung delivery)
What medications can be given for premature/preterm labor?	<u>Premature/Preterm labor</u> : contractions + cervical changes < 37 wks. Rx: Tocolytics (Mag [IV 4-6g then infusion], terbutaline). Don't delay labor if there is concern for other serious OB complications or the fetus is nonviable. Consider steroids to promote lung development.
What medications are typically used (and safe) in pregnancy for HTN?	Alpha-methyldopa, labetalol, hydralazine
What distinguishes chronic HTN vs. pregnancy-induced HTN vs. preeclampsia/eclampsia?	<u>Chronic HTN</u> : onset prior to pregnancy or before 20wks. <u>Pregnancy-induced HTN</u> : onset >20wks but no sx. <u>Preeclampsia/eclampsia</u> (vascular endothelial dysfunction): HTN >20wks (up to 6 wks post-partum) and sx to include proteinuria, edema, seizures.
What is the time range in which pregnant women are at risk for preeclampsia/eclampsia?	20wks gestation until 6wks postpartum
What are risk factors for preeclampsia/eclampsia?	First pregnancy, <20y/o or >35y/o, multiple gestation (e.g. twins), HTN, DM
What are typical clinical findings in preeclampsia?	HTN, proteinuria, edema (don't need all 3)

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What defines mild preeclampsia vs. severe preeclampsia vs. eclampsia?	<u>Mild</u> : BP 140-160/90-110, proteinuria >300mg/24hr but < 5g/24hr. <u>Severe</u> : BP >160-180 or >110 diastolic on 2 occasions 6hrs apart, proteinuria >5g/24hr (or Udip 4+ protein), Cr > 1.1, LFTs 2x normal, pulmonary edema cerebral/visual sx. <u>Eclampsia</u> : preeclampsia + seizures.
What are clinical symptoms for severe preeclampsia?	Headache, blurred vision, RUQ pain, clonus
What is the appropriate treatment for severe preeclampsia or eclampsia?	Emergent delivery, hydralazine/labetolol/nifedipine for BP control, steroids if <36wks (fetal lung development), IV Mg sulfate (4-6g) to treat/prevent seizures. Mag toxicity: neurotoxic (loss of DTRs, resp. failure, asystole); Rx: IVF, calcium
What defines HELLP syndrome and how is it treated?	Hemolysis, Elevated Liver enzymes, Low Platelets (<100); will have schistocytes on smear. Rx: similar to severe preeclampsia/eclampsia with HTN control, Mg, steroids if <36wks, emergent delivery.
Abdominal pain in a woman w/ HELLP?	Subcapsular liver hematoma
What patients are at risk for Rh incompatibility and what is the associated complication?	Rh- mom with Rh+ baby after bleeding event; mom makes antibodies to baby's blood, causing immune response to future Rh+ pregnancies. Risk of fetal hydrops (hemolysis causing fetal anemia), usually with next exposure to fetal blood
When should Rh immune globulin (RhoGam) be given during pregnancy?	Usually given to Rh- mom at 28-29wks and delivery; should also be given to Rh- mom with any chance of fetal blood exposure (vaginal bleeding, any trauma, ectopic pregnancy)
How much RhoGam do you give?	Gestational age less than 12 wk: 50mcg// Gestational age greater than 12wks or UNKNOWN gestational age: 300mcg. Must be given w/in 72 hrs of the bleeding event.
How much blood does 300mcg of Rho-Gam neutralize?	300ml. If pt undergoes significant trauma they may need a second dose.
What is the Kleihauer-Betke Test? And who should have it done?	Used for certain Rh- moms to detect and quantify the amount of fetal RBCs in maternal circulation. ONLY used in cases of significant maternal-fetal hemorrhage (test is insensitive, requires 5ml of fetal hgb and it only takes 0.01ml of fetal RBCs to cause maternal Rh sensitization). This is used to see if another dose of Rho-Gam is needed.
Review the definitions of threatened abortion vs. inevitable abortion vs. incomplete abortion vs. missed abortion?	<u>Threatened</u> : vaginal bleeding + IUP + closed os. <u>Inevitable</u> : vaginal bleeding + IUP + open os. <u>Incomplete</u> : vaginal bleeding + open os + some POC expelled/some still in uterus. <u>Complete</u> : vaginal bleeding + closed os + complete passage of POC. <u>Missed</u> : nonviable fetus (no heart tones) aged <20 wks in the uterus for at least 8 wks w/o passage. <u>Septic</u> : infxn of uterus during SAB. Staph infxn. open os with purulent drainage
What is the appropriate management of threatened abortion in the ED?	Confirm IUP (vs. ectopic), refer for serial hCG (should double every 48hr), important if early ectopic possible), US, pelvic rest and opt OB f/u, give RhoGam if Rh- mom

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Dx: Young woman with abdominal pain, +FAST but no trauma	Ruptured ectopic pregnancy
What is the most common location for ectopic pregnancy implantation?	Fallopian tube ampulla
What is the most common cause of ectopic pregnancy?	Adhesions/scarring = most common often from PID, previous surgery; others: previous ectopic (greatest risk factor), IUD, previous abortion, and tubal ligation
What is the discriminatory zone for visualization of IUP on transvaginal and transabdominal US?	Transvaginal: hCG 1500mU/mL. Transabdominal: hCG 2,400-4000mU/mL.
What must be seen on US to confirm an IUP?	Gestational sac AND YOLK SAC; otherwise ectopic is still on the differential
What are the requirements for giving methotrexate to treat ectopic pregnancy?	Hemodynamic stability, gestational sac <3.5cm, no fetal cardiac activity, no evidence of rupture, pt reliable for followup
Pt presents 1 week after being started on methotrexate for an ectopic. What is wrong?	"separation pain" - thought to be from tubal abortion or hematoma formation. EITHER WAY SHE NEEDS AN US and LABS to rule out treatment failure.
What vaccines are safe in pregnancy; what common vaccines are unsafe?	SAFE: Tdap, HepB, Influenza (inactivated). UNSAFE: live virus vaccines including Hep A, MMR, Varicella, Pneumococcal, Polio.
Most effective ER contraception if used 7 days after sexual intercourse?	Copper IUD. Antiprogestin can be used up to 5 days after sexual intercourse, progestin only can be used up 120 hrs but is not as effective as Copper IUD, progestin and combined progestin/estrogen can also be used but are less effective and have more side effects.
What suggests hyperemesis gravidarum?	No strict clinical definition, however nausea and vomiting causing ketonuria and loss of > 5% of body weight are commonly used.
High risk time for fetal radiation exposure?	Between 2-7 weeks, during organogenesis
Pathophysiology, Dx, and Tx: Mastitis/breast abscess	Due to blocked duct and secondary infection (<i>Staph</i> > <i>Strep</i>). SSx: breast pain, fever, erythema and induration. Rx: warm compresses, I&D if abscess is present, antibiotics (dicloxacillin, cephalexin). **Pt should continue breastfeeding**

Renal & Genitourinary	
Bizz	Buzz
What defines acute renal failure?	50% increase from baseline Cr OR 50% decrease in Cr clearance
How do you diagnose a prerenal cause of acute renal failure?	MCC acute of AKI in the community. ↓ Renal hypoperfusion = MCC (ACEI NSAIDs); ↓ Intravascular volume (hypovolemia, sepsis, blood loss, etc). Labs: BUN:Cr ratio >20 and FENa < 1%, urine Na <20, relatively normal UA
How do you diagnose an intrinsic cause of acute renal failure (AKI)?	2/2 pathology within the kidney; acute tubular necrosis = MCC (90%). Labs: BUN:Cr ratio < 20, FENa > 2% (damaged kidney is unable to retain Na), low urine osmolality (injured kidney is unable to concentrate causing dilute urine), granular casts on UA
How do you diagnose a postrenal cause of acute renal failure?	2/2 obstruction of urine outflow (bladder CA, ureteral stone, urethral stricture, BPH = MCC). Labs: relatively normal UA; Dx: ultrasound; post-void residual (>100cc is abnormal)
What is the most likely cause of a code before and after HD in a patient with ESRD?	Before: hyperkalemia. After: hypokalemia or blood loss.
What are indications for emergent HD?	"AEIOU": A cidosis, E lectrolytes (hyperK refractory to medical management), I ntoxication (toxins e.g. ethylene glycol, methanol, Li, etc.), O verload (volume, any pulmonary edema), U remia with symptoms (e.g. pericarditis, AMS, BUN 100 or Cr 10)
What is the initial treatment for bleeding AV fistula?	Apply pressure to the arterial supply proximal to the AV fistula. Give local and IV DDAVP (desmopressin).
What are symptoms of uremia?	Pericardial effusion/tamponade, pulmonary edema, encephalitis, n/v, anemia/bleeding (2/2 platelet dysfunction)
What percentage of kidney stones <5mm will pass spontaneously?	90%
What life threat should always be considered on the differential of a patient with potential kidney stone?	AAA
What is the most common site of impaction for kidney stones?	Ureterovesical junction (UVJ)
What is the composition of most kidney stones and what patients are at increased for these stones?	Calcium oxylate; pts with hypercalcemia (2/2 sarcoid, multiple myeloma, hyperthyroid and hyperparathyroid, cancer), Crohn's disease (2/2 increased oxalate absorption)
Dx and Tx: Struvite kidney stones?	Magnesium-ammonium-phosphate stones. MCC of staghorn calculi. Increased risk with chronic UTIs, caused by urease-splitting bacteria (e.g. <i>Proteus</i>). ± staghorn calculi, ± pneumoturia. Rx: surgical removal, abx

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Dx and Tx: Uric acid kidney Stones?	Increased risk with gout, leukemia, myeloproliferative disorders, tumor lysis syndrome. XR: radio <u>l</u> ucent (don't show up)** Rx: IVF, bicarb to alkalinize urine, surgical removal PRN.
How often is there hematuria on UA with + kidney stone?	75-80%
What are absolute indications for admission for kidney stones?	Obstruction + infection, obstruction + solitary kidney, intractable pain or vomiting, urinary extravasation, hypercalcemic crisis
What is the most common cause of glomerulonephritis?	Post-streptococcal GN
What are signs and symptoms of glomerulonephritis and nephritic syndrome?	Proteinuria, hematuria, edema, HTN, renal failure (AKI/intrinsic); UA may show red cell casts. Tx: largely supportive, find and treat cause.
What is an important secondary risk for patients with nephrotic syndrome?	Thromboembolism 2/2 loss of anticoagulant proteins in urine
What are the signs and symptoms of nephrotic syndrome?	"NAPHROTIC": <u>N</u> a decrease (hypoNa), <u>A</u> lbumin decrease (hypoalbuminemia), <u>P</u> roteinuria (>3.5g/day), <u>H</u> yperlipidemia, <u>R</u> enal vein thrombosis, <u>O</u> rbital edema, <u>T</u> hromboembolism, <u>I</u> nfection (lose Ig's in urine), <u>C</u> oagulability (lose ATIII in urine). Rx: IVF, Na restriction, steroids, ACE-I (dilates efferent arterioles, reduces glomerular pressure, and decreases protein loss), VTE prevention
What are the most common causes of nephrotic syndrome in kids and adults?	Kids: Minimal change disease. Adults: Focal segmental glomerulosclerosis
Painless hematuria?	In old men: Bladder cancer followed by renal cancer. In children: glomerulonephritis. Young adults and older women: UTI.
Dx: UTI + fever	Pyelonephritis; cystitis rarely presents with fever.
Dx: UA with WBC but no bacteria	Think of STIs and non-urinary causes (appy, diverticulitis, etc.)
Interpretation of +nitrites on UA	Specific for nitrite reducing bacteria - Gram negative infection (esp. <i>E. coli</i>), not sensitive
What distinguishes direct from indirect inguinal hernias?	<u>I</u> ndirect: through inguinal canal into scrotum (lateral to inferior epigastric arteries). <u>D</u> irect: through muscle of abdominal wall.
What are potential complications of hernias?	Bowel obstruction, incarceration (hernia gets stuck out), strangulation (no blood flow, dead tissue)
What is the usual cause of balanitis/balanoposthitis?	Inflammation of glans 2/2 fungal infection, less commonly bacterial; seen in uncircumcised men, diabetics, obese.
Cause of bilateral orchitis	Mumps virus
Most common cause (and treatment) of epididymitis/orchitis in young vs. old men?	<u>Y</u> oung (<35yr): STIs; Rx: CTX + azithro x1 OR doxycycline x10 days. <u>O</u> ld (>35 yr): <i>E. coli</i> ; Rx: fluoroquinolone x10 days.
What is Phren's sign?	Relief of pain with scrotal elevation in patients with epididymitis/orchitis
Dx and Tx: Prostatitis	SSx: dysuria, urinary frequency, pain with defecation, tender prostate. **Avoid Foley as this will increase inflammation.** If <35yr cover for STDs, otherwise give cipro.

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What are the key differences between low-flow and high-flow priapism?	<u>Low-flow</u> : most common form; ischemic & painful. Causes: sickle cell (MCC), meds (antipsychotics, penile injections). <u>High-flow</u> : usually painless, arterial. Cause: trauma = MCC.
ABG analysis of ischemic priapism	acidemic (pH < 7.25), hypoxic (pO ₂ <30), hypercapnic (pCO ₂ >60)
What is appropriate treatment for priapism?	Pain control (opiates, dorsal penile v ring nerve block); Intracavernosal aspiration (first line Rx); Intracavernosal phenylephrine (Rx after irrigation has failed), consider terbutaline, and consult urology. In sickle cell patients consider exchange transfusion (but low threshold to drain).
Dx and Tx: Testicular torsion	SSx: Acute severe unilateral testicular pain, n/v/abd pain, scrotal swelling and tenderness, absent cremasteric reflex . Dx: US with Doppler (although this may be normal - trust your exam). Rx: emergent urologic consultation for orchiopexy, can try manual detorsion via external rotation. **Consider this dx in young male child with nonstop crying or abdominal pain.**
What is the appropriate technique for manual detorsion of testicular torsion?	Medial to lateral rotation, "open the book"
What is the most sensitive sign for RULING OUT testicular torsion?	A normal cremasteric reflex
What clinical finding is characteristic of torsion of the appendix testis?	"Blue dot sign" (tender bluish nodule on the upper pole of the testis on physical exam - present in 25%); Dx: US. Rx; scrotal support, NSAIDs
What is the most common misdiagnosis in patients with testicular cancer?	Epididymitis; testicular ca is the most common cancer in men aged 15-35; exam will show a painless, firm, fixed nodule or mass.
What is the characteristic finding on CXR with metastatic testicular cancer?	"Cannonball" lesions in lungs
What are extrarenal problems commonly associated with polycystic kidney disease?	Liver cysts, cerebral aneurysm
What is the most common sign of bladder injury?	Gross hematuria
What medication can cause epididymitis?	Amiodarone
Dx and Tx: Peritonitis in a patient on peritoneal dialysis	Dx: cloudy effluent, UA: 100 WBC, > 50% neutrophils or + Gram stain. Rx: <u>Stable</u> : intraperitoneal antibiotics and continued use of catheter. <u>Unstable</u> : admission + IV abx. All ABx should cover skin flora (Strep and Staph).

