

Foundations of Emergency Medicine



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of Emergency Medicine

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Comprehensive Board Review

Third Edition, 2024

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DEDICATION

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ABOUT THIS BOOK

The Foundations Comprehensive Board Review resource is intended to provide a high-yield, systems-based approach to studying for the Emergency Medicine In-Training Exam (ITE) and American Board of Emergency Medicine Written Board Exam. The first version, created in 2016, was developed as a comprehensive reservoir of test relevant information based on a multitude of board review references. Each year, content has been edited by a recent emergency medicine resident while they study for the written board exam. This review is divided by system, with the highest yield (highest % on the test) first and the lower yield content topics towards the end. This is meant to be a low cost resource that learners of emergency medicine can use for independent study. For a more interactive approach, consider following instructions for flashcard review noted below.

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Cardiology

Bizz	Buzz
What underlying pathologic process distinguishes myocardial infarction from angina/unstable angina?	Common pathway: Atherosclerotic plaque rupture → exposed endothelium → clot attaches → reduced blood flow; If cell death occurs → positive troponin and MI; If no cell death occurs → negative troponin and angina/unstable angina.
What is the difference between transmural and nontransmural infarction?	Transmural: usually STEMI, large vessel affected, benefit from thrombolytics/PCI; Non-Transmural: 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/frequency, pain with less exertion, hemodynamic changes with pain; No troponin elevation
Diagnosis: Acute chest pain at night, ECG with STEMI, all symptoms and ECG changes resolve with nitro.	Prinzmetal's Angina - coronary vasospasm; Many do not have CAD. Treat with diltiazem. Smoking is major risk factor.
What are early to late ECG changes with ACS?	Hyperacute T's and Giant R (very early and transient) → ST Elevations with reciprocal ST Depression → T wave inversions → Q waves (1 square wide, 1/3 height QRS)
Diagnosis: Crushing central chest pain with diaphoresis, and nausea. Resolved on arrival. Initial ECG without STEMI criteria but has biphasic T-wave in V2/V3.	Wellens Syndrome: biphasic (type A) or deeply inverted, symmetric (type B) T wave in septal leads; Can have resolution of pain; Signifies early proximal LAD lesion
Treatment for patient with chest pain and ECG concerning for Wellen's Syndrome ?	Cardiology consult and urgent LHC; Signifies early proximal LAD lesion
Chest Pain with STE V1-V4 with STD II, III, aVL. What region is affected and where is the most likely culprit lesion ?	Anterior MI 2/2 LAD occlusion; May affect large territory of LV, septum and conduction system (at risk for high grade blocks, wide complex bradycardias), commonly have shock, possible septal or papillary ruptures
Chest Pain with STE I, aVL, V5, V6 with STD V1. What region is affected and where is the most likely culprit lesion ?	Lateral MI 2/2 LAD vs left circumflex occlusion; May affect LV
Chest Pain with STE II, III, aVF with STD V1-V4. What region is affected and where is the most likely culprit lesion ?	Inferior MI 2/2 occlusion of 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. What region is affected and where is the most likely culprit lesion ?	Right Ventricular MI 2/2 occlusion of proximal RCA ; Associated with Inferior MI; Should get R-sided leads (STE in V4R, V5R)
Chest Pain with STD V1-3 with upright T waves. What region is affected and where is the most likely culprit lesion ?	Posterior MI 2/2 occlusion of PDA (RCA > L circ) Get posterior leads to dx (requires only 0.5 mm elevation for STEMI dx).