Psychiatry & Behavioral	
Bizz	Buzz
Define Axis 1-5 (these are not present in DSM-5, but may still be tested)	Axis 1: psychiatric disorders/ Axis2: personality disorders and intellectual disability/ Axis 3: medical conditions such as Alzheimer's / Axis 4: environmental and psychosocial factors such as homelessness which result in mental health disorders / Axis 5: global assessment of functioning
What is the difference between substance abuse and dependency?	<u>Abuse</u> : compulsion to use substances despite adverse consequences (car crash, arrested, fired). <u>Dependency</u> : life organized around drug use (e.g. AM fix), may have tolerance, withdrawal, social retreat
What are the key differences between anorexia nervosa and bulimia nervosa?	<u>Anorexia nervosa</u> : refusal to maintain normal weight, calorie restriction > purging, associated with successful suicide, bradycardia (dysrhythmia = MCC death), 50% good outcome. <u>Bulimia nervosa</u> : binge eating, often normal weight, purging type vs non-purging type, more likely to attempt suicide (usually not successful), overall better prognosis
What are some potential side effects of frequent purging?	Russell's sign (lesions on knuckles from sticking fingers in throat to activate gag reflex, may swallow toothbrush for same reason), oral lacerations, Mallory-Weiss tears, poor dentition 2/2 stomach acid exposure
What are the classic electrolyte abnormalities associated with eating disorders?	Starvation ketosis, metabolic alkalosis, ↓ Na/Cl/K/Mag/Phos
What is the appropriate treatment for Anorexia vs Bulimia?	<u>Anorexia</u> : IVF, lyte repletion, admit, avoid TPN; <u>Bulimia</u> : CBT + SSRI. **DO NOT GIVE THEM BUPROPION as it lowers the seizure threshold.**
What SSx typically distinguish psychiatric and medical (organic) causes of psychosis?	<u>Psychiatric</u> : auditory hallucinations, flat affect, intact orientation, symptoms are continuous, younger patient, gradual onset. <u>Medical</u> : visual hallucinations, labile affect, ± disoriented, symptoms wax and wane, older patient, abrupt onset
What are the classic positive and negative symptoms of schizophrenia?	<u>Positive</u> : hallucinations, delusions, disorganized speech, catatonia. <u>Negative</u> : blunted/flat affect, poverty of speech, anhedonia, social withdrawal
Dx and Tx: Bipolar disorder	SSx: Mania (or hypomania) + depression, often comorbid with SI and substance abuse. Disorder is thought to have heavy genetic component with environmental influences. Rx: mood stabilizers (e.g. lithium, valproate) and antipsychotics (if psychotic features are present)
Mania vs hypomania?	mania: 1 week / hypomania: 4 consecutive days.
What are the criteria for diagnosis of depression?	Depressed mood x2wks + 4 of "SAD CAGES" (changes in Sleep, changes in Appetite, Depressed mood, poor Concentration, decreased Activity, feelings of Guilt/worthlessness, decreased Energy, Suicidal ideation)

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What are significant risk factors for completed suicide?	"SAD PERSONS": <u>S</u> ex (male), <u>A</u> ge (<19 or >45), <u>D</u> epression (or hopelessness), <u>P</u> revious attempt (most concerning risk factor), <u>E</u> xcessive alcohol or drug use, <u>R</u> ational thinking loss (e.g. 2/2 psych dx, dementia, etc.), <u>S</u> eparated (divorced or widowed), <u>O</u> rganized (or serious) attempt, <u>N</u> o social support, <u>S</u> tated future attempt. Marriage and pregnancy are protective.
Do no self harm contracts work?	No.
What is the most common method of attempted and completed suicides?	Attempted: girls/women, drug ingestions (antidepressants = MC). Completed: boys/men, firearms. **Note: check acetaminophen level on all overdose/SI patients.**
What distinguishes malingering, factitious disorder, and somatoform disorder?	Diagnosis is based on intention and objective. Malingering: Intentional symptoms + gainful incentive (e.g. drugs, money, bed). Factitious disorder: Intentional symptoms + "sick role" incentive (e.g. Munchausen syndrome). Somatoform: Unintentional symptoms + no incentive.
Dx: Kid with unusual presentation of disease, biological mom happy with abnormal results	Munchausen by proxy (form of child abuse)
Generalized Anxiety Disorder?	Frequent and prolonged periods of worry and anxiousness (greater than 6 months).
Panic Disorder	sudden, brief episodes of intense fear that are associated with somatic complaints including nausea, vomiting, diaphoresis, tremor and paresthesias. Make sure to exclude medical causes such as hyperthyroidism, PE, MI.
Agoraphobia	Specific fear of open/public places.
Specific phobia	Clinically significant anxiety or worry that develops in response to a specific situation or object leading to avoidance behavior
What distinguishes the anxiety-related symptoms of PTSD, OCD, GAD, panic disorder, agoraphobia, social phobia, and specific phobia? What is the treatment for all?	<u>PTSD</u> : long-lasting anxiety response following a traumatic/catastrophic event; <u>SSx</u> : sx > 1mo, flashbacks, hyper-vigilance, insomnia, poor concentration, irritable with angry outbursts. <u>OCD</u> : recurrent thoughts, images or urges (obsessions) and repetitive acts (compulsions). <u>GAD</u> : extreme multifaceted and uncontrollable worrying most days >6mo. <u>Panic disorder</u> : frequent panic attacks, at least some of which are not triggered. <u>Agoraphobia</u> : panic attacks triggered by being in (or the expectation of being in) situations that are difficult to escape (e.g. crowds). <u>Social phobia</u> : panic attacks or excessive fear triggered by anticipating or being in situations of social scrutiny. <u>Specific phobia</u> : panic attack + specific fear (e.g. snakes, spiders, enclosed spaces). Rx: cognitive behavioral therapy (CBT) and SSRI; benzos can help abort panic attacks.

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Pt with a panic attack hyperventilates and syncopizes. What lab value and resultant physiologic response is associated with this phenomenon?	Hypocarbica → respiratory alkalosis → cerebral vasoconstriction
What is the difference between delirium and dementia?	<u>Delirium</u> : 2/2 medical problem/drugs/tox; rapid onset, sx fluctuate, last hours to weeks, impaired attention/alertness/orientation, sleep-wake cycle disrupted, agitated, incoherent speech, +delusions/hallucinations. <u>Dementia</u> : gradual onset, slow decline, lasts months to years, attention intact (early in dz), sleepw-wake normal, normal alertness, orientation intact (early in dz), behavior normal, word-finding problems, perceptions intact (early in dz)
Dx: Pt suddenly unable to recall where he lives, but has no other neuro SSx?	Transient global amnesia. Temporary disruption of short-term memory loss. NOT an infarct (no sensory/motor deficits). SSx: sudden onset, pt often repeats questions. Workup negative. Rx: self-resolves without intervention.
Dx and Tx: Delirium tremens	Severe ETOH withdrawal sx + autonomic instability + hallucinations + delirium + seizures. Peaks 2-5 days (~72 hours) after etoh cessation. Rx: Benzos, alt. phenobarbital.
What prophylaxis should be offered to patients after sexual assault?	Pregnancy prophylaxis (e.g. Plan B), STD prophylaxis (no need to test, empiric tx ok; GC/Chlamydia/Trichomonas abx), HBV vaccine , HIV post-exposure prophylaxis. Do not give HIV PEP or Plan B after 3d (ineffective).
Five clinical features of psychosis	1. Delusions 2. Disorganized thinking 3. Hallucinations 4. Negative symptoms 5. Grossly disorganized or abnormal motor behavior
Flight of ideas vs disorganized thinking	<u>Disorganized thinking</u> : seen in psychosis. inferred from speech, pt's answers are loosely related or unrelated, pt switches from one topic to the other, word salad (disorganized and incomprehensible speech). <u>Flight of ideas</u> : seen in mania. They frequently shift from one topic to another w/ continuous, accelerated speech pattern.
What is the difference between paranoid, schizoid, and schizotypal personality disorders? (Cluster A: "weird", odd, and eccentric)	<u>Paranoid</u> : suspicious of others. <u>Schizoid</u> : social detachment with restricted emotions (isolated). <u>Schizotypal</u> : social detachment with eccentric behavior (e.g. magical thinking).
What is the difference between histrionic, narcissistic, borderline, and antisocial personality disorders? (Cluster B: "wild", dramatic, emotional, erratic)	<u>Histrionic</u> : excessive emotional lability & attention-seeking behaviors. <u>Narcissistic</u> : grandiose, constant need for admiration, lacks empathy. <u>Borderline</u> : unstable relationships, labile affect/mood, poor self-image, impulsive, demonstrates splitting (quickly regards others as the "worst" or "best person ever"). <u>Antisocial</u> : disregard for rights of others, frequent lying/cheating/stealing (associated with malingering).

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What is the difference between avoidant, dependent, and obsessive-compulsive personality disorders? (Cluster C: "worried", anxious or fearful)	<u>Avoidant</u> : social withdrawal (awkward/uncomfortable in social situations), constantly feels inadequate, hypersensitive to criticism. <u>Dependent</u> : indecisive (and needs others to make decisions), lacks self confidence, submissive. <u>Obsessive-compulsive</u> : perfectionism/order valued over flexibility/efficiency. <u>Passive-aggressive</u> : forceful, stubborn, dependent on others
What is the difference between somatization, hypochondriasis, conversion disorder? (psychosomatic disorders, all unintentional)	<u>Somatization</u> : unexplained physical complaints, multiple different symptoms of multiple different systems (GI, GU, neuro) with unexplained cause, often affects life. <u>Hypochondriasis</u> : preoccupation with and fear of disease, conviction one is sick, symptoms out of proportion to clinical findings, often displays "doctor shopping." <u>Conversion disorder</u> : SUDDEN unexplained neuro symptoms (e.g. blindness, paralysis), often but not always in response to an emotional stressor. MUST RULE OUT ORGANIC DISEASE IN ALL BEFORE MAKING THESE DIAGNOSES.
What is the most common personality disorder?	Borderline
Dx: Patient with wide variety of complaints, complicated medical history, no clear cause of symptoms	Somatization disorder
Dx: Patient intentionally fakes symptoms (e.g. seizure with quick return to baseline and normal lactate) with goal of hospital admission and workup	Malingering; often present over the weekend or after hours.
Dx: Patient is has recurrent infections of a non-healing wound on their leg despite appropriate ABx treatment and multiple hospitalizations for same, with discharge upon clinical improvement	Munchausen syndrome; may have extensive and complex medical records, often well spoken
Dx: Sudden paralysis after a traumatic event	Conversion disorder
Dx: Drug ingestion + violent behavior	PCP: dissociative agent. SSx: sympathomimetic effects, bizarre & violent behavior, perceptions of superhuman strength; ± horizontal, vertical or rotatory nystagmus. Rx: supportive care, sedate/restrain to ensure pt and staff safety (benzos, AVOID Haldol), monitor for rhabdo and seizures.
What criteria are required for a new diagnosis of schizophrenia?	2+ of the following: delusions, hallucinations disorganized speech or behavior, negative symptoms AND ≥ 6 months; rule out mood disorder and drug abuse.

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What is the difference between a brief psychotic disorder, schizophreniform disorder, schizophrenia, and schizoaffective disorder?	<u>Brief psychotic disorder</u> : psychotic features < 1mo. <u>Schizophreniform</u> : psychotic features for 1-6mo. <u>Schizophrenia</u> : psychotic features >6mo. <u>Schizoaffective</u> : psychotic features + mania or depression
What is the appropriate treatment of an elderly patient presenting with signs of elder abuse who wants to return home?	If they have decision-making capacity they can be discharged, but adult protective services should be notified (in most states MDs are mandatory reporters).
Review the timeline for the following symptoms of alcohol withdrawal: tremor, hallucinations, seizure, DTs	Tremor (6-12hrs), hallucinations & seizures (12-48hrs), DTs (>48hrs)
What are the five stages of grief?	Denial → anger → bargaining → depression → acceptance. Pathological grief reaction if lasting >6 months + causes severe functional impairment, suicidal ideation, psychotic symptoms.
Frontaltemporal neurocognitive disorder	Younger than 65 yo. characterized by disruptive behavioral changes: hyperorality, wondering, and generally disinhibited. Characterized by a rapid decline.
Neurocognitive disorder w/ Lewy bodies	Age: mid-70s. Executive functions and attention are affected. Visual hallucination and REM sleep behavior disorder. Symptoms wax and wane.
Prion Disease	Any age. Neurocognitive deficits + motor abnormalities (Ataxia, myoclonus, dystonia, chorea). Examples: Mad cow disease, kuru, Creutzfeldt-Jakob Dx.

Musculoskeletal / Rheumatology	
Bizz	Buzz
Review Salter-Harris classification for pediatric fractures	"SALTR" describes relationship to epiphyseal plate (picture a bone with diaphysis shown superiorly and joint inferiorly): I : S lip; separation straight across physis. II : A bove; fx through physis and metaphysis). III : L ower; fx through physis and growth plate. IV : T hrough; fx through metaphysis, physis, and epiphysis. V : R ammed; crush injury to the physis. Distal fibula = MC location. More advanced fracture types (=higher number) leads to more likely growth disturbance; I & V can have normal XR, II = MC.
Dx and Tx: Torus or buckle fracture in kids	Incomplete fracture 2/2 impaction/axial load. Dx: buckling or bulging of the cortex on one side of the bone without clear fracture line, with periosteum intact; may have associated angulation. Rx: splint, outpatient ortho f/u.
Dx and Tx: Greenstick fracture	Incomplete fracture 2/2 impaction/axial load, causes fracture line in only one side of bony cortex with opposite side bent but otherwise intact. Rx: splint, outpatient ortho f/u.
Dx and Tx: Toddler fracture	Spiral fx of distal tibia in kids 9mo-3yr (NOT a fracture pattern of abuse)
What fracture patterns suggest child abuse?	Metepiphyseal corner fx, rib fractures (posterior = pathognomonic), fx of sternum/scapula/spinus process, long bone fx in nonambulatory infant, multiple fx in various stages of healing, b/l acute long bone fx, vertebral body fx's without h/o high force trauma, digital fx in children <36mo, severe skull fx's in children <18mo
Spiral tibial fractures in an child that's walking but less than 7 yrs old?	Fx in the distal third of the tibia: this is a CAST fx (Childhood accidental spiral tibia–spiral fractures of the tibia) likely fine. No NAT. Fx in the proximal tibia or femur: suspect NAT
Identify the general sensory and motor functions of the radial nerve	<u>Sensory</u> : dorsal/radial aspect of hand (1st dorsal web space). <u>Motor</u> : wrist extension.
Identify the general sensory and motor functions of the median nerve	Sensory: palm & palmar aspect of distal dorsal digits 1-3.5. <u>Motor</u> : "tea drinking" (pincer grasp, flexor at wrist & elbow, pronators)
Identify the general sensory and motor functions of the recurrent branch of the median nerve	<u>Sensory</u> : NONE (pure motor nerve). <u>Motor</u> : "thumb OAF" - opposition, adduction, flexion
Identify the general sensory and motor functions of the ulnar nerve	<u>Sensory</u> : ulnar aspect of palm & palmar aspect of digits 3.5-5. <u>Motor</u> : hand intrinsic muscles.
Dx and Tx: Adhesive capsulitis (frozen shoulder)	Adhesions between joint capsule and humeral head → stiffness and ↓ ROM; Causes: injury or spontaneous. Rx: Codman's exercises (pendulum swing with light hand weights)