What are the J-point elevation criteria for STEMI? How does this differ in leads V2-V3 ?	V2-V3: ≥ 2.5 mm in MEN < 40yrs, ≥ 2 mm in MEN ≥ 40 yrs, or ≥ 1.5 mm in WOMEN; All other leads: STE at the J-point of ≥ 1 mm in two contiguous lead
What distinguishes Type I-Type V MI ?	Type I: MI caused by acute atherothrombotic CAD (plaque rupture); Type II: MI 2/2 mismatch of oxygen supply and demand (sepsis, extreme tachycardia); 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: a) STE >1 mm with concordant (same direction) QRS, b) concordant ST depression >1 mm V1-V3, c) STE >5 mm with discordant (opposite direction) QRS Modified Sgarbossa changes this last rule to discordant STE $>25\%$ preceding S wave.
Diagnosis, Treatment: Chest pain with upsloping ST depressions and tall symmetrical T waves in the precordial leads.	Dx: DeWinter T waves = STEMI equivalent; Tx: Cards consult \rightarrow Cath lab
What is unique about the management of inferior MIs ?	1. Always consider concurrent RV involvement and get right-sided ECG leads . 2. Patient is preload dependent, no nitroglycerin or other preload reducing interventions.
What is unique about the management of 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 (< 24 hours) of MI?	Arrhythmia (most common), shock 2/2 pump failure or valve dysfunction (valve rupture).
What are potential late complications (> 24 hours) of MI?	Thromboembolism, myocardial rupture, valve rupture, CHF, pericarditis, LV aneurysm
Diagnosis, Treatment: Pleuritic chest pain 4 weeks after MI	Dx: Dressler's syndrome: autoimmune pericarditis, typically occurs 2-6 weeks s/p MI. Tx: NSAIDs
What artery typically supplies the SA node and AV node ?	SA node- RCA 60%, LCx 40%; AV node- RCA 90%, LCx 10%; Concern for bradycardias if inferior MI
What is the most common cause of cardiac tamponade after MI ? When will this occur?	Myocardial wall rupture Bimodal distribution: first few days and 1-2 weeks
ECG finding: cardiac tamponade	Electrical alternans
Cause, Treatment: New murmur and shock after MI	Papillary muscle rupture leading to mitral regurgitation ; Tx: reduce afterload and dispo to OR; Same treatment if septal wall rupture
What potential treatments for acute MI 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 the preferred treatment for STEMI ?	Percutaneous coronary intervention (PCI) is preferred for STEMI. Thrombolytics should only be considered if PCI is not available at center within 90 min, or after transfer within 120 min.
What are the AHA recommended " door to balloon " times for STEMI at a PCI center and non-PCI center?	PCI center - 90 minutes ; Non-PCI center must transfer - 120 minutes
What is the AHA recommended time to administration of thrombolytics for STEMI at a non-PCI center if you cannot transfer for PCI within 2 hours?	If cannot transfer for PCI within 120 min from a non-PCI center, give thrombolytics (to those eligible) within 30 minutes of arrival.
What ECG changes are included under indications for thrombolysis ?	STEMI (STE > 2 mm for men, > 1.5 mm for women in V2-3, STE > 1 mm in 2+ other leads), STD V1-3 (posterior MI), old LBBB + Sgarbossa . ACC/AHA Updated in 2023 to include deWinter T waves and posterior MI.
What are absolute contraindications for thrombolysis?	Absolute contraindications: prior brain bleed or mass, ischemic stroke or sig closed head trauma < 3 mo, brain or spine surgery < 2 mo, possible dissection, active bleeding, bleeding disorder, INR > 1.7, platelets < 100k, DOAC use, and HTN > 185/110 (despite IV therapy).
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 ECG changes may occur with reperfusion ?	Accelerated idioventricular rhythm (wide complex, no p waves, rate 40-50's), non-sustained VT, PVCs ; These should be transient, are overall benign and do not require additional treatment
Treatment: ST elevation after cocaine use	First treat with benzodiazepines, aspirin, and nitrates . Use thrombolysis only if ST does not return to baseline after these treatments.
Treatment: HTN after cocaine use	Tx: Benzodiazepines, calcium channel blockers, alpha blockers (e.g., phentolamine)
What medications are contraindicated in cocaine-induced chest pain ?	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
What heart valve and what organism is most common in infective endocarditis ?	General Population: - Left sided > right sided - Most common valves are mitral > aortic > tricuspid > pulmonary - Staph aureus is most common pathogen but viridans strep if s/p tooth extraction; If IV drug use: Tricuspid valve infected by 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 ; While the above are classically tested, fever and new murmur are the most common exam findings.
What are the two major Duke Criteria for infective endocarditis?	1. Positive blood culture 2. Valvular vegetation on echo; All other classical exam findings are minor criteria.
What is the appropriate management and treatment of a patient with suspected infective endocarditis ?	Blood cultures x 3 (different locations), Echo (transesophageal preferred), broad spectrum antibiotics to cover staph/strep/gram negatives (vancomycin + penicillin + gentamicin)
When should a patient receive antibiotic prophylaxis for infective endocarditis prior to a procedure?	<u>High-risk procedures:</u> Dental or invasive respiratory (i.e., bronchoscopy) ; GI/GU procedures don't need abx. <u>High risk patients:</u> Artificial or damaged valves, ANY congenital heart disease history , previous endocarditis diagnosis; Rx: Amoxicillin (dental procedures)
What left sided murmurs are systolic ?	Aortic stenosis and mitral regurgitation
What left sided murmurs are diastolic ?	Aortic regurgitation and mitral stenosis
What valve disease do you consider in patient with syncope + systolic murmur radiating to neck?	Aortic Stenosis ; Syncope is poor prognostic sign, typically causes angina and dyspnea → syncope → heart failure
What is the vasopressor of choice in a patient with cardiogenic shock from critical aortic stenosis ?	Phenylephrine ; Left ventricular afterload is fixed at the level of the stenotic valve. Alpha agonism increases diastolic BP beyond the valve and helps perfuse the ventricle.
Diagnosis: chest pain and new diastolic murmur	Aortic dissection causing aortic insufficiency
Patient presents with a new diastolic murmur , widened pulse pressure , and head bobbing . What valve is most likely affected?	Aortic insufficiency
What valve disease do you think of in pregnant women with sudden cardiovascular collapse during labor?	Mitral Stenosis High output during labor causes LA enlargement, AFib, and arrhythmia.
Treatment: Decompensating patient with diastolic murmur and opening snap	Tx: Cardioversion Suspect mitral stenosis and unstable Afib from atrial dilation.
Diagnosis, Treatment: valve pathology in new MI followed by hypotension and new murmur	Dx: Mitral Regurgitation 2/2 ruptured chordae tendineae/papillary muscle Tx: decrease afterload and cardiac surgery

Causes and signs/symptoms: Right Heart Failure	Causes: 2/2 L-sided failure, lung disease (chronic: COPD, sleep apnea, asthma) → pulmonary hypertension, PE (acute) Symptoms: JVD, peripheral edema, hepatic congestion
Causes and signs/symptoms: Left Heart Failure	Causes: 2/2 ischemia, valvular dysfunction, longstanding HTN Symptoms: 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 to treatment of decompensated heart failure?	Decrease LV preload to improve SV and cardiac output (Starling curve); Reduce preload with nitroglycerin and diuretics (Lasix; caution if diastolic failure), BiPAP (improves ventilation and preload); consider afterload reduction (nitroglycerin); give inotropes for shock
What are the classic causes of high output cardiac failure?	Hyperthyroidism, Beriberi, AV fistula, Paget's disease, severe anemia, pregnancy
What are classic CXR findings with heart failure ?	Enlarged cardiac silhouette, bilateral fluffy infiltrates, Kerley B lines, blunted costovertebral angle (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)
Etiology, Diagnosis, and Treatment: Dilated Cardiomyopathy	Etiology: H/o HTN or ischemia; Dx: cardiomegaly on CXR, low EF on echo; Tx: underlying cause/CHF/dysrhythmias, anticoagulate if mural thrombus, transplant if severe
Etiology, Diagnosis, and Treatment: Restrictive Cardiomyopathy	Etiology: fibrosis, radiation, TB causing stiffness; Dx: normal heart on CXR, poor filling on echo; Tx: underlying cause/CHF/dysrhythmias
Diagnosis and Treatment: Hypertrophic Cardiomyopathy	Dx: severe symptoms/syncope with exercise. septal hypertrophy on echo, ECG 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 and worsening obstruction); ↓ squatting , trendelenburg (↑ LV blood volume and decreased obstruction). Note: this is the same pattern as mitral valve prolapse
What is the typical time frame for developing peripartum cardiomyopathy ?	Third 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 (unless concurrent myocarditis)
ECG changes: Pericarditis	PR depression (most specific), PR segment elevation (aVR), diffuse STE , TW flattening followed by TW inversion
Treatment: Pericarditis	Tx: NSAIDS, ± colchicine (↓ recurrent pericarditis). Pearl: Must get Echo to r/o pericardial effusion
What are the classic clinical clues for diagnosis of Myocarditis ?	Dyspnea (most common symptom), chest pain, viral prodrome , CHF (wet lungs, edema), arrhythmias , *unresolving sinus tachycardia*; usually (+) troponin; Echo usually with global hypokinesis and dilated chambers
ECG changes: Myocarditis	Sinus tachycardia, non-specific STE; can be similar to pericarditis
Treatment: Myocarditis	Supportive care, avoid early NSAIDs or steroids, ICU admit if severe/CHF
Causes: Myocarditis	Idiopathic (most common overall), Parvovirus (most common viral cause), Chagas disease (most common worldwide)
What are the collective signs of JVD, decreased heart sounds, and hypotension called and what does it represent?	Beck's Triad of Pericardial Tamponade
What are the clinical features of hypertensive emergency ?	CNS: dizziness, n/v, confusion, weakness, encephalopathy, ICH, SAH, CVA Eyes: ocular hemorrhage, papilledema, vision loss Heart: ACS, aortic dissection, shock Kidneys: hematuria, proteinuria, acute renal failure
What are the differences between Asymptomatic HTN, HTN Urgency and HTN Emergency?	Asymptomatic HTN: BP >140/90 without apparent symptoms HTN Urgency: BP >180/110 but <i>WITHOUT</i> signs of end organ dysfunction HTN Emergency: BP >180/110 <i>WITH</i> signs of end organ dysfunction
What is appropriate ED management of Asymptomatic HTN, HTN Urgency, and HTN Emergency?	Asymptomatic: no workup needed (Cr = only screening test shown to change management for ITE), no treatment needed, restart home meds (if any), refer to PCP. HTN Urgency: rule out end organ dysfunction (based on sx), gradually lower BP over 1-2 days with PO meds (restart home or HCTZ, BB/CCB). HTN Emergency: 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 ?	Encephalopathy : Nicardipine Aortic Dissection : Beta blockers FIRST (Esmolol or labetalol to reduce rate & shear stress), ± Nitroprusside AFTER rate reduction) Cocaine use : benzos and phentolamine (no beta blockers) Pregnancy : IV Mg, Hydralazine, Labetalol (preeclampsia), nifedipine ACS/CHF : Nitroglycerin
How should HTN be managed for Ischemic and Hemorrhagic Strokes ?	Ischemic Strokes : permissive HTN (up to 220/120) to protect penumbra if no thrombolytics BUT reduce to <185/110 if considering tPA . Hemorrhagic Strokes : 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, seizure), 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 (HOCM, critical aortic stenosis), electrical abnormalities (Brugada, WPW, Prolonged QT, arrhythmogenic right ventricular dysplasia), others (PE, MI). Screen all ECGs for these findings
What minimum workup should be completed on young female patients with syncope ?	Pregnancy test (ruptured ectopic may only present with syncope), ECG
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: Admit patients with CHES , as they are high risk for serious outcomes. CHF , Hct < 30 , ECG that is abnormal, Shortness of Breath , Systolic BP < 90 . Other high risk features: family history of sudden death, syncope with exertion, structural heart disease.
ECG changes : Wolff-Parkinson-White	Slurred upstroke of QRS (delta wave), wide QRS (QRS > 120ms), short PR interval (most common, PR < 120ms)
ECG changes and Treatment : Brugada Syndrome	ECG : Pseudo-RBBB, STE V1-3 (types: coved/downsloping STE followed by TWI, or "saddle-back" STE) Tx : AICD
ECG changes : Long QT	End of T wave > 1/2 R to R interval