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Dx and Tx: Rotator cuff injuries	Affects SITS muscles (S upraspinatus, I nfraspinatus, T eres minor, S ubscapularis). Cause: repetitive (overuse) movements, trauma. SSx: shoulder pain, cannot ABduct or externally rotate. Dx: XR (r/o other injury), MRI (tears). Rx: NSAIDS, ortho referral for further care. Do not place in a sling, as this increases the risk of developing adhesive capsulitis.
Appropriate management of clavicular fractures	MC fracture in kids. Middle 1/3 = MC location. Dx: CT only additional injury is suspected. Rx: Sling (Peds: figure of 8 sling; adults: sling), outpatient ortho f/u. Non-displaced/minimally displaced: supportive. Skin tenting: reduction to prevent open fx. Open fx: admission, IV, and surgery.
Appropriate management of AC separation	Occurs with direct blow (contact sports). XR: grades I-IV. Rx: <u>Mild to moderate</u> : sling, ortho f/u; <u>moderate to severe</u> :sling, ortho referral, surgery
List potential complications of shoulder dislocations	<u>Anterior</u> : Injury to axillary nerve (check for sensation over deltoid); Hill-Sachs lesion (most common injury; compression fx of posterolateral humoral head); Bankart lesion (tear in glenoid labrium). <u>Inferior</u> : injuries to axillary artery or brachial plexus. <u>Others</u> : Adhesive capsulitis, particularly with recurrent dislocations.
What circumstances increase the chance of a posterior shoulder dislocation?	Most dislocations are anterior; possible posterior if seizure or lightning strike, as the contracting shoulder extensors will be stronger than the shoulder flexors, dislocating the shoulder posteriorly. Pt cannot externally rotate or abduct the arm. Often missed. If it presents late, DO NOT reduce. Call ortho.
List potential complications of humeral head and humeral shaft fractures	<u>Head</u> : adhesive capsulitis (most common), avascular necrosis. <u>Shaft</u> : radial nerve injury → wrist drop, brachial artery injury, difficulty with wrist supination
List Dx and potential complications of a supracondylar fracture	Most common Peds elbow fx (FOOSH). Dx: XR abnormal anterior humeral line (should pass through middle of the capitellum). Complications: high risk for brachial artery injury, compartment syndrome/Volkmann's contracture. Admit any type 3 supracondylar fx. Posterior long arm splint for other supracondylars
What is a Volkmann ischemic contracture?	Consequence of supracondylar fracture. Compartment syndrome leads to ischemic necrosis of the wrist and finger flexors in the forearm, causing the muscles to scar and contract, and resulting in a wrist flexion contracture and claw-hand deformity.
If no obvious fracture is present, what signs on XR might indicate an occult supracondylar fracture (kids) or radial head fracture (adults)?	Anterior fat pad elevation called a "sail sign," (small anterior fat pad is a normal finding), posterior fat pad elevation (always abnormal), abnormal anterior humeral line (should intersect middle 1/3 of capitellum), abnormal radiocapitellar line
Tx for non displaced radial head fracture in adult? Stable supracondylar fx in a child?	adult: sling and early mobility/ child:m long arm posterior splint w/ ortho follow up

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List potential complications of elbow dislocations	2nd most common dislocated joint (shoulder = first). Posterior (ulna is posterior to humerus) > anterior. Complication: brachial artery (rare) & ulnar nerve injury (MCC) and compartment syndrome. Median nerve can be injured as well. Low threshold for CT angiography
Dx and Tx: Nursmaid's elbow	Children 1-3 yo. Caused by axial traction (e.g. parent pulls kid up by his arm) → radial head to jump out of the annular ligament. Dx: no XR needed, Rx: try immediate reduction with hyperpronation and/or supination and flexion, monitor for normal use 30min later.
Review paired fracture/dislocations of forearm: Monteggia, Galeazzi, and Essex-Lopresti	<u>Monteggia</u> : proximal ulnar fracture + radial head dislocation (look for radial nerve injury at radial head dislocation w/ wrist drop). <u>Galeazzi</u> : distal radial fracture + DRUJ disruption. <u>Essex-Lopresti</u> : comminuted radial head crush fracture + DRUJ disruption. Rx: ALL require ORIF. Remember "MUGgER" (Monteggia with Ulnar fracture, Galeazzi & Essex with Radial fractures)
Dx and Tx: Nightstick fracture	Midshaft ulnar fracture 2/2 direct blow (e.g. while trying to protect oneself from being struck with a policeman's nightstick). Rx: r/o Monteggia fx, ACE wrap.
What is the difference between a Colles' fracture and a Smith's fracture?	<u>Colles</u> : distal radius fracture with dorsal angulation usually 2/2 fall onto outstretched hand. <u>Smith's</u> : distal radius fracture with volar (palmar) angulation, usually 2/2 fall onto back of hand. Complication: BOTH with risk for median nerve injury (weak 'pincer grasp.") Rx (both): closed or open reduction, sugar tong splint.
Dx and Tx: Triquetral fracture	2nd most common carpal bone. fx. Dorsal chip fx of triquetrum 2/2 FOOSH. Dx: lateral hand XR. Rx: volar splint, outpatient hand surgery f/u.
Dx and Tx: Scaphoid fracture	Most common carpal bone fx; 2/2 FOOSH. SSx: snuffbox TTP or pain with axial loading of thumb; XR: may be normal. Rx: if in doubt place thumb spica and provide outpatient hand surgery followup. If no overt fx on XR, splint + repeat XR in 10-14d. Complication: avascular necrosis/nonunion
Dx and Tx: Lunate fracture	Dx: Focal TTP of dorsal proximal hand and with axial load of 3rd digit; may have normal XR. Rx: if clinical suspicion should place sugar tong splint with outpatient hand surgery f/u. high risk of avascular necrosis.
Dx and Tx: Scapholunate dislocation	Dx: >3mm widening between scaphoid and lunate, "Terry Thomas (or David Letterman) sign," localized ttp. Tx: splint, hand surgery consultation, usually requires surgical repair
Dx and Tx: Lunate dislocation	Dx: XR with "piece of pie sign" on AP and "spilled teacup" (volar displacement of lunate) on lateral; Rx: splint, immediate ortho consult for reduction, ORIF. Complications: median nerve injury, avascular necrosis.
Dx and Tx: Perilunate dislocation	Perilunate dislocation = capitate dislocation. Dx: Capitate is displaced dorsally with normal lunate alignment over radius. Tx: splint, immediate ortho consult for reduction

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Dx and Tx: Boxer's fracture	Fx of 5th metacarpal neck or shaft. Rx: must repair any rotational deformity, place in ulnar splint, give abx/washout if there is an associated lac/open fracture (do not close). Note that metacarpal neck fractures require reduction with more than 30 degrees of angulation, and shaft fractures require reduction with more than 20 degrees of angulation.
Identify injury in mallet finger, boutonniere deformity, swan neck deformity, jersey finger	<u>Mallet finger</u> : digital extensor tendon disruption ± avulsion fx, unable to extend DIP joint. <u>Boutonniere deformity</u> : slip of central extensor tendon at PIP joint → PIP in flexion + DIP in hyperextension. <u>Swan neck deformity</u> : PIP in hyperextension + DIP in flexion. <u>Jersey finger</u> : flexor digitorum profundus (FDP) avulsion 2/2 hyperextension during active flexion, can't flex DIP. Typically affects the ring finger.
Dx and Tx: Gamekeeper's thumb	Tear/sprain of ulnar collateral ligament. SSx: weak pincher grasp, laxity with valgus stress. Rx: thumb spica splint, refer to hand surgery.
What is a Bennett fracture-dislocation?	Two-part intra-articular fracture of the base of the 1st metacarpal (requires surgery)
What is a Rolando fracture?	Comminuted intra-articular fracture of the base of the 1st metacarpal (requires surgery)
Dx and Tx: Carpal Tunnel Syndrome	Most common entrapment neuropathy of the wrist, compression of the median nerve in carpal tunnel. Risk factors: obesity, female gender; prolonged computer work is NOT. SSx: numbness/weakness first 1-3.5 digits, worse at night, improved with "shaking their hands out." Dx: median nerve compression testing (Durkan's; most sensitive test); Phalen's and Tinel's signs have poor sensitivity/specificity. Rx: wrist splint at night, NSAIDs, hand surgery referral PRN.
What are Kanavel's signs for flexor tenosynovitis? Tx for flexor tenosynovitis?	1. Tenderness along course of the flexor tendon 2. Fusiform ("sausage digit") or symmetrical swelling of the finger 3. Pain with passive extension 4. Flexed posture of the finger. Rx: IV broad-spectrum antibiotics, hospital admission, and immediate hand surgery consultation (operative intervention)
Dx and Tx: Compartment syndrome	Classic "6 Ps" are typically later findings: pain out of proportion (pain on passive stretch = earliest finding) paresthesias (loss of 2 point discrimination = most common exam finding), pallor, paralysis, pulselessness, poikilothermia. Normal compartment pressure: 0-10; ischemic necrosis ≥30-40. Delta pressure (more reliable than direct pressure alone): Diastolic BP - direct pressure; ≤30-40 consistent with acute compartment syndrome. Rx: fasciotomy. [Needle is inserted at 90 degree angle to the skin to get an accurate measurement]
Disposition for high-pressure injection injuries?	Always go to OR despite benign-appearing wound
Contraindications for finger reimplantation	Mangled tissue, >6hr elapsed since injury, fingertip amputation only
Appropriate care of amputated digit	Wrap in saline-soaked gauze, place in plastic bag, put bag in ice water.

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List possible red flag symptoms for concerning cause of low back pain	Trauma, fever, spinal surgery, focal neuro deficits, HIV/immunosuppression, TB, cancer, age >55, symptoms >4wks, IVDA, pain at rest or mostly in the evening, saddle anesthesia, constipation/urinary retention, urinary incontinence
Appropriate management of low back pain if NO red flag symptoms vs. YES red flag symptoms	<u>NO RED FLAGS</u> : short course of pain control, early back to work, no imaging or additional workup needed. <u>+RED FLAGS</u> : start with labs and XR, consider MRI depending on SSx.
Dx and Tx: Ankylosing spondylitis	Classic SSx: 30s-40s y/o, male >female. SSx: AM back pain/stiffness, improved by motion. Association: anterior uveitis = most common, IBD. Dx: XR shows "bamboo spine" (fusion of vertebrae), genetic link to HLA-B27. Rx: PT, NSAIDs.
What finding is most sensitive for diagnosis of cauda equina syndrome?	Urinary retention: Post-void residual (PVR) >50-100cc. Other SSx: saddle anesthesia, sexual dysfunction, neuro deficits, bowel/bladder dysfunction, BILATERAL symptoms.
Identify the spinal cord level and nerve associated with each reflex: Patellar, Babinski, Achilles, Biceps, Brachioradialis, Triceps	Patellar: L3-4 (femoral nerve). Babinski: L4-5, S1-2 (tibial nerve). Achilles: S1 (tibial nerve). Biceps: C5 (musculocutaneous nerve). Brachioradialis: C6 (radial nerve). Triceps: C7 (radial nerve).
Identify the spinal cord level associated with each joint movement: shoulder abduction, elbow flexion, wrist flexion, finger flexion, finger abduction, finger extension, wrist extension, elbow extension	Shoulder abduction: C5. Elbow flexion: C6. Wrist flexion: C7. Finger flexion: C8. Finger abduction: T1. Finger extension: C7. Wrist extension: C6. Elbow extension: C7.
Identify the spinal cord level associated with each joint movement: hip flexion, knee extension, ankle dorsiflexion, great toe extension, ankle plantarflexion, knee flexion	Hip flexion: L2. Knee extension: L3. Ankle dorsiflexion: L4. Great toe extension: L5. Ankle plantarflexion: S1. Knee flexion: S2.
Dx and Tx: Unstable pelvic fracture	Dx: XR shows "open book" fracture (complete separation of the pubic symphysis) or Malgaigne's fracture (two ipsilateral pelvic ring fractures with bilateral sacroiliac dislocation, 2/2 vertical shear). Can cause extensive blood loss, mostly posterior/retroperitoneal and venous. Rx: with pelvic binder, angio or surgery, blood transfusion.
Dx and Tx: Hip fractures	Ground level fall = most common cause. SSx: High risk in elderly (esp. femoral neck), intertrochanteric fx = most common. Exam: ABducted + externally rotated + shortened (classic). XR: shows most; MRI if neg XR + high suspicion + unable to ambulate. Rx: Ortho consult, ORIF

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What is the most common type of hip dislocation? What associated injuries and sequelae should you anticipate?	Posterior (80-90%). Usually due to high-energy force (e.g. MVC). Posterior on exam: hip flexed, ADducted, internally rotated & shortened. On AP xrays you cannot see the lesser trochanter. Dx/Rx: neurovascular compromise + obvious dislocation → reduction without delay or XR; neurovascularly intact → XR. Complications: delay in reduction >6hrs → avascular necrosis; posterior: sciatic nerve injury, acetabular fx; anterior: femoral artery/vein/nerve injury.
what neurological findings are most likely presents w/ a posterior hip dislocation?	Sciatic nerve palsy. Peroneal branch. Injured more than tibial branch. You'll see: foot drop (weakness w/ ankle dorsiflexion and foot eversion) as well as decreased sensation along the entire posterior leg below the knee.
Identify components of the Ottawa Knee Rules	Age >55, isolated patellar TTP, TTP over fibular head, inability to flex knee 90°, inability to bear weight (4 steps) immediately after injury AND in ED. If any of these are true, then get an XR.
Dx and Tx: Osgood-Schlatter syndrome	Tibial tuberosity apophysitis 2/2 trauma or overuse, occurs in M>F, mainly preteens. Dx: localized TTP, clinical Dx, no XR needed (but you may see an avulsion fx of the tibial tuberosity if you get one). Rx: RICE.
Dx and Tx: Meniscal injury	Medial > lateral, occurs 2/2 rotational force. Dx: joint line TTP, feeling of clicking and locking with knee giving way, ± knee effusion, +Appley Grind Test and McMurray's; can get outpatient MRI to confirm. Tx: RICE, NSAIDs, surgery for refractory SSx.
Dx and Tx: Cruciate ligament injuries	Occurs when pivoting while running/cutting; ACL tear = most common. SSx: audible "pop," followed by knee instability, hemarthrosis. Dx: + Lachman's test (most sensitive test); Dx: XR then ± outpatient MRI. Rx: leg immobilizer ONLY if very unstable, non-weight bearing, ortho referral; ACL tears are associated with Segond fx (avulsion at lateral tibial plateau, Rx: knee immobilization).
Dx and Tx: Tibial plateau fx	Occurs with high force blow to tibia (e.g. MVC or pedestrian vs. auto). SSx: localized TTP, eval for popliteal artery injury; Dx: XR often negative, get CT if significant clinical suspicion exists. Dx: ORIF, place knee immobilizer, give crutches (pt should be totally non-weight bearing), and place ortho consult.
Dx and Tx: Knee dislocation	Don't confuse with patella dislocation! Dislocation of tibia in relation to fmur. SSx: 50% spontaneously relocate prior to ED eval, so have a high index of suspicion esp. if bicruciate instability is found on exam; Types: anterior (tibia's relation to femur) = most common; Dx: CTA = study of choice. Rx: knee immobilizer, ortho consult. Complications: popliteal artery injury, common peroneal nerve injury (foot drop)
Potential nerve injury resulting from knee injuries (e.g. prox fibular fx, tibial plateau fx)	Deep peroneal nerve: unable to dorsiflex and loss of sensation to 1st web space
Identify components of the Ottawa Ankle Rules	Unable to walk (4 steps) immediately after the injury AND in the ED, bony TTP over posterior medial malleolus or posterior lateral malleolus, TTP of navicular or base of 5th metatarsal. XR if any of these are positive - follow this for exam purposes.

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Most commonly injured ligament during ankle sprains?	Anterior talofibular → calcaneofibular → posterior talofibular. The lateral collateral ligament complex is made up by these three ligaments. The ligaments rupture in an anterior to posterior direction with spraining.
Dx and Tx: Maisonneuve fracture	Medial malleolus fx (or deltoid ligament injury) + proximal fibular fracture. Rx: requires splint, non-weight bearing, and ortho referral for ORIF.
Dx and Tx: Jones fracture (vs pseudo-Jones/dancer's fx)	<u>Jones fracture</u> : Fx of proximal diaphysis of 5th metatarsal (1.2-3cm distal to proximal tuberosity); Rx with post-mold and non-weight bearing, usually requires surgery. <u>Pseudo-Jones/dancer's fracture</u> : avulsion at base of 5th metatarsal; Rx: splint and refer for ortho followup, nonsurgical.
Dx and Tx: Lisfranc fracture/dislocation	Unstable midfoot due to disruption of the Lisfranc joint (arch of the foot), usually from high-energy shearing force (e.g. falling from a horse with your foot in a stirrup) or with axial loading onto a hyper-plantarflexed foot. Dx: check if 1st and 2nd cuneiform bones line up with metatarsals on AP/lateral/oblique films, fleck sign (avulsion fx of 2nd metatarsal= pathognomonic), consider stress view if high suspicion. Rx: splint, non-weight bearing, and most require surgery.
Dx and Tx: Achilles tendon rupture	Associated with landing from jumping (e.g. in basketball). Risk factors: chronic steroids, fluoroquinolones. SSx: feels audible pop over Achilles tendon. Dx: consider US for dx of partial tears, +Thompson squeeze test (most sensitive sign). Rx: Splint in equinus (plantarflexion), complete tears require surgery.
Dx and Tx of septic arthritis	Knee = most common joint (50%). Hematogenous spread = most common. Bacterial = most common cause (Staph aureus = most common overall; N. gonorrhoea = most common in young, sexually active adults <35). SSx: acute joint pain (esp. with passive ROM) & swelling with fever. Dx: arthrocentesis ; use Kocher criteria in kids (see Peds section). Rx: IV abx, ortho consult with surgical drainage.
Dx of septic arthritis based on synovial fluid analysis	Purulent/yellow/green synovial fluid, WBC >50K, PMNs >75%, glucose <25, or culture positive
Dx and Tx of osteomyelitis	Inflammation/infection of bone. Children: hematogenous, monomicrobial; adults: contiguous, mono-/polymicrobial. <i>S. Aureus</i> = most common overall; <i>Salmonella</i> in sickle cell pts. SSx: local tenderness, warmth, erythema, and swelling, systemic sx (fevers); Dx: elevated ESR/CRP (not specific), bone biopsy with bacteria (definitive dx), XR: periosteal elevation or bony erosions, MRI: most sensitive test; Rx: IV abx, surgery consult (debridement)

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Dx and Tx of necrotizing fasciitis	Rapidly progressing infxn of the fascia with necrosis of the SQ tissue. Type 1: polymicrobial (most common), abdomen/perineum, DM2 = risk factor. Type 2: monomicrobial (GAS = 2nd most common), extremities. SSx: severe pain out of proportion to exam, rapid progression, erythema (most common finding), crepitus, necrosis, cellulitis turns dusky blue with bullae/vesicles; XR: subcutaneous emphysema (LRINEC score can aid in dx); Rx: broad spectrum IV abx (including Clinda for antitoxin effects) AND surgical debridement (definitive tx).
What is the most common primary bone cancer, and how is it diagnosed?	Multiple myeloma ("CRAB"): hyper C alcemia, R enal failure, A nemia, B one lesions/ B ack pain. Dx: abnormal SPEP (M-spike) & UPEP (Bence-Jones protein), peripheral smear: rouleaux formation, XR skull: "punched out lesions." NOTE: most bone tumors are metastatic and found in the spine (prostate, breast, kidney, thyroid, skin). Complications: hypogammaglobulinemia (leads to sepsis), hyperviscosity syndrome.
What are clues for diagnosis of osteosarcoma?	Second most common primary bone cancer. Located in metaphysis of long bones (distal femur = most common site, tibia, humerus). Bimodal: teens & >65 (bimodal), associated with radiation for childhood cancer. SSx: persistent bone pain (worse at night). Dx: XR: lytic lesion at metaphysis with "sunburst" pattern, Codman's triangle (elevation of periosteum at periphery of tumor). Labs often not helpful but may show elevated AlkPhos, LDH and ESR
What are clues for diagnosis of Ewing sarcoma?	Painless mass in femur, occurs in adolescence (M>F), XR: "onion peel"
Differentiate crystals in gout vs. pseudogout	<u>Gout</u> : negatively birefringent, needle-like crystals (urate). <u>Pseudogout</u> : positively birefringent, rhomboid crystals (calcium pyrophosphate).
Dx and Tx: Polymyositis	Inflammation of striated muscle. SSx: similar clinical picture to dermatomyositis; proximal limb and neck muscle weakness, but NO rash; strong association with malignancy. Dx: elevated ESR/CRP, CPK, and aldolase. Rx: high dose steroids, methotrexate
Dx and Tx: Dermatomyositis	Inflammation of striated muscle. SSx: similar clinical picture to polymyositis; proximal limb and neck muscle weakness, +heliotrope rash around eyes; strong association with malignancy. Dx: elevated ESR/CRP and CPK. Tx: methotrexate.
Dx and Tx: Polymyalgia rheumatica	Bilateral, symmetric proximal muscle weakness ("cape-like" distribution), stiffness worse in AM, associated with temporal arteritis. Dx: elevated ESR, rheum consultation. Rx: steroids.
Dx and Tx: Reactive arthritis (formerly Reiter Ssyndrome)	Seronegative arthritis occurring after infection; associated with HLA-B27. SSx: urethritis, conjunctivitis, asymmetric large LE joint arthritis with normal-appearing synovial fluid, often occurring after STI (<i>Chlamydia</i>) or GI infection. Tx: NSAIDs, physical therapy.

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Dx and Tx: Rheumatoid arthritis	SSx: polyarticular, symmetric, deforming arthritis, especially affecting hands (Boutonniere & swan neck deformities) but sparing DIP joint; has broad extraarticular involvement. Dx: elevated RF (70-80%, but also present in 5-10% of healthy population) or anti-CCP, elevated ESR/CRP, XR with bony destruction. Tx: NSAIDS, DMARDs, steroids. **Note: Atlantoaxial joint instability: DO NOT hyperextend neck with intubation**
Dx and Tx: Juvenile idiopathic arthritis	<u>Systemic JIA</u> (Still's disease): daily fever, myalgia, polyarthritis, "salmon-pink" rash, anemia, thrombocytosis, Dx: ANA neg, RF neg; Rx: NSAIDS, steroids, methotrexate. <u>Polyarticular JIA</u> : symmetric chronic arthritis, ≥5 joints, mild systemic sx, uveitis (rare); Dx: ANA pos, RF pos (5-10%), elevated ESR ; Rx: NSAIDS, steroids, methotrexate. <u>Pauciarticular JIA</u> : chronic arthritis in 1-4 joints, +uveitis ; Dx: ANA pos , RF neg, ESR normal; Rx with NSAIDS, steroids, methotrexate.
Dx and Tx of osteoarthritis	A polyarticular condition that is worse with activity and better with rest. Middle aged women. Knees are the most effected. Herberden's nodes on the DIP and Bouchard's nodes on the PIP
Dx and Tx: Psoriatic arthritis	Psoriatic skin lesions usually precede joint disease. SSx: symmetric polyarthritis, "sausage digits" (dactylitis), nail pits. Dx: anemia, RF usually neg , ANA+, XR: erosion AND new bone formation, "pencil in cup" deformities. Rx: NSAIDS, DMARDs, NO STEROIDS (causes pustular psoriasis), or antimalarials.
Dx and Tx: Wegener's granulomatosis (granulomatosis with polyangiitis)	Medium vessel vasculitis. Upper & lower respiratory tract involvement + renal failure. SSx: sinusitis, hemoptysis (pulmonary infiltrates), nephritis (hematuria), cutaneous nodules, and palpable purpura. Dx: +c-ANCA; Rx: steroids, DMARDs.
Dx and Tx: Goodpasture's syndrome	Small vessel vasculitis. SSx: cough/dyspnea, hemoptysis from alveolar hem., glomerulonephritis. Dx: anti-basement membrane antibodies. Tx: steroids, DMARDs, plasmapheresis.
Dx and Tx: Churg-Strauss syndrome	Medium vessel vasculitis: vasculitis + eosinophilia + asthma. SSx: bronchospasm, sinusitis, possible cardiac + GI SSx. Dx: peripheral eosinophilia. Tx: steroids, DMARDs.
Dc and Tx: Polyarteritis nodosa	Medium-vessel vasculitis. Multiorgan involvement but SPARES the lungs, Associations: chronic HBV, intra-renal aneurysm. Age 40-60s, M>F. SSx: skin ulcers, nephritis, mesenteric ischemia , lacy rash (livedo reticularis); Dx: ESR/CRP elevation, ANCA+. Rx: high-dose steroids, DMARDs.
Dx and Tx: Systemic lupus erythematosus	Autoimmune chronic inflammatory dz with multiorgan involvement. Pt will be African-American female. Causes: drug-induced ('HIPPS'): H ydralazine, I NH, P rocaïnamide, P henytoin, S ulfonamides. SSx (≥4): classic "butterfly" malar rash, or discoid rash, photosensitivity, oral ulcers, arthritis, renal dz (nephritis), encephalopathy (sz or psychosis), serositis (pericarditis), cytopenia (any cell line); high risk of thrombosis (ACS, PE). Dx: ANA+, anti-dsDNA+, anti-Smith, antiphospholipid ab. Rx: NSAIDS, steroids, antimalarials (hydroxychloroquine), DMARDs.

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Dx and Tx: Scleroderma (systemic sclerosis)	Collagen deposition in skin + other organs. SSx: fatigue, stiff joints, loss of strength, pain, sleep disturbances, CREST syndrome (C alcinosis, R aynaud's syndrome, E sophageal dysmotility, S clerodactyly, T elangiectasias), renal failure (most common cause of death) presenting with HTN. Rx: supportive, rewarm digits, CCBs.
Dx and Tx: Sjögren syndrome	Autoimmune chronic inflammation of salivary & lacrimal glands. SSx: dry eyes and mouth. Dx: Anti-Ro & Anti-La antibodies, RF+, ANA+, Schirmer's test (tests tear production). Rx: lubricants, pilocarpine, DMARDs.
Dx of rhabdomyolysis	Acute necrosis of skeletal muscle fibers & leakage of cellular contents into the circulation. Causes: trauma, heat, alcohol/drugs, exercise. SSx: myalgias, stiffness, weakness, malaise and low-grade fever. Dx: CK >5x upper limit normal, UA (+blood, but NO RBCs), electrolyte abnormalities (hypocalcemia = most common, hyperK, hyperPhos), EKG: ALL patients
What is the appropriate treatment for rhabdomyolysis?	IVF with UOP 3cc/kg/hr, ± bicarb gtt to alkalinize urine (controversial & may not be helpful); goal is to prevent renal failure (causes ATN), relative endpoint CK <1000 (although the initial CK level does not correlate with the likelihood of renal failure).
Dx: Rheum disease + higher risk with intubation	Rheumatoid arthritis with atlantoaxial joint instability (don't hyperextend with intubation)
Dx: non-caseating granulomas to multiple organs	Sarcoidosis (commonly eyes and chest, skin lesions)
What are characteristic lab abnormalities with sarcoidosis?	Elevated ACE levels, decreased PTH, hypercalcemia/hypercalciuria
For arthrocentesis, why is the extensor surface most commonly used?	Extensor surfaces avoid neurovascular structures that typically overlie the flexor aspect of joints
WBC needed for septic bursitis?	> 10,000 (less than septic joint which is 50,000)

Hematology & Oncology	
Bizz	Buzz
Transfusion + Fever + Otherwise well	Febrile non-hemolytic transfusion reaction. Most common reaction. Rx: tylenol, hold for 30min, likely restart
Transfusion + Urticaria + Otherwise well	Simple Allergic (Urticarial) Reaction. Rx: benadryl (premedicate in future) but don't need to stop transfusion
Transfusion + Shock + AKI	Acute Hemolytic Transfusion Reaction. Often 2/2 ABO incompatibility. SSx: fever, flank pain, shock; Labs: +Coombs; Rx: stop transfusion, IVF, diuretics, treat hyperK; Alternate Dx SEPSIS
Transfusion + Shock + Angioedema + Normal CXR	Severe Allergic Reaction (Anaphylactic). Associated with hereditary IgA deficiency. Rx: stop transfusion, epinephrine, supportive care
Transfusion + Pulmonary Edema without other signs of heart failure	Transfusion Related Acute Lung Injury (TRALI). ARDS after transfusion. SSx: HIGH fever, hypoxemia, hypotension. CXR: pulmonary infiltrates. Rx: stop transfusion, supportive, NO furosemide. Most common cause of death following blood transfusion.
Transfusion + Pulmonary Edema WITH other signs of heart failure	Transfusion-Associated Circulatory Overload (TACO). Presentation similar to TRALI BUT....HTN, + sx of overload (e.g JVD, peripheral edema, high bnp), NO Fever; Rx: stop transfusion, supportive care, furosemide OK
What patients are higher risk for developing TRALI?	Those with existing systemic inflammation (e.g. sepsis, trauma); linked to platelet and FFP transfusions
What is the most common infection transmitted by blood transfusion?	Hepatitis B
What is the underlying pathology in Hemophilia A and B	Bleeding disorder due to lack of Factor 8 (A; 85%) or Factor 9 (B); both X-linked recessive and clinically indistinguishable; Dx: factor activity levels, normal PT BUT abnormal PTT
What are the common clinical features of Hemophilia A&B?	Minor trauma causing large amounts of bleeding or hemarthrosis (hallmark sign); <u>Children</u> : ankle (most common joint); <u>Adults</u> : knee (most common) > elbow & ankle. CNS bleeding = leading cause of death in hemophilia. In CNS bleeding, factor replacement should precede diagnostic imaging.
What is the appropriate dosage of factor replacement for a pt with Hemophilia A and Minor, Moderate and Severe Bleeding?	# Factor VIII units = wt (kg) x (desired % increase in factor activity) x 0.5. <u>Minor</u> (hemarthrosis): 20-30% factor desired (10-15U/kg of Factor VIII); <u>Moderate</u> (epistaxis, GI bleed): 50% factor desired (25U/kg of Factor VIII); <u>Severe</u> (CNS, RBP): 100% factor required (50U/kg of Factor VIII)
What is the appropriate dosage of factor replacement for a pt with Hemophilia B and Minor, Moderate and Severe Bleeding?	# Factor units = wt (kg) x (desired % increase in factor activity). <u>Minor</u> (hemarthrosis): 20-30% factor required (25U/kg of Factor IX); <u>Moderate</u> (epistaxis, GI bleed): 50% factor required (50U/kg of Factor IX); <u>Severe</u> (CNS, RBP): 100% factor required (100U/kg of Factor IX)

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What are alternative treatments if Factor is not available for bleeding hemophiliac patient?	FFP (1cc FFP = 1U F8); Cryo (1 bag = 100U F8), DDAVP: 0.3 mcg/kg IV/SQ, 150 vs 300mcg nasally, increases F8 activity & vWF (carries F8); PCC
What does Von Willebrand Factor (vWF) do during hemostasis	<u>1° hemostasis</u> : attaches subendothelium to platelets (platelet aggregation); <u>2° hemostasis</u> : protects F8 from degradation + delivers FV8 to site of injury (Factor VIII carrier protein)
Dx and Tx of Von Willebrand's Disease	Most common inherited bleeding disorder. SSx: easy bruising, skin bleeding, prolonged bleeding from mucosal surfaces (mouth, GI/GU); Labs: platelet count normal , normal PT/INR, prolonged bleeding time; Rx: DDAVP (first-line Rx, increases release of vWF), non-recombinant F8, Cryo NOT recommended (risk of viral transmission), no FFP (very little F8); ± Antifibrinolytics (Amicar, Tranexamic acid) which inhibit clot breakdown
Dx and Tx of Polycythemia Vera	Clonal proliferation of RBCs/increased RBC mass. SSx: pruritis (aquagenic, plethora (facial), HTN, engorged retinal veins, thrombosis, erythromelalgia (burning of hands/feet), splenomegaly; Labs: all cell lines inc (esp. RBC). Rx serial phlebotomy, hydroxyurea, ASA
How does heparin work and how can it be reversed?	Activates antithrombin III (inactivates F10 & thrombin), monitored with PTT; Reversal: Protamine Sulfate 1mg per 100U heparin, give slowly to avoid anaphylactoid reaction
How does LMWH work and how can it be reversed?	Activates antithrombin III (inactivates ONLY F10), monitored with Xa level; 60% reversal with Protamine Sulfate (dose based on timing since last LMWH injection)
How does coumadin work and how can it be reversed?	Inhibits vitamin K clotting factors (2, 7, 9, 10, proteins C & S), monitored with INR; Reversal: FFP/VitK (alternate PCC), dosage based on type of bleeding and INR
Review appropriate treatment to reverse coumadin based on severity of bleeding and INR	<u>INR < 5 & NO bleeding</u> : lower or skip 1 dose; <u>INR ≥5 but ≤10 & NO bleeding</u> : skip next 1-2 doses, alt: skip 1 dose + Vit K 2.5-5mg PO; <u>INR ≥10 & NO serious bleeding</u> : hold med until INR is therapeutic + Vit K 5mg PO; <u>ANY serious bleeding regardless of INR</u> : hold med + Vit K 10mg IV + FFP or PCC; <u>Life Threatening bleeding</u> : hold med + Vit K 10mg IV + FFP or PCC or F7a
How does tPA work and how can it be reversed?	Converts plasminogen to plasmin to breakdown clots; Nothing specific reversal agent. Can give large amount of everything (pRBCs, cryo, FFP, platelets, PCC, amicar, transexamic acid)
How does clopidogrel work and how can it be reversed?	Blocks glycoprotein 2b/3a & prevents platelet activation (crosslinking with fibrin); nothing specifically reverses, can give platelets
How does Dabigatran (Pradaxa) work and how can it be reversed?	Direct thrombin inhibitor, associated with GIB; Reversal agent: Idarucizumab, PCC/pRBCs/platelets, can also do HD
How does Rivaraxaban (Xarelto) work and how can it be reversed?	Factor 10a inhibitor; no specific reversal, NOT dialyzable, can try thrombin activation with PCC, FFP, cryo