ECG changes: Arrhythmogenic Right Ventricular Dysplasia	Epsilon wave (positive notch at end of QRS)
Underlying pathology and Treatment: Arrhythmogenic Right Ventricular Dysplasia	Pathology: 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. Tx: antiarrhythmics, AICD
ECG changes: Hypertrophic Obstructive Cardiomyopathy (HOCM)	LVH, LARGE voltages (tall QRS), deep/narrow Q waves ("dagger-like") in lateral (I, aVL, V5-6) and inferior (II, III, aVF) leads
What are some risk factors for Aortic Dissection ?	Prolonged HTN (#1 most common), 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 chest pain, radiating in direction of propagation (neck/arms vs back/abdomen); chest pain + something else = thoracic dissection; 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 (dissecting carotid). Note: BP can be high, low, or normal.
What is the most common CXR finding in acute aortic dissection ?	Mediastinal widening
Management and Treatment: suspected Aortic Dissection	HR (<60) and SBP (<120) control to decrease shear stress (Esmolol/labetalol followed by Nitroprusside , or Labetalol), control pain, T&C x 10-15 units. 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?	Type A: Ascending Aorta, managed surgically Type B: 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 ?	> 3 cm is pathological, > 5 cm is high risk and requires surgery
Management, Treatment: 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**