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Elderly with chronic back pain, lytic lesions on XR	Multiple myeloma ("CRAB"): hyperCalcemia, Renal failure, Anemia, Bone lesions/Back pain. Dx: abnormal SPEP (M-spike) & UPEP (Bence-Jones protein), peripheral smear: rouleaux formation, XR skull: "punched out lesions." Complications: hypogammaglobulinemia (leads to sepsis), hyperviscosity syndrome.
What symptoms suggest aggressive Lymphoma?	"B symptoms": fever, night sweats, lymphadenopathy
What distinguishes Non-Hodgkins from Hodgkins Lymphoma?	<u>NHL</u> : more common, more widespread, less curable, leading cause of non-solid organ cancer-related death; <u>HL</u> : less common, related to viral infection; often presents with B symptoms and local spread, high cure rates
What are the two most common types of Non-Hodgkins Lymphoma and what distinguishes them?	<u>Follicular Lymphoma</u> : indolent, slow growing, widespread at dx, no cure; <u>Diffuse Large B cell Lymphoma</u> : aggressive and symptomatic, rapid spread, 50% cured
What are the two types of Burkitt's (non-Hodgkin's) lymphoma?	Associated with EBV. <u>Endemic (African) Burkitt lymphoma (eBL)</u> : most common; jaw and facial bone including the orbit (> 50%); <u>Sporadic Burkitt lymphoma (sBL)</u> : less common; abdominal tumors with bone marrow involvement
Dx and Tx of Hodgkins lymphoma	Bimodal age (teens/young adults, older adults). SSx: nontender cervical LAD, mediastinal mass on CXR, B symptoms; Labs: Reed-Stenberg cell ("owls eye"); Rx: chemo, radiation
What is the difference between Acute and Chronic Leukemia?	<u>Acute</u> : rapid increase in blasts, most common in children; <u>Chronic</u> : mature abnormal WBCs, slow growing, most common in elderly
What is the difference between Lymphocytic and Myelogenous Leukemia?	<u>Lymphocytic</u> : B & T cells; <u>Myelogenous</u> : RBCs, platelets & other WBCs
What is the difference between the presentation of ALL vs AML?	BOTH: bony pain, big liver/spleen, anemia, bleeding, thrombocytopenia, infection and blasts in blood; <u>ALL</u> : most common childhood leukemia, +LAD; <u>AML</u> : more common in adults, no LAD, + gingival infiltration, Auer rods on blood smear
What is the difference between CLL and CML?	BOTH: slow onset, elevated WBCs; <u>CLL</u> : most common adult leukemia, smudge cell, worst prognosis; <u>CML</u> : mostly adults, Philadelphia chromosome, high platelets, good prognosis
Dx and Tx of neutropenic fever	1 oral temp $\geq 38.3^{\circ}\text{C}$ or $\geq 38^{\circ}\text{C}$ for ≥ 1 hour + ANC < 500. Leading cause of cancer death: infection. Obtain cultures (gram pos. most common). Rx: admission, empiric ABX
Dx and Tx of Hyperviscosity syndrome	\uparrow serum viscosity that causes sludging & vascular stasis. Cause: Leukemias (AML or CML in blast crisis, WBC > 100k), multiple myeloma Waldenstrom macroglobulinemia (most common cause), polycythemia vera. SSx: mucosal bleeding (epistaxis), CNS sx (blurred vision, headache, AMS, stroke), end-organ ischemia; Labs: Rouleaux formation; Rx: phlebotomy (polycythemia) + IVF, plasmapheresis (high proteins), leukapheresis for blast transformations (induction chemotherapy = definitive tx)
Leukostasis syndrome	Pretty specific to AML or CML in blast crisis, WBC >100k.

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Dx and Tx of Tumor Lysis Syndrome	Massive cytolysis + release of the intracellular contents, can occur with aggressive heme malignancies, large solid tumors/steroids after start of tx. Metabolic derangements: HIGH uric acid, phosphate, potassium & LOW calcium. Tx: aggressive IVF, correct lytes (hyperUA: Allopurinol, Rasburicase; hyperPhos: aluminum hydroxide, Renagel, HD; hyperK: calcium, insulin/glucose, biarb, kayexalate, HD; hypoCa: 2/2 high phos, treat phos first, only treat if symptomatic). Complications: cardiac arrhythmias, renal failure
Name the criteria for emergent HD in tumor lysis syndrome	K ≥ 6, uric acid ≥ 10, Cr ≥ 10, phosphorus ≥ 10, volume overload, symptomatic hypoCa
Thrombocytopenia, otherwise normal labs, well patient	Idiopathic Thrombocytopenic Purpura (ITP), results from rapid destruction of plts (fxn is normal). Types: <u>children</u> (2-6yo): acute, post-infectious; adults (20-50). SSx: petechiae (most common), pupura, gingival bleeding, epistaxis, menorrhagia; Dx: thrombocytopenia; Rx: observation (only if asymptomatic + plt <50k), supportive (kids), steroids (plt<50k) & IVIG, platelets (only for severe bleeding, VERY low plt), others: splenectomy (refractory cases)
Thrombocytopenia, Hemolytic Anemia, Neuro symptoms	Thrombotic Thrombocytopenic Purpura (TTP): enzyme defect leads to unstable platelet plugs & hemolytic anemia. SSx (PENTAD): F ever, A nemia (MAHA, schistocytes; HIGH indirect bili, LDH, retic count; LOW haptoglobin), T hrombocytopenia (10-50k), R enal failure, N euro sx. Dx: decreased ADAMTS-13 activity, schistocytes, normal: PT/INR, fibrinogen, dimer; Rx: supportive, plasmapheresis (Rx of choice); others: plasma exchange transfusion, steroids, DMARDs, IVIG, splenectomy; DO NOT GIVE PLATELETS
What types of patients are at higher risk for developing TTP?	African-American females, Lupus, HIV, drugs (Clopidogrel, Quinine)
Kid with thrombocytopenia, hemolytic anemia, renal failure	Hemolytic Uremic Syndrome (HUS)- often after diarrheal illness (O157:H7- shiga-like toxin), labs with eo hemolysis (schistocytes, high unconj bili, high LDH), supportive care, transfuse prbcs Hb <6, DO NOT GIVE PLATELETS OR ANTIBIOTICS
What defines Heparin Induced Thrombocytopenia (HIT) and what is the treatment?	Antibodies that inactivate platelets usually at 5d if naive and only min/hours if prior exposure. Dx (4 T's): T hrombocytopenia (platelets <150K or >50% drop after starting heparin [less often LMWH]), T ime of onset (5-10d), T HROMBOSIS (thrombosis, skin reactions, PE, CVA, MI), no o ther cause. Labs: +HIT antibody. Rx: STOP heparin or LMWH (dont cont while waiting for tests), can change to DTI (Argatroban, Dabigatran), NO platelets, change to direct thrombin inhibitor (DTI)
Dx and Tx of Disseminated Intravascular Coagulation (DIC)	Microvascular <u>thrombosis</u> AND <u>consumptive coagulopathy</u> causing multi organ failure. Related to underlying severe illness (sepsis = most common cause) & massive inflammation (trauma, pregnancy complications, cancers); Labs: LOW: platelets (most common) & fibrinogen; HIGH: PT/INR, fibrinogen degradation, dimer; Rx underlying cause, Rx (primarily bleeding): FFP, plts, RBCs; Rx (primarily thrombosis): heparin, LMWH

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In what thrombocytopenic disorders are platelets contraindicated?	TTP, HIT, HUS
What are the 3 main causes of microangiopathic hemolytic anemia?	TTP, HUS, DIC
Diseases with thrombocytopenia	TTP, HUS, DIC, SLE
What are classic causes of microcytic and macrocytic anemias?	<u>Microcytic</u> (MCV < 80): iron deficiency, thalassemia, anemia of chronic disease; <u>Macrocytic</u> (MCV > 100): B12 or Folate deficiency
Anemia + low retic, low ferritin, low iron, high TIBC	Iron Deficiency Anemia
Anemia + high retic, nl/high ferritin, nl/high iron + target cells (smear)	Thalassemia- defective Hb chains (A- Africa, B- India)
Anemia + Headache, abdominal pain, basophilic stippling (smear)	Chronic Lead Poisoning, may also see Burton's line (blue line on gums)
Anemia + low retic, low iron, normal ferritin, normal TIBC	Anemia of Chronic Disease: microcytic or normocytic
Anemia + Hypersegmented neutrophils + Neurologic changes	B12 deficiency, hypersegmented neutrophils (on periperal smear)
What patients are at higher risk for B12 deficiency?	Crohns, on PPI, vegan diet
Anemia + Hypersegmented neutrophils + NO neurologic changes	Folate Deficiency (also consider in alcoholicswith anemia)
What pateints are at higher risk for Folate deficiency?	alcoholics, tea and toast elderly
What are the most common causes of pancytopenia?	Malignancy (leukemias), nutritional deficiency (B12 or folate deficiency), infection, toxin exposure, aplastic anemia (complication of hepatitis)
Most common initial presentation of sickle cell in infants?	Acute Dactylitis: pain and swelling of hands and feet 2/2 vasoocclusive crisis, 2/2 infarction NOT infection; Rx: supportive
Treatment of Sickle Cell pt with Priapism?	Low-flow (venous/ischemic) causes erect penis with soft glans; Rx: aspirate corpus, intra-cavernous phenylephrine, surgical drainage pm
Treatment of Sickle Cell pt with Stroke?	Emergent exchange transfusion
Dx and Tx of Acute Chest Syndrome	Sickle cell pt with fever, SOB and infiltrate on CXR, HIGH mortality (most common cause of death in sickle cell pts). Causes: infection, VOC, fat embolism; Rx: ICU admit, supportive (incentive spirometer, IVF, O2, pain control) antibiotics for CAP, pRBCs vs. exchange transfusion (severe crises marked by PaO2 < 60 mm Hg, not first line)

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Kid with sickle cell + non-traumatic rapid drop in Hb	Aplastic Crisis. SSx: pallor, weakness/lethargy, shock; arthralgias, arthritis (adults). Dx: Hgb drop by at least 2 points from their baseline, LOW retic <2%; associated with Parvovirus B19. Rx: pRBCs, IVIG
Kid with sickle cell, abdominal pain + rapid drop in Hb	Splenic Sequestration: rapid sequestration of RBCs in the spleen causing splenomegaly and severe anemia. SSx: pallor, splenomegaly. Dx: low Hgb, high retic. Rx: IVF, transfuse pm, splenectomy
What infections are more common in sickle cell patients?	Encapsulated organisms: <i>S. pneumoniae</i> , <i>H. influenzae</i> , <i>N. meningitides</i>
African American +HIV + anemia after starting on dapsone	G6PD deficiency. X-linked recessive. Most common disease-producing enzymopathy in humans. Found in African, Asian, and Mediterranean ancestry. Oxidative stress causes hemolytic anemia. Protective against malaria. Dx: neg. Coombs, Heinz bodies on smear. Rx:
What are potential G6PD triggers?	Fava beans, Infections, Meds: dapsone, TMP-SMX , phenazopyridine, nitrofurantoin, antimalarials, rasburicase, and methylene blue
Old person, gradual face swelling, periorbital edema, cough, and cyanosis. Hx of smoking.	Think superior vena cava syndrome. order: CT chest w/ contrast.
Thresholds for platelet transfusions in adults	Trauma or active bleeding, ITP: less than 50,000k / coagulation disorder: 20,000k / everyone else: 5,000-10,000k

Endocrine & Metabolic	
Bizz	Buzz
Review expected bicarb and pCO2 levels for Metabolic Acidosis, Metabolic Alkalosis, Respiratory Acidosis, Respiratory Alkalosis	M.Acid: ↓ HCO ₃ , ↓ pCO ₂ (hypervent); M.Alk: ↑ HCO ₃ , ↑ pCO ₂ (hypovent); R.Acid: ↑ pCO ₂ , ↑ HCO ₃ (↑ renal reabsorption); R.Alk: ↓ pCO ₂ , ↓ HCO ₃ (↓ renal reabsorption). Normal values: pH 7.4 / HCO ₃ 24 / pCO ₂ 40 / AG 12 ±
What is the appropriate metabolic compensation for Respiratory Acidosis and Alkalosis?	(Delayed metabolic compensation) <u>A</u> cid/ <u>A</u> lk: '1325': Acute/Chronic; R.Acid: for every ↑ of pCO ₂ by 10, HCO ₃ should ↑ by 1 (Acute) and 3 (Chronic); R.Alk: for every ↓ in pCO ₂ by 10, HCO ₃ should ↓ by 2 (Acute) and 5 (Chronic); If NOT true then a mixed disorder is present
What is the appropriate respiratory compensation for Metabolic Acidosis and Alkalosis	(Immediate respiratory compensation) Acid: Rule of 15s; Alk: 007; <u>M.Acid</u> - 1.5 x HCO ₃ + 15 (±2) = PCO ₂ & last two digits of pH (±2); <u>M.Alk</u> : for every ↑ of HCO ₃ by 1, pCO ₂ should ↑ by 0.7; If NOT true then a mixed disorder is present
What is the differential for an anion-gap metabolic acidosis?	A CAT MUDPILE: <u>A</u> spirin, <u>C</u> arbon monoxide, <u>C</u> yanide, <u>C</u> affeine, <u>A</u> cetaminophen, <u>T</u> heophylline, <u>M</u> ethanol, <u>M</u> etformin, <u>U</u> remia, <u>D</u> KA (AKA), <u>P</u> ropylene Glycol, <u>I</u> soniazid, <u>I</u> buprofen, <u>I</u> ron, <u>L</u> actic Acidosis, <u>E</u> thylene glycol
What are the most common causes of non-anion-gap metabolic acidosis?	Diarrhea & spironolactone
What are the most common causes of metabolic alkalosis?	Vomiting, diuretics
What is the primary difference between Type I and Type II diabetes?	<u>I</u> : insulin deficiency (auto-immune), <u>II</u> : insulin resistance
What are the criteria for diagnosis of diabetes?	Fasting blood sugar >126 (2 separate occasions), Random glucose >200 with sxs of DM, Glucose >200 after OGTT, HbA1c > 6.5%
Suspected DKA and coffee-ground emesis	Erosive esophagitis & hemorrhagic gastritis in up to 9% of DKA, rarely need treatment/endoscopy
What are the keys to diagnosis of DKA?	SSx: Polyuria, polydipsia, abd pain, vomiting, acetone (fruity) smell, ± unstable vitals/shock, AMS, possible coffee-ground emesis; Labs: glucose > 250, pH < 7.3 (VBG ok), HCO ₃ <18, AG >10, +Serum/urine ketones; Note: workup should include eval for cause of DKA (infection rule out) and EKG due to lyte abnormalities
What lab value is critical to know prior to giving insulin for DKA?	Serum potassium: patients have an overall deficiency of K (initial labs may show high K), if initial low K and pt given insulin they will become too hypokalemia and code
What is the appropriate approach to fluid resuscitation in DKA?	Rx: 2L NS IVF bolus (kids: 10-20cc/kg), when glucose < 250, start glucose-containing fluids (D51/2 NS)

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What is the appropriate approach to electrolyte repletion in DKA?	Key = <u>POTASSIUM!</u> Know level before insulin. <u>K<3.3</u> : HOLD Insulin & give K, <u>K 3.3-5.2</u> : can start insulin but give K in each liter of IVF; <u>K>5.2</u> : Start Insulin & NS, no supplemental K needed; <u>BICARB</u> : controversial, only Rx if severe DKA + intubated; <u>MAG</u> : replete with K; <u>SODIUM</u> : falsely low, abnormal Na will typically correct with fluids.
What is the appropriate approach to insulin administration in DKA?	Eval K level first, then give 0.1 U/kg/hr gtt (double if glucose not down by 50 after first hour); initial bolus not necessary; can also follow SQ regimen; **Transition to regular insulin SQ when gap closed and pH >7.3, dose 5U per 50 over 150 (max 20U), stop insulin gtt 1hr after pt given SQ insulin
How do you correct sodium for hyperglycemia (pseudo hyponatremia)?	Add 1.6 to Na for each glucose value of 100 over 100 mg/dL
Treatment for DKA followed by new AMS or seizure	Cerebral edema, more common in kids and new onset type I, Rx: mannitol (1-2g/kg)
What is the mechanism of action and possible adverse effect of Sulfonylureas (Glipizide, glyburide)	Stimulates insulin release from the pancreas, can cause prolonged hypoglycemia in overdose
What is the mechanism of action and possible adverse effect of Biguanides (Metformin)	Suppress hepatic gluconeogenesis (NO hypoglycemia), ± GI upset, lactic acidosis
What is the mechanism of action and possible adverse effect of Thiazolidenediones (TZDs- Actos, Avandia)	Increase sensitivity to insulin (muscle & fat); ± hepatitis & edema; NO hypoglycemia
What distinguishes Hyperosmolar Coma from DKA?	Occurs in Type II DM. SSx: more likely to have CNS ssx (stupor/coma). Labs: glucose > 600, pH >7.3, HCO ₃ >18, ± urine ketones, AG <12, severe dehydration (8-12L deficit). Rx: IVF, insulin
What lab test can help identify factitious hypoglycemia (exogenous administration)?	C-peptide, value will be low 2/2 suppression of endogenous insulin
What is the rule to calculate MIVF rate?	4cc/kg for first 10kg, 2cc/kg for second 10kg, 1cc for each additional kg to max of 120cc/hr total
What are typical causes and appropriate treatment for Hypervolemic, Euvolemic and Hypovolemic Hyponatremia?	<u>HyperV</u> : CHF, ESRD, cirrhosis; Rx: water restrict + diuretics; <u>EuV</u> : SIADH, psychogenic polydipsia; Rx: water restrict; <u>HypoV</u> : vomiting, diarrhea, third spacing, diuretics; Rx: NS vs 1/2 NS
What is the approach to correction of hyponatremia?	Overall goal correction rate 0.5 mEq/hr or 10-20 mEq/day. **Note: rapid correction risks central pontine myelinolysis/demyelination.** Rx: asymptomatic & Na >120 = no emergent treatment; Na <120 & +neuro ssx, give 3% NaCl (100cc over 10m, additional 100cc over 50m)
How and when does central pontine myelinolysis present?	how: AMS (lethargy and coma) w/ spastic quadriplegia after treatment for hyponatremia. When: 1-6 days after treatment for hyponatremia. Pts with chronic hyponatremia (e.g. alcoholics) are most at risk for this.

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What is the approach to treatment of hypernatremia?	Overall goal correction rate 1-2 mEq/hr. **Note: rapid correction risks cerebral edema. ** . Calculate Free Water Deficit $(0.6 \times \text{wt(kg)} \times (\text{Na}/140 - 1))$ and replace with NS until euvolemic, then D5W vs D5 1/2 NS. Give 50% over 12h, remainder over the next 24hr
What is the most common cause of hyperkalemia?	Lab error, resend lab
What EKG changes are seen in hyperkalemia?	Peaked TW, PR long, loss of P wave, wide QRS, sine wave, VT/VF
What is the general approach to treatment of hyperkalemia?	<u>Cardioprotection</u> (Calcium Gluconate or Chloride ONLY if egg changes, <u>K shifters:</u> Insulin/glucose, bicarb if acidotic, albuterol), <u>K excretion:</u> lasix, kayexylate, HD
Dx and Tx of hypokalemia?	Most common electrolyte disturbance. 2/2 ↑ losses (GI loss or diuretics), intracellular shift. SSx: cramps, weakness, arrhythmias, cardiac arrhythmias, rhabdo; Rx: K repletion 100 mEq K for every 0.3 below normal, give 10-20 mEq/hr; **Note: supplement with Magnesium (↑ absorption)
What is the most specific EKG change associated with hypokalemia?	Flattened or inverted T wave, U waves = specific, prolonged QT, STD
Dx and Tx of Hypercalcemia	Ca > 10.5. Causes: hyperparathyroid (overall most common cause), malignancy (most common inpatient cause); SSx: BONES (bone pain), STONES (renal, biliary), GROANS (abd pain, n/v), THRONES (polyuria) and PSYCHIATRIC OVERTONES (depression, anxiety, insomnia); EKG: short QT; Rx: immediate if Ca > 14 (12-14 per ssx) with IVF (first step), Calcitonin (↑ excretion, inhibits osteoclasts), Bisphosphonates (inhibits osteoclasts, requires days to work), Steroids (↓ GI absorption), Lasix (if volume overload)
Dx and Tx of Hypocalcemia	Ca < 8.5. Causes: 2/2 hypoparathyroid (thyroidectomy = most comon cause), Vit D deficiency, high phos, low or high Mg; SSx: paresthesias, tetany, Chvostek's sign, Trousseau's sign, seizure. EKG: QT prolongation; Rx: IV calcium (if <7.5 and severe ssx), give Vit D and Mg pm
Dx and Tx of Hypermagnesemia	SSx: weakness, loss of reflexes, dysrhythmias, respiratory depression; Rx: calcium gluconate
Alcoholic with AMS, ataxia, nystagmus	Wernicke's Encephalopathy 2/2 Thiamine (B1) deficiency; Rx: Thiamine 500 mg IV, improvement in hours
Alcoholic with short term memory loss	Korsakoff's Psychosis 2/2 Thiamine deficiency, irreversible
Poor nutrition and high output cardiac failure (dyspnea, peripheral edema)	"Wet" Beriberi 2/2 chronic thiamine deficiency; Rx: Thiamine 100mg IV
Diarrhea, Dermatitis, Dementia	Pellagra 2/2 Niacin (B3) deficiency
Crohn's patient with macrocytic anemia and paresthesias	Cobalamin (B12) deficiency; high risk include Crohns, vegans, alcoholics, PPIs, pernicious anemia (antibody to intrinsic factor); causes megaloblastic anemia + neuro deficits, hypersegmented neutrophils

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Alcoholic with macrocytic anemia	Folic Acid deficiency. High risk: alcoholics, elderly, phenytoin; no neuro changes. Hypersegmented neutrophils seen as well.
Child with poor diet and bowed legs	Rickets 2/2 Vitamin D deficiency (Calcium absorption); Osteomalacia: adult equivalent, normal height
Bad skin, bleeding gums and poor wound healing	Scurvy caused by Vitamin C deficiency (collagen formation)
What vitamins are toxic in overdose?	Fat soluble ADEK; A : bear liver consumption, skin changes, pseudotumor cerebri, D : hypercalcemia, hypercalciuria, E : ↑ bleeding, K : hemolytic anemia, jaundice in newborns
What hormones are secreted from the pituitary?	GOAT FLAP: G rowth Hormone, O xytocin, A DH, T SH, F SH, L H, A CTH, P rolactin; ALL but Oxy/ADH are from anterior pituitary
What are potential causes of hypopituitarism?	Mass lesions, bleeds (pituitary apoplexy), hypothalamic disease, Sheehan's syndrome; Dx: check hormone levels
Low cortisol but normal aldosterone	ACTH deficiency, causes 2° adrenal insufficiency
Inability to lactate post-partum	Sheehan's syndrome causing prolactin deficiency (and panhypopit)
Visual field deficits, headache, hormonal abnormalities	Pituitary adenoma (macro if >1cm), Rx: transsphenoidal surgery
What is the treatment for prolactinoma?	Most common pituitary tumor. Rx: Bromocriptine (does not require surgery)
Dx and Tx of Cushing's syndrome	Cortisol excess. ACTH secreting pituitary adenoma or exogenous steroids (most common cause): SSx ('CUSHING'): C entral obesity, U rinary cortisol ↑, S triae, H TN/ H yperglycemia/ H irsutism, I atrogenic, N eoplasms, G lucose intolerance. Rx: surgery (tumor)
Headache and tunnel vision in oversized person	Growth Hormone secreting pituitary adenoma; Labs: ↑ GH & IGF1. Children: gigantism, adults: acromegaly; Rx: surgery
What hormones are produced by the adrenal glands?	Medulla : epinephrine & norepinephrine; Cortex : cortisol, androgens, aldosterone
Identify the key differences between primary and secondary adrenal insufficiency	Deficiency of adrenal gland hormone production. 1° (adrenal disease): ↑ CRH & ACTH , Addison's dz (autoimmune) = most common cause, rapid withdrawal of steroids = most common cause in US, SSx: shock, hypoglycemia, ↓ mineralocorticoid = ↓ Na/gluc, ↑ K, HYP ERpigmentation (buccal, 2/2 ↑ ACTH); 2° (pituitary dz): ↑ CRH, ↓ ACTH: ↓ Na/gluc, normal K, NO hyperpigmentation; Rx: IVF, glucocorticoids, vasopressors
Dx and Tx of adrenal crisis	Life threatening exacerbation of adrenal insufficiency. Hallmark = shock that is refractory to IVF / vasopressors. Cause: acute stressor (infxn, trauma, MI); SSx: shock, ↓ Na/gluc, ↑ K; Rx: glucocorticoids (Hydrocortisone v Dexamethasone)
Young child with mass in abdomen and HTN	Neuroblastoma (adrenal medulla tumor)
HTN, headache, palpitations, elevated catecholamines	Pheochromocytoma (adrenal medulla tumor)

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Review the hormone cascade and general function of thyroid hormones	Thyroid Releasing Hormone (hypothalamus) → Thyroid Stimulating Hormone (Ant Pituitary) → T4 (inactive from thyroid gland) → converted to T3 (active form) in peripheral tissues, requires iodine for conversion; T3 functions in glucose absorption, muscle building, increases catecholamines, increases basal metabolic rate
What are common causes of hyperthyroidism	Graves (most common cause, young person), Toxic nodular goiter (elderly), iodine-induced (amiodarone), Thyroiditis (amiodarone)
Review common SSx and Dx of hyperthyroid	SSx: heat intolerance, palpitations, wt loss, tachycardia, anxiety, hyperreflexia, goiter, exophthalmos, pretibial edema; Labs: ↓ TSH level, ↑ T4/T3
Difference between thyrotoxicosis and thyroid storm	Thyrotoxicosis: any condition that results in excessive thyroid hormone concentration. Thyroid storm: life threatening decompensation of thyrotoxicosis (hyperthyroid + acute event)
What is the appropriate treatment for Thyroid storm?	1) Beta-blockers (Propranolol): ↓ sympathetic activity + blocks peripheral conversion of T4 → T3. 2) Antihormone Rx: PTU (if P regnant) or Methimazole (blocks new hormone synthesis). 3) Potassium Iodine (AFTER above, blocks release of preformed hormone). 4) Steroids (blocks peripheral conversion of T4 → T3. 5) Treat precipitant & prevent decompensation (IVF, tylenol, cool pm) // The order is controversial. Some sources say give PTU before beta-blockers, but just know that PTU comes BEFORE iodine. That's what they typically test.
What are common causes of hypothyroid	Hashimoto's (MCC in US, autoimmune), meds, postpartum, Iodine deficiency (MCC worldwide)
Review common symptoms, diagnosis and treatment of hypothyroid	SSx: fatigue, weight gain, cold intolerance, brittle hair and nails, constipation, periorbital edema, slow reflexes, edema; Labs: TSH (↑ with 1°, ↓ with 2°), ↓ free T3 / T4; Rx: synthroid.
Hypothyroidism + AMS	Myxedema coma. Rx: steroids (for concurrent adrenal crisis), IV Levothyroxine (T4)
Dx and Tx of Thyroid Ca	5% thyroid nodules are cancerous, common CA overall but low mortality; Dx: FNA; Rx: thyroidectomy, radioactive iodine-131, thyroid supplementation
Dx and Tx of Hyperparathyroidism	↑ PTH → ↑ Ca, ↓ Phos; SSx of hypercalcemia (see above); Rx: lower Ca with IVF (first line), calcitonin, bisphosphonates, steroids, surgery
Dx and Tx of Hypoparathyroidism	↓ PTH → ↓ Ca, ↑ Phos; may be 2/2 thyroid surgery; SSx of hypoCa; Rx: replacement of Ca, Vit D
Pt w/ hx of anorexia, presents with signs of heart failure after starting an outpatient refeeding program.	Refeeding syndrome. Happens when refeeding begins before correcting electrolyte abnormalities. Leads to profound hypophosphatemia, hypokalemia, hypomagnesium, and ultimately volume overload and CHF. Tx: stop refeeding, correct electrolyte abnormalities.