Diagnosis: history of repaired AAA with massive GI bleed	Aortoenteric fistula
Diagnosis, Treatment: Acute Arterial Occlusion	Look for medical problems related to thromboemboli (Afib = most common embolic cause, MI, or endocarditis) 6 P's: pain (out of proportion to exam), pallor, pulselessness, poikilothermia, paresthesias, or paralysis Dx: CT angio vs duplex US; emergent vascular surgery consultation. Tx: heparin vs thrombolysis vs embolectomy (vascular surgery consult)
What is Homan's sign and how sensitive is it for DVT?	Homan's sign is pain in the calf on dorsiflexion of ankle while the knee is fully extended. 50% sensitivity for DVT.
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 (3 days within last 4 weeks); <u>Inherited:</u> ATIII deficiency, Protein C/S deficiency, Factor V Leiden; exam- tender vein or distended superficial veins, unilateral calf swelling > 3 cm, unilateral pitting edema
What is the appropriate workup for patients with clinical symptoms and low versus high risk of DVT?	Low risk: D-dimer Moderate-high risk: D-dimer and duplex US (alternative is CT venography); if high risk, may require serial dopplers and whole leg US
Management: isolated calf DVT	Anticoagulation <u>not</u> required unless within 5 cm of popliteal vein (ASA otherwise); repeat ultrasound (in 2-5 days) to rule out propagation. This is only true for low risk pts with transient risk factors (such as recent travel).
What are considered distal veins in the evaluation of a DVT? How are clots in these veins managed ?	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, etc.) distal DVTs can be monitored with repeat US and no anticoagulation.
ECG findings: 1st vs. 2nd (type I and II) vs. 3rd degree AV Block	1st Degree: PR >200 and otherwise normal 2nd Degree: Mobitz Type I (Wenckebach) - increasing PR interval then dropped beat Mobitz Type II - stable long PR, then sudden, non-conducted PR → dropped beat 3rd Degree: P waves entirely dissociated from QRS
What types of heart block typically require pacemaker placement?	2nd degree (Mobitz Type II) and 3rd degree

Treatment: unstable bradycardia	<p>Temporizing measures: Atropine (may help if narrow QRS); Others: Dopamine, Epinephrine, Isoproterenol <u>Unstable:</u> transcutaneous pacing (\pm transvenous pacing) Definitive treatment is a permanent pacemaker</p>
Explain the 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 (this is painful, so start low!)
Explain the steps of transvenous pacing.	Place IJ or SC introducer. introduce catheter into right IJ or left subclavian, advance wire into vein and inflate balloon, advance to ~20 cm, set pacer at 80 bpm and output to 20 mA, advance catheter to RV (will show STEMI pattern on monitor, LBBB pattern on ECG), confirm mechanical capture (pulse or pulse ox correlate with pacer spikes), deflate balloon, secure and decrease output to x1.5 lowest current setting with continued capture.
Treatment: arrhythmia and unstable patient	Electricity. Cardioversion (synchronized) if they have a pulse, Defibrillation (unsynchronized) if no pulse.
What is most common side effect of amiodarone IV ?	Hypotension (due to solvent medication is in). Other side effects to monitor in chronic use: LFTs, thyroid function tests, pulmonary fibrosis, blue-gray discoloration of nose.
Differential, Treatment: Narrow complex tachyarrhythmia WITHOUT P waves	<p>DDx: Atrial fibrillation, SVT, AVNRT, orthodromic AVRT Tx: Adenosine or AV nodal blockers; electricity if unstable!</p>
Differential, Treatment: Wide complex tachyarrhythmia WITHOUT P waves	<p>DDx: VT (Vfib possible but likely unstable), SVT with BBB, Antidromic AVRT (e.g. WPW); Tx: Procainamide or Amiodarone (AVOID AV nodal blockers); electricity if unstable!</p>
Diagnosis: chaotic P waves, irregularly irregular rhythm	Atrial Fibrillation
Diagnosis: "sawtooth" symmetrical P waves, regularly irregular rhythm	Atrial Flutter
Diagnosis, Cause: multiple types of P waves, irregularly irregular rhythm	<p>Dx: Multifocal Atrial Tachycardia Cause: 2/2 pulmonary disease (COPD = most common cause)</p>
Diagnosis, Treatment: regular tachycardia and narrow QRS	<p>Dx: AV nodal reentrant tachycardia (AVNRT) or Orthodromic Atrioventricular Reentrant Tachycardia (AVRT). Tx: Adenosine (first line), consider BB or CCB, electricity if unstable!</p>
Diagnosis, Treatment: regular tachycardia and wide QRS	<p>Dx: Antidromic Atrioventricular Reentrant Tachycardia (AVRT) <u>OR</u> Ventricular Tachycardia Tx: Procainamide vs Amiodarone, consider Mag, electricity if unstable! (Avoid AV nodal blockers in these patients)</p>