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Dermatology	
Bizz	Buzz
Dx and Tx of Erythema Multiforme	Hallmark = TARGET lesions, SYMMETRIC on palms & soles (± trunk, face), minimal to no mucosal involvement, -Nikolsky; Rx: remove trigger, supportive
What is the most common cause of Erythema Multiforme?	Infections: HSV (most common viral cause) > Mycoplasma (most common bacterial cause)
What drugs are most commonly associated with Erythema Multiforme?	SOAPS: S ulfa, O ral hypoglycemics, A nticonvulsants, P enicillin, N SAIDS
What is the difference between Stevens Johnson Syndrome and Toxic Epidermal Necrolysis?	BOTH: mucosal involvement, drugs = most common cause, flu-like prodrome, painful target lesions, +Nikolsky's; SJS : <10% TBSA, most common in children; TEN : >30% TBSA, more common in elderly, fluid / lyte problems common; Rx (both): supportive, remove trigger
What distinguishes Staph Scalded Skin Syndrome (SSSS) from SJS/TEN?	SSSS : NO mucosal involvement, younger children/infants/newborns, caused by infection (Staph exotoxin) & treated with antibiotics (Nafcillin/Dicloxacillin), NO STEROIDS; BOTH: painful rash, bullae, + Nikolsky
Dx and Tx of Necrotizing Fasciitis?	Type 1 : polymicrobial (most common), abdomen/perineum, DM2 = risk factor. Type 2 : monomicrobial (GAS), extremities. SSx: severe pain out of proportion to exam, rapid progression, erythema (most common finding), crepitus, necrosis, cellulitis turns dusky blue with bullae/vesicles, dirty dishwater discharge, La Belle Indifference (pt unconcerned); XR: SQ emphysema; Rx: broad spectrum IV abx (Clinda halts toxin production) AND surgical debridement (definitive tx)
Dx and Tx of Urticaria	Transient pruritic edematous plaques, red border with central clearing, NOT symmetric; Rx: remove trigger, benadryl/steroids/epi pm
Dx and Tx of Rocky Mountain Spotted Fever?	<i>Rickettsia rickettsii</i> . Transmission: wood tick (must be attached for 6 hours to transmit, eastern US (Carolinas, Oklahoma). SSx: fever (MC sx), centripetal (wrists/ankles → trunk) maculopapular rash (palms + soles), calf tenderness. Labs: low platelets, hypoNa. Rx: Doxycycline
College kid with petechiae → purpura presents in shock	Meningococemia; seen in college kids, military barracks (close quarters), caused by <i>N. meningitidis</i> (requires airborne precautions); Rx: ceftriaxone, supportive, Note: tx high-risk contacts with Rifampin, CTX or Cipro
What is the difference between Pemphigus Vulgaris and Bullous Pemphigoid?	Pemphigus : S uperficial, flaccid bullae → break easily & crust, +mucosal involvement, +Nikolsky; Associations: Myasthenia, thymoma; Rx: steroids; Pemphigoid : D eeper, elderly, pruritic papules → tense bullae, NO mucosa, -Nikolsky, Tx: steroids, tetracycline or dapsone

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Shock + Erythroderma and possible foreign body	Toxic Shock Syndrome. Bacteria that produce toxins. <u>Staph</u> (TSS): more common; erythematous rash w/ desquamation + hypotension + high fever ≥ 3 organ systems, assoc. w/ foreign body; <u>Strep</u> (STSS): fever, but less rash often with existing wound, not associated with foreign bodies. Rx: remove foreign bodies FIRST, supportive care, and antibiotics (clinda first to reduce protein production, then empiric broad-spectrum for sepsis coverage), IVIG for refractory cases
Gunmetal gray pustules on palms	Disseminated Gonococemia (arthritis-dermatitis syndrome). SSx: fever + migratory arthritis + rash (papules \rightarrow pustules with gray necrotic or hemorrhagic center); Dx: genital + throat culture; Rx: ceftriaxone. Complications: tenosynovitis, septic arthritis
Dx and Tx of Impetigo	Most often in kids, facial vesicles rupture and become "honey-crusted", + contagious, Staph more common cause than strep, Tx topical mupirocin (if small area) vs systemic keflex (more extensive or bullous)
What is the characteristic rash and cause of Erysipelas?	Well demarcated, slightly raised, beefy red plaque. Group A Strep = most common cause
Obese woman with red macular rash under breasts, noted satellite lesions	Candida; also associated with immunocompromised state; Rx: PO nystatin for thrush, Topical azoles for rashes, dry skin care
What is the difference between Candida and Tinea rashes?	<u>Candida</u> : seen in babies, immunocompromised, DM, fat adults (intertriginous), rash: red + macular with characteristic satellite lesions, Rx PO nystatin for thrush, Topical azoles for rashes, dry skin care; <u>Tinea</u> : sharply marginated, annular lesion with raised or vesicular margins with central clearing, Rx: topical azoles for everything except scalp and nails (griseofulvin)
What are the names for Tinea infections in the following areas: groin, foot, scalp, nail	Groin: Crura (jock itch), Foot: Pedis, Scalp: Capitis, Nail: Unguium
Compare the rashes of HSV and HPV	HSV: vesicular clusters with painful erosions (T1- mouth, T2- genitals, Rx: acyclovir); HPV: cauliflower-like and painless (anogenital warts) = most common STD in US (> Chlamydia)
Vesicle or ulcer noted on tip of nose or ear?	Herpes Zoster (shingles), tip of nose (Hutchinson sign) for herpes ophthalmicus (V1), ear (Ramsay-Hunt) if CN 7/8; Rx: acyclovir, steroids
What is the characteristic rash Molluscum Contagiosum?	Dome-shaped fleshy papule with central umbilication on face/torso/ext; most common in kids in daycare or adults with HIV; caused by MCV (pox virus), Rx: self-limited, cryotherapy
Compare the rashes of Scabies and Pediculosis	<u>Scabies</u> : linear burrows in interdigital web space and intertriginous areas with extreme pruritis; <u>Pediculosis</u> (lice): erythematous macules/wheals, extreme pruritis, nits visible; Rx (BOTH): decontamination, Permethrin cream (often repeat 1wk, esp lice)

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Compare atopic dermatitis and psoriasis	<u>Atopic dermatitis</u> (eczema): usually kids <5, allergy/asthma history, winter months, dry pruritic skin with lichenification (hyperpigmentation/thickening) in flexural areas, <u>Psoriasis</u> : well-demarcated erythematous plaques/papules with silvery white scales in extensor areas, +Auspitz sign (small bleeding points after successive layers of scale have been removed from the surface of psoriatic papules or plaques), Rx (BOTH): emollients, topical steroids
Dx and Tx of Seborrheic Dermatitis	Cradle cap. Occurs in infants. Yellowish, greasy scales on scalp, ± diaper area & axillae; Rx: emollients, topical antifungals, steroids
What is associated with seborrheic dermatitis in adults?	HIV
Dx and Tx of Contact Dermatitis	Discrete, well-defined or demarcated rash (papules/vesicles/bullae) 2/2 direct irritant vs allergic reaction; Tx remove trigger, protect skin, steroids
What is the duration of steroid treatment for poison oak/ivy?	3wks
What are the distinguishing features of Basal Cell vs Squamous Cell Carcinoma?	<u>BCC</u> : pink, pearly papules with telangiectasia in sun-exposed areas, more common; <u>SCC</u> : UV exposure, ulcerated center with firm-raised border; Rx: BOTH referred for biopsy
What characteristics are concerning for melanoma?	ABCDE: <u>A</u> symmetry, <u>B</u> order (irregular), <u>C</u> olor (different shades, not uniform), <u>D</u> iameter (>6 mm), <u>E</u> volution; Rx: excisional biopsy; depth = most important prognostic factor
Purple papule on gums	Kaposi Sarcoma; lesions most commonly oral, also GI and pulm, they are painless and nonpruritic, seen in HIV/AIDS patients, Rx: treat HIV
Blanching strawberry lesion on infants head	Hemangioma; 50% resolve by 5yrs; head > trunk > extremity
What distinguishes a Lipoma from a Sebaceous Cyst?	<u>Lipoma</u> : well-circumscribed, mobile and painless, "Slippage sign" with normal overlying skin; <u>SC</u> : central punctum, cottage cheese discharge, no slippage, may have secondary infection; Rx (both): referral for excision
What defines the stages of decubitus ulcers?	<u>I</u> : nonblanching erythema, intact skin; <u>II</u> : partial thickness, exposed dermis; <u>III</u> : full thickness skin loss, exposed SQ fat; <u>IV</u> : full thickness tissue loss, exposed bone/tendon/muscle
Painful red nodules on shins	Erythema Nodosum; Associated with IBD, malignancy, infection (strep most common), meds (OCPs); Rx: supportive, high dose ASA 650mg q4hrs or NSAIDs. <i>Pts have a prodrome of fever, malaise and arthralgias.</i>
Characteristic rash of Pityriasis?	Herald patch → "Christmas tree" distribution rash to trunk, ± pruritis, Rx: self-limited, antihistamines; Rule out syphilis as cause
What is the difference between the rashes of Pityriasis and Secondary Syphilis?	Syphilis is asymmetric and involves palms and soles
What are the appropriate precautions for patients with Shingles?	If pt is immunocompromised or possible disseminated infection then airborne + contact precautions are required; if pt is immunocompetent with localized zoster then standard precautions can be followed

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What rashes are associated with palmar lesions?	Syphilis (secondary), RMSF, Scabies, Erythema Multiforme
What rashes are associated with + Nikolsky sign?	SJS, TEN, SSSS, Pemphigus Vulgaris
What rashes are associated with vesicles/bullae?	Bullous pemphigoid, Pemphigus Vulgaris, Necrotizing fasciitis, Disseminated Gonorrhea
What rashes are associated with Petechiae/Purpura?	RMSF, Meningococemia, DIC, Endocarditis
What rashes are associated with target lesions?	Lyme disease, Erythema Multiforme, SJS
Dx and Tx of Henoch-Schonlein Purpura (HSP)	Patient will be 4-12-years-old. SSx: recent URI, abdominal pain, arthralgia, and a rash (buttocks +lower extremities; exam: maculopapular rash (palpable purpura), non-pruritic; Most commonly caused by IgA mediated vasculitis; Rx: supportive care. Complications: nephropathy, intussusception

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Procedures & Skills	
Bizz	Buzz
Describe adequate CPR prior to placement of a definitive airway	Minimize interruptions, adequate rate (>100/min), adequate depth (>50mm in adults, 1/3 the anterior-posterior (AP) chest diameter in children), allow full chest recoil (avoid leaning), avoid excessive ventilations (30 compressions: 2 BVM breaths); rhythm check every 2 min
What is the appropriate compression to ventilation ratio in a newborn?	30:2 compression-to-ventilation ratio for single rescuers, 15:2 compression-to-ventilation ratio for 2 person rescuers
What level of ETCO2 indicates adequate chest compressions during resuscitation?	10-20 mmHg; maintaining level >15 is associated with better outcomes; <15 rarely with ROSC; waveform will abruptly increase with ROSC
What are possible reversible causes of cardiac arrest?	Hypoxia, Hydrogen Ion (Acidosis), Hyperkalemia, Hypothermia, Hypovolemia/hemorrhage, Tamponade, Tension Pneumothorax, Thrombosis (ACS or PE), Toxicologic, Trauma
What medications should be considered for VFib/VTach arrest?	Epinephrine 1mg q3m, Amiodarone 300mg x1, Lidocaine 1-1.5mg/kg (repeat 0.5mg/kg), Magnesium 2g IVP (esp if torsades), Calcium chloride 1amp (esp if possible hyperkalemia), Bicarb (esp if prolonged arrest)
Are pads or paddles better for defibrillation?	Pads- better skin contact and safety
What rhythms during cardiac arrest should be defibrillated and at what dose?	Shock VFib or VTach; 360J for monophasic, 150-200J for biphasic
What are general criteria for Therapeutic Hypothermia after cardiac arrest?	VFib/VTach arrest, ROSC < 60min, induction time < 6 hrs from ROSC, Comatose (or GCS < 9), MAP > 80 mmHg. Consider if PEA with ROSC < 30min, no contraindications
What are contraindications to Therapeutic Hypothermia?	DNR order, sepsis, cancer with brain mets, active bleeding, advanced dementia
What is the temp goal with Therapeutic Hypothermia?	33-36°C
Patients with what ASA classes are likely inappropriate for procedural sedation in the ED?	Class III (severe systemic disease) or worse
What volume of pericardial fluid can be identified on bedside US?	15mL
What are general indications for endotracheal intubation?	Failure of ventilation (hypercarbia) or oxygenation (hypoxia), failure of airway maintenance or protection, anticipated clinical course
What factors predict difficulty with BVM ventilation?	Obesity (or pregnancy), Beard, Elderly (>55), Potential airway obstruction, Edentulous
Review the LEMON Rule for predicted difficult intubation	LEMON: L ook externally, E valuate 3-3-2, M allampati Score (Class IV = strongest predictor of difficult intubation), O bstuction, N eck mobility

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Review definitions of Mallampati I-IV	I : full view of uvula and tonsillar pillars, II : full view of uvula, III : partial view of uvula/base, IV : only hard palate visible
What meds may be given as pretreatment prior to intubation and what are the theoretical benefits of each?	Lidocaine : blunt increased ICP, bronchospams; Fentanyl : thought to blunt sympathetic response to intubation; Atropine : often given in kids to prevent reflex bradycardia with intubation
What complication of fentanyl can not be reversed with Narcan?	Chest wall rigidity (rare complication)
For what patient types is Succinylcholine contraindicated?	Known hyperkalemia, denervation injuries (stroke, spinal cord) > 5 days old until 6 months post-injury; Neuromuscular diseases indefinitely (e.g. MS: may not work, AML); Intra-abdominal sepsis > 5 days until resolution, recent cocaine or amphetamine use, cholinergic toxidromes (e.g. organophosphates as it will cause prolonged paralysis).
What are the rules regarding oral intake prior to procedural sedation?	No oral intake > 3hr (may accept small clear liquids). Note: dislocation reduction and other urgent / emergent procedures should not be delayed to allow time for fasting.
What defines Minimal, Moderate, Deep sedation vs General Anesthesia?	Minimal : anxiolysis, no affect on breathing or vitals; Moderate : purposeful response to stimulation, none to minimal effects on breathing or vitals; Deep : purposeful response only to deep pain, likely depressed breathing; General : no response, requires support of breathing and vitals
What are potential side effects of the following agents used for prodedural sedation: Etomidate, Ketamine, Fentanyl/Midazolam?	Etomidate : myoclonus, adrenal suppression, respiratory depression; Ketamine : emergence reaction, laryngospasm, nystagmus, vomiting; Fentanyl/Midazolam : respiratory depression, cardiac depression; Propofol : hypotension, apnea
What is the main contraindication to the use of Ketamine?	Schizophrenia (may increase psychosis)
What are the potential benefits of ultrasound guided central lines?	Increases rate of success on initial attempt, decreases number of attempts but similar complication rate to non-US guided
True or False- Ultrasound guidance can be used for subclavian central line placement	True- it can be used for the supraclavicular approach but not the traditional infraclavicular approach
What is the appropriate depth for placement of right and left internal jugular and subclavian lines?	R IJ: 13cm, R SC: 15cm, L SC: 17cm, L IJ: 15cm; all ± 2 cm
Equipment needed for needle cricothyroidomy	Syringe (3- or 5-ml), ETT (3.0 mm), over-the-needle catheter (14G)

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What is the appropriate intervention for a failed airway in a pediatric patient (<8-10yr)?	Surgical cricothyrotomy is contraindicated in this age group due to small membrane. Perform needle cric with transtracheal ventilation. 3.5mm ETT cap can be attached to the angiocatheter and BVM can be attached to cap for ventilation
What are the steps for performing a emergent cricothyroidomy?	1) Prepare the skin with antiseptic solution; 2) Locate cricothyroid membrane (below thyroid cartilage& above cricoid cartilage); 3) Make a vertical incision in the midline through the skin and SQ tissues; 4) Dilate the cricothyroid membrane; 5) Place a 6.0 cuffed endotracheal tube or tracheostomy tube and inflate
Block to anesthetize ipsilateral forehead and scalp	Supraorbital
Block to anesthetize area between lower eyelid and upper lip	Infraorbital
Block to anesthetize ipsilateral lower lip and chin	Mental
Block to anesthetize ipsilateral maxillary molars	Posterior superior alveolar
Block to anesthetize ipsilateral mandibular teeth, lower lip, chin	Inferior alveolar
What are the relative contraindications to arthrocentesis?	Overlying skin infection, bleeding diathesis, bacteremia
Location of arthrocentesis for the ankle	Medial to the anterior tibial tendon and directed toward the anterior edge of the medial malleolus
Location of arthrocentesis for the elbow	Lateral aspect distal to the lateral epicondyle and directed medially
Location of arthrocentesis for the knee	Midpoint or upper portion of patella and directed beneath the posterior surface of patella into joint
Location of arthrocentesis for the shoulder	Inferior and lateral to the coracoid process and directed posteriorly toward the glenoid rim
Synovial fluid differences between inflammatory and infectious arthritis	<u>Inflammatory</u> : clear to opaque, low viscosity, WBC 2-50k, PMN ≥ 50%, culture neg, tot protein 3-5, LDH high, glucose >25 (lower than serum). <u>Infectious</u> : opaque, variable viscosity, WBC >50k, PMN ≥ 75%, culture often pos, tot protein 3-5, LDH variable, glucose <25 (MUCH lower than serum)
Location for escharotomies	<u>Neck</u> : incise from clavicle to mastoid process. <u>Chest wall</u> : incise along anterior axillary line from 2nd to 12th rib. <u>Extremities</u> : incise on medial and lateral aspects 1 cm proximal to 1 cm distal to burn
Review the basics of local anesthetics	Amides: lidocaine, bupivacaine (2 Is). Esters: tetracaine, benzocaine (1 I), allergenic 2° to (PABA); Benzocaine: cardiotoxicity, methemoglobinemia, Lidocaine: seizures, hypotension; toxicity Rx: lipid emulsion
Greater saphenous vein cutdown location	1-2cm anterior and superior to the medial malleolus
Contraindication for tonometry	Suspected or confirmed globe rupture