What is the difference between Orthodromic and Antidromic Atrioventricular Reentrant Tachycardia (AVRT)?	Reentry circuit with accessory pathway (WPW- Bundle of Kent). Orthodromic travels anterograde down AV node and back up accessory pathway resulting in regular and narrow QRS complex (looks like SVT). Antidromic travels anterograde down accessory pathway and back up AV node (retrograde) resulting in regular and wide QRS complex (looks like VT).
Treatment: tachydysrhythmia and suspected Wolff-Parkinson-White	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 AV nodal blocker
Diagnosis, Treatment: multiple chaotic ventricular foci that are wide and irregular	Dx: Ventricular Fibrillation Tx: defibrillation
What BP measurements define Stage I and Stage II HTN?	Stage I HTN: systolic 140-159 mmHg or diastolic 90-99 mmHg Stage II HTN: systolic >160 mmHg or diastolic >100 mmHg
ECG findings: Ventricular Aneurysm	Persistent STE > 2 weeks 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
Diagnosis, Treatment: "Holiday Heart Syndrome"	Dx: typically atrial arrhythmia (Afib) after excessive alcohol intake Tx: observation (if stable), typically self-resolves within 48 hours
ECG findings: Ventricular Tachycardia	AV dissociation, QRS > 120, HR > 100, fusion beats & capture beats (both help distinguish from SVT with BBB)
What are some common mimics of Ventricular Tachycardia on ECG?	Hyperkalemia, Sodium Channel Blockade (TCA's, Benadryl, etc.), accelerated idioventricular rhythm . Suspect these mimics when the QRS is too wide, HR is < 130, or the rate is variable.
What is the most common cause of Cor Pulmonale ?	Cor Pulmonale = right heart failure 2/2 respiratory disease Most common CHRONIC cause: COPD Most common ACUTE cause: PE Others: pulmonary 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. Use the HAS-BLED score.
Review CHA2DS2 VASc scoring to determine need for anticoagulation with AFib.	CHF (1), HTN (1), Age ≥75 (2), DM (1), Stroke history (2), Female (1). Tx: 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 . Inflate BP cuff and listen for flow in brachial artery with doppler as you deflate. This is the MAP (goal 70-80). No systolic/diastolic measurement.

Diagnosis: Pt with an LVAD and elevated LDH	Pump thrombosis. Thrombosis leads to <u>hemolysis</u> which then leads to elevated LDH. LDH levels are typically > 1000 .
What is the most common site for infection in a LVAD ?	Drive line (Wire connecting external controller and the internal pump), followed by pump pocket
What radiating characteristic of chest pain is most consistent with a cardiac cause ?	Radiation of pain down Right arm > radiating 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 factory rate pacing mode; should be done if the patient is receiving inappropriate shocks . Note all AICDs are also pacemakers (on XR AICDs have a thicker wire in the distal lead)
Definition, presentation, treatment: oversensing in a pacemaker	Definition: pacemaker interprets external noise/interference as native heart beats and does not generate a paced beat when indicated. Presentation: individual with a pacemaker that is bradycardic and symptomatic and withOUT appropriate pacer spikes on the ECG. Tx: place magnet over the pacemaker to switch it back to pacer mode at a factory set rate (60-80).
Management: patient with an AICD with unstable VT	Immediate electrical cardioversion for AICD/pacemaker malfunction
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 of the above except vasopressin
What medications should be used in stable ventricular tachycardia ?	Amiodarone, procainamide, or lidocaine
What medication can cause a bidirectional ventricular tachycardia or slow atrial fibrillation?	Digoxin
At what heart score should a patient be admitted for further risk stratification?	Heart score of 4-6 is moderate risk (12-16.6% risk of major adverse cardiac event). Score > 7 has 50-65% risk of major adverse cardiac event, warranting cardiology consult in addition to admission.
What medication should be administered for patients in asystole or pulseless electrical activity ?	Epinephrine 1mg IV every 3 to 5 minutes (these are not shockable rhythms. No amiodarone)
What class of antiarrhythmics categories has a similar mechanism of action as Tricyclic Antidepressants (TCA)?	Class 1A drugs (quinidine, procainamide, disopyramide) work by inhibiting fast sodium channels similar to TCA (will have prolonged QRS duration, prolonged action potential, lengthening of QT interval).

ECG finding: hypothermia	Bradycardia and J waves or Osborne waves (upward deflection at the terminal portion of the QRS complex).
What physical exam findings associated with aortic stenosis ?	Crescendo-decrescendo systolic murmur that radiates to the carotids , S4 gallop, paradoxically split s2. Murmur decreases with valsalva.
What medication if given to patients with pericarditis has risk of causing recurrence ?	Prednisone (steroids)
Definition, Causes, Signs and Symptoms, and Treatment: Constrictive Pericarditis	Definition: Scarring and loss of elasticity of pericardial sac Causes: idiopathic, infectious, post radiation, post cardiac surgery SSx: fluid overload, diminished CO, Kussmaul sign Tx: pericardiectomy
Signs and Symptoms, Diagnostic Test, Treatment: Atrial Myxoma	SSx: right heart or left heart failure, embolic phenomena, constitutional symptoms Diagnostic test: Echocardiogram Tx: Surgical removal by sternotomy. (Most common primary cardiac tumor!)
Diagnosis, ECG findings, Treatment: complication occurring two weeks after STEMI that increases risk for thrombus formation	Dx: Left ventricular aneurysm ECG findings: persistent ST segment elevation Tx: ACE inhibitors, anticoagulation if mural thrombus present, and aneurysmectomy if refractory to medical therapy

Trauma

Bizz	Buzz
Difference between tension pneumothorax and pericardial tamponade	Tension pneumothorax: tracheal deviation, decreased breath sounds, subQ air Pericardial tamponade: decreased heart sounds BOTH: JVD, hypotension, tachycardia
How to determine GCS	E4 V5 M6 = Max 15 Eyes: 4- Spontaneous, 3- Voice, 2- Pain, 1- None Voice: 5- Normal, 4- Confused, 3- Words, 2- Sounds, 1- None Motor: 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 ACS 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)
What is Cushing's Reflex ?	Cushing's reflex is due to increased ICP and impending herniation Triad: hypertension (widened pulse pressure), bradycardia, irregular respirations
Compare subfalcine, uncal and tonsillar herniation	Subfalcine: most common, frontal lobe under falx, ssx abnormal gait Uncal: temporal lobe under cerebellar tentorium, ssx CN3 palsy (blown pupil, down and out), ipsilateral hemiparesis, coma Tonsillar: rare, brainstem herniation, coma and death
Traumatic injuries CT can commonly miss	Diaphragmatic injury, pancreas injury, basilar skull fracture, hollow viscus injuries
Next step for a patient with high suspicion of diaphragmatic injury and a negative CT ?	OR for diagnostic lap and direct visualization of the diaphragm (gold standard)
Classification of LeFort fractures	Midface fx resulting in detachment of maxilla from skull, all the fractures involve pterygoid plate; Dx with CT LeFort I: palate mobile (fx below nose) LeFort II: palate + nose mobile (inferior orbits) LeFort III: entire midface is mobile (zygoma bone), ± CSF rhinorrhea. LeFort IV: a III that involves the frontal bone
Signs & Symptoms, Diagnosis, Treatment: mandibular fractures	SSx: malocclusion, trismus, lower lip paresthesias; BODY = most common Dx: CT or panorex Tx: manage non-condylar fx as open fx with empiric unasyn, ENT/OMFS consult