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Describe the steps required for an emergent lateral canthotomy & cantholysis	1) Anesthetize the lateral canthus 2) Crush lateral canthus with a straight Kelly clamp x1-2 min 3) Remove clamp + cut the inferior crus of the lateral canthal tendon. **Note: If unsuccessful, cut superior crus of the lateral canthus**
What are the clinical indicators of a successful lateral canthotomy + cantholysis	1) Improved visual acuity; 2) Resolution of a previously detected APD; 3) ↓ in IOP to < 40 mm Hg
Proper lateral neck radiograph positioning	Neck in extension, end-inspiration
Approach to epistaxis management	Apply external pressure to the nasal bridge (often ineffective) → topical vasoconstrictors / anesthetics → packing (anterior packs left in 2-5d; includes ribbon gauze, nasal nponges, tampons & balloon catheters). Consider cautery (will not work on actively bleeding vessels), TXA.
Approach to failed anterior pack	Consider posterior bleed! Same approach as above but...remove anterior pack and replace with posterior pack. Can use commercial device or Foley catheter.
Approach to the avulsed tooth	Handle tooth by the crown (AVOID handling the root). Rinse (DON'T brush or debride) with normal saline. <u>Extraoral time <20 min</u> : gently rinse tooth and replace. <u>Extraoral time >60 min</u> : soak in citric acid/fluoride and consult oral surgeon. Do not reimplant primary teeth (6mo - 6yr).
Best transport medium for avulsed tooth?	Hank's balanced salt solution. Others: milk, saliva, saline
When draining a peritonsillar abscess, what structure is at risk and how can it be avoided?	Internal carotid artery (2.5cm posterolateral to tonsil), jugular vein. Keep the needle as medial as possible, and cut the needle cap to make a needle guard, ensuring a maximum of 1 cm insertion depth.
Maneuvers to diagnose meningitis	B rudzinski's: b end the b rain, K ernig's: extend k nees, jolt accentuation
Name the contraindications for an LP	Increased ICP, bleeding diathesis, cardiopulmonary instability, soft tissue infection at LP site, vertebral trauma (e.g. fractured vertebra)
Name the indications for a CT brain PRIOR to an LP	ALOC, immunocompromised state, focal neuro deficit, increased ICP, h/o CNS lesion, new onset sz in previous wk, suspected SAH
Anatomic landmarks for LP in an adult and infant	<u>Adult needle placement</u> : L2-3 to L5-S1 (spinal cord ends L1-L2 in adults); L3-L4 interspace is at the level of the iliac crest; <u>Infant needle placement</u> : L4-L5 or L5-S1 (spinal cord ends at L3 in infants)
What finding in CSF is pathognomonic for Subarachnoid Hemorrhage?	Xanthochromia (may have yellow tinge), can be found from a few hours post bleed up to 4 weeks
Dx and Tx of postdural puncture headache	Most common complication of LP. SSx: 24-48 hours after the proc, bilateral frontal/occipital, worse when upright or with maneuvers that increase ICP or + improves or resolves when supine. Rx: hydration, NSAIDs, caffeine, epidural blood patch (severe sx). Prevention: small caliber / blunt needle, replace stylet, bevel parallel to nerve fibers
What are the indications for a perimortem C-Section?	>24wks, loss of vitals in ED, no worse outcome for mother, should be done within 5min

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Needle locaton for paracentesis	RLQ/LLQ entry: 4-5 cm superior and medial to ASIS; can also go infraumbilical midline 2 cm below the umbilicus. Spinal needle in obese
Indications for thoracentesis	<u>Diagnostic</u> : suspected pleural space infection, new onset pleural effusion. <u>Therapeutic</u> : relieve dyspnea
Needle location for thoracentesis	1-2 IC spaces below effusion, 5-10cm lateral to spine. DO NOT perform below level of 9th rib to avoid intraabdomial injury
What incision should be made for a perimortem C-Section?	Midline vertical from 5cm below xiphoid process to pubic symphysis
What is the appropriate management for an insect in the external auditory canal?	Lidocaine to sedate before extraction, extract with forceps or bulb syringe. With an uncooperative patient (child) consider mineral oil to suffocate bug; must examine canal and TM for injury after removal.
How long does it a take a tracheostomy to mature?	One week. (fyi: it takes a G-tube 2-3 weeks).
List the steps for a bleeding tracheostomy	1) Attempt tamponade with cuff overinflation 2) Secure airway with endotracheal intubation 3) Remove trach tube 4) digital compression of innominate artery.
Describe the proper technique for paronychia drainage	I&D with unilateral longitudinal incision on ulnar aspect of digits II-IV or on radial aspect of digits I & V. **Avoid the pincher surfaces (spares the sensate portion of the finger).**
proper treatment for chronic paronychia	Topical corticosteroids
EKG findings with successful placement of tranvenous catheter	Left bundle branch block with left axis deviation
Proper location for chest tube?	4th or 5th ICS @ anterior or midaxillary line. ~ Level of the nipple or infra scapular line
Complications of chest tube drainage system	<u>Absence of respiratory fluctuation or a ↓ in drainage</u> : indicates system is blocked or that the lung is fully expanded. <u>Air leak</u> : indicates continuous bronchial injury or problem with the mechanics of the chest tube system. To test for air leak, have pt cough and bubbles will form in water seal chamber.
Re-expansion pulmonary edema	Rare complication of chest tube insertion. Pt's at risk: > 30% pneumothorax, pneumothorax present for > 3 days. Tx: supportive. Turn off suction and keep chest tube to water seal only. Tx like you would treat any non-cardiogenic pulmonary edema.
Describe manual detorsion of a testicle	"Open Book" technique. Medial to lateral rotation or "opening of a book" (at least 1.5 turns).
Location for intracavernosal aspiration/injection for priapism tx	Performed at 2 or 10 o'clock position on the proximal shaft at the dorsal surface. This is the location of the corpus cavernosum
Tx of a thrombosed hemorrhoid	Clot excision with elliptical incision, do not do this if it has been present for >48hrs as they will heal spontaneously at this point and excision does not help.

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Grab Bag	
Bizz	Buzz
What is the difference between an Emergency Medical Responder, Emergency Medical Technician, Advanced EMT and Paramedic?	<u>EMR</u> : immediate life-saving care (e.g. hemorrhage control, AED use); <u>EMT</u> : emergency care (e.g. BLS, O2, pt's own meds, patient transport); <u>Advanced EMT</u> : add limited advanced life support (e.g. some meds, airway management, EKGs, IVF); <u>Paramedic</u> : licensed, advanced care (e.g. ACLS meds, advanced airway)
What is the difference between Off-line and On-line medical control for EMS providers?	Physician input and surveillance; <u>Off-line/Indirect</u> : develops protocols and standing orders by situation, training and education, quality review; <u>On-line/Direct</u> : direct orders while in field, direct observations
When can a patient refuse care/transport by EMS?	Any patient who is conscious and competent can refuse care. Must be well documented; if pt is deemed incompetent they can NOT refuse care and should be transported even if against their will/requiring restraint or police
What legal guidelines exist for on-scene physicians?	Must provide proof of identity or medical licensure to provide patient care; can assist with treatment in line with existing EMS protocols without assuming legal responsibility; On-scene MD may officially assume medical control from on-line MD but must accept legal responsibility and transport patient to the hospital
What is the difference between Helicopter (rotary-wing) and Plane (fixed-wing) transport programs?	<u>Rotary</u> : limited by weather, less safe, expensive but access to more locations; <u>Fixed</u> : less limited by weather but more limited by location (near airport)
What defines a medical disaster?	When the needs of a community (due to natural or man-made disasters) overwhelm the ability of the health care system to manage them under normal operating procedures
What defines a Level I, Level II and Level III medical disaster?	<u>I</u> : local resources sufficient; <u>II</u> : requires resources from adjacent communities; <u>III</u> : requires state or federal resources (declared by governor or president)
What is an Incident Command System and what are the general responsibilities of the following parts: Operations, Planning, Logistics, Finance?	Standardized but flexible template for local disaster operations; <u>Operations</u> : field work including search and rescue, treat and transportation, triage; <u>Planning</u> : collects data, communicates and coordinates plans; <u>Logistics</u> : facilities, supplies, equipment, food, people; <u>Finance</u> : manages money, payment
What are the categories of primary triage using the Simple Triage and Rapid Treatment (START) protocol?	Used in mass-casualty events to perform a quick assessment of respiration, perfusion, and mental status (RPM). Color coded system, divide injured people into groups: <u>BLACK</u> : deceased, hopelessly injured (no breathing despite repositioning airway, no resuscitation, palliative care ok); <u>RED</u> : immediate priority - requires immediate treatment/stabilization for survival (abnormal respirations/perfusion/mental status); <u>YELLOW</u> : delayed priority - seriously injured but delayed treatment ok without loss of life or limb (stable respirations/perfusion/mental status); <u>GREEN</u> : minor priority, walking wounded

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What are the only two interventions performed under START triage?	Airway repositioning, hemorrhage control
What is the exception to the typical Primary Triage "BLACK" (hopelessly injured, no resuscitation) rule?	Lightening strikes/electrical injuries; pulseless or apneic patients may be more easily resuscitated with immediate ACLS; "Reverse Triage"
What is the most common problem in any disaster	Communication
What are the key components of the Emergency Medical Treatment and Active Labor Act (EMTALA, part of COBRA)?	Patients presenting to ED by EMS require a medical screening exam to ID and stabilize life-threatening conditions; once ambulance on hospital property the hospital is obligated to eval the patient; if the facility can not provide care to stabilize or treat an identified emergent condition the patient must be transferred to a facility that can (transferred under safe/stable conditions); hospital must declare "diversion" if unable to screen patients (internal disaster); Pts must be identified as stable or unstable by an MD (EMTALA does not apply to stable patients)
What rules exist under EMTALA for transferring a patient from the ED to another facility?	Must transfer patient to facility able to handle unstable condition (if current hospital unable), there must be a documented medical benefit to transfer, patients may request transfer and sign informed consent, transfer must be made with appropriate personnel and equipment
What are federal guidelines defined by the Joint Commission regarding language translation in the ED?	ED must provide language assistance to non-English speaking patients; family members should not be used as interpreters
What is the difference between Expressed, Implied and Informed Consent?	<u>Expressed</u> : verbal or written willingness to be treated, covers "usual" care; <u>Implied</u> : action implies willingness; <u>Informed</u> : pt informed of risks/benefits/ alternatives before given verbal or written consent
What is a potential legal outcome of failure to secure informed consent for an invasive procedure?	Court may find MD guilty of battery (unconsented intentional touching) or false imprisonment (unlawful detention or restraint of an individual's personal liberty or freedom); under these it is not necessary to prove negligence (as typical for malpractice) only intent
How should informed consent be documented in the medical chart?	Signed form in the chart is important but MD documentation of discussion in the note may be of equal or greater use
How is implied consent used in emergent conditions with an unconscious patient?	MD has consent to carry out procedures reasonably required to stabilize the patient's condition until consent can be obtained
How does consent apply to minors or mentally incompetent persons	These patients are unable to provide consent (guardian must obtain consent) but under EMTALA MD can stabilize emergent conditions without guardian consent
For what conditions can minors consent to without guardian permission?	Treatment of STDs, mental health, drug abuse, pregnancy care, possibly pregnancy prevention

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What variables define a "mentally incompetent" patient?	Intoxicated (etoh or drugs), psychotic, confused, disoriented or unconscious
When can parents NOT refuse care for their child?	They can not forbid life saving treatment (this include religious freedom arguments); if parents withhold consent under such circumstances the MD can take temporary protective custody of the child
When can MDs commit patients with mental illness?	If they are deemed a threat to themselves or others
What 4 elements are required in a malpractice suit to prove negligence/liability?	Duty: reasonably competent care, Breach of Duty/Abandonment, Causation: proximate cause, Damages: tangible injury occurred
What takes precedence, a Living Will or decisions made by the Durable Power of Attorney (POA)?	POA decisions override those in a living will (when the patient lacks medical decision making capacity) or those expressed by family members
For what patients are MDs mandatory reporters for abuse?	Children/Minors and Elders (>60) (true in most states)
True or False- Emergency MDs should inform patients about all medical errors	TRUE
What do half of lawsuits in Emergency Medicine involve?	Discharge Instructions
What is the difference between the following types of lab errors: preanalytic, analytic, postanalytic?	<u>Pre</u> : occur during specimen collection and prior to processing; <u>Analytic</u> : processing error; <u>Post</u> : after results complete, incorrect reporting or interpretation; Preanalytic errors = most common
What factors specific to emergency medicine hinder physician wellness?	Shift work, 12hr shifts, night shifts, diversity of practice environment- all contribute to burnout
Which federal agency provides oversight to EMS systems?	The National Highway Safety and Traffic Administration under the Department of Transportation
What is the difference between Sensitivity and Specificity?	<u>Sensitivity</u> : proportion of ppl who test+ among those who have the disease (True Pos / TP + False Neg); <u>Specificity</u> : proportion of healthy patients known not to have the disease, who will test- (True Neg / TN + False Pos)
What type of test (high sensitivity or high specificity) is best for Screening/Ruling Out disease verses Confirming disease?	High sensitivity tests are best for Screening/Ruling Out disease (low False Neg rate); High specificity tests are best for Confirming disease (low False Pos rate)
What is the difference between Positive Predictive Value (PPV) and Negative Predictive Value (NPV)?	PPV: proportion of ppl with +Test who also have +Disease (True Pos / TP + False Pos); NPV- proportion of ppl with -Test who also have - Disease (True Neg / TN + False Neg)
How is Number Needed to Treat defined?	The number of patients in a population that need to be treated in order to make one good outcome

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What are exceptions to confidentiality rules?	Public safety threat, mandated reporting and minors or mentally disabled
Can police require that you disclose medical information about an arrested patient?	No
When is review (confirmation of cause of death) by a Medical Examiner required?	Accidental deaths, death is sudden/unexpected, where law may have been broken (i.e. violence), children
What is the difference between Primary, Secondary and Tertiary Prevention?	<u>Primary</u> : targets at risk and prevents problem, includes vaccines, education, water treatment; <u>Secondary</u> : detect disease early to prevent progression, includes PAP smear, colonoscopy, mammography, etc; <u>Tertiary</u> : limits progress of known disease, includes risk factor modification, strict glucose control for DM, post MI meds
What is necessary to document on a patient to leaves Against Medical Advice?	Patient had decision making capacity (understands the consequences of accepting or refusing treatment) and was educated about the risks of refusing treatment; Note a patient leaving AMA should still be provided appropriate outpatient treatment (antibiotics, etc), discharge instructions, return and follow-up instructions
According to HIPPA, under what scenarios can a patients PHI (protected health information) be shared without explicit consent?	Another medical provider with direct patient care responsibilities, pt's insurance for billing purposes
What components of a chart are required for Level 5 billing?	4 descriptors in HPI, 2 PMH/FHx/SH, 10 ROS, 8 PE
What patients are appropriate for disposition to an ED Observation Unit?	Unclear diagnosis in the ED and require a limited amount of further evaluation (completed within 24hr)
How do you calculate a Positive Likelihood Ratio?	Sensitivity divided by (1 – specificity)
What volume of pericardial fluid can be identified on bedside US?	15mL