Signs & Symptoms, Diagnosis, Treatment: orbital fractures	SSx: diplopia, proptosis, limited EOM, ↓ visual acuity; check for infraorbital paresthesia, inhibited upward gaze (sign of inferior rectus entrapment), globe injury Dx: CT orbit Tx: consult ophtho/ENT, decongestants, abx (Augmentin) for sinus involvement
Which facial bone fx has the lowest rate of infection ?	Zygomatic
Diagnosis, Treatment: Nasal septal hematomas	Dx: dark red mass/hematoma associated with nasal fx/trauma Tx: MUST incise & pack (NO needle) to prevent saddle nose deformity/pressure necrosis. Pack with gauze bilaterally (keeps septum midline). Antibiotics while packing in place
Classification of neck zones	Zone I: sternum/clavicles to cricoid cartilage Zone II: cricoid to angle of mandible, (MC site of injury) Zone III: angle of mandible to base of skull. (think of it like an elevator. Zone I is the ground floor)
Discuss the evaluation of penetrating neck injury and how it is managed	Intubate early, straight to OR if unstable vitals or HARD signs of vascular injury "HARD BRUIT": Hypotension (shock), Arterial bleeding , Rapidly expanding hematoma , Deficit [pulse/neuro], Bruit/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
Diagnosis: Car Accident + neck pain + ipsilateral facial and arm weakness	Carotid artery dissection until proven otherwise. This goes for any new neurologic deficits.
Signs & Symptoms, Diagnosis, Treatment: Traumatic aortic dissection	SSx: high speed deceleration, chest pain/back pain, new murmur, pulse deficits Dx: stable- CXR (look for mediastinal widening but 1/3 are normal, 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; Tx: OR on beta blocker for BP control Most common location: aortic isthmus
Diagnosis, Treatment: Flail chest	Dx: ≥3 adjacent rib fractures at 2 different points; leads to paradoxical chest motion with respirations; often associated with pulmonary contusion Tx: early intubation, ± chest tube
Appropriate imaging to evaluation 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 bronchial injuries 9-11: associated with liver and spleen lacerations 4-9: most common location multiple ribs: associated with underlying lung contusion
Indications for OR thoracotomy 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	Small: O2, repeat CXR Large: chest tube **Pearl: if intubating with ptx, do chest tube first to prevent tension ptx**
Indications for ED thoracotomy	Penetrating trauma: field arrest <15 min with initial vitals, ED arrest, SBP <50 after IVF Blunt trauma: Field arrest <10 min with initial vitals, ED arrest Other: suspected air embolism
General approach to traumatic abdominal injury	OR (penetrating): unstable vitals, peritonitis, evisceration, or transabdominal GSW If none of the above and stable, get CT DO NOT send unstable patient to CT Tx: blood for shock even if initial H/H is normal
Most common injury sites for abdominal GSW, abdominal stab wound, blunt trauma	GSW: small bowel Stab wound: Liver Blunt trauma: spleen > liver
Signs & Symptoms, Diagnosis, Treatment: 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 and Tx: laparoscopy/otomy in OR
Abdominal pain secondary to bike handlebar injury	Duodenal/pancreas hematoma/injury
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?	250 ml
Where is the most common location for intraperitoneal fluid to appear on the eFAST exam?	RUQ/Morrison's pouch
What traumatic injuries would NOT be identified on FAST?	Poor sensitivity for solid-organ injury, hollow viscus injury, and retroperitoneal injury
When is diagnostic peitoneal lavage 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 diagnose retroperitoneal injuries	CT with IV contrast , FAST will be negative

<p>Signs & Symptoms, Diagnosis, Treatment: Scrotal/testicular injuries</p>	<p>SSx: straddle injury, hematuria, scrotal ecchymosis/hematoma, tenderness to palpation Dx: doppler US, CT AP Tx: urology consult</p>
<p>Time limit to reimplant amputated penis</p>	<p>8-12 hr max</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Bladder/urethral Injuries</p>	<p>SSx: pelvic fx, gross hematuria, blood at meatus, urinary retention, high prostate, perineal bruising, females may have vaginal bleeding Dx: NO FOLEY (before imaging), RUG first to evaluate for urethral injury, CT cystogram for bladder injury Tx: urology consult, partial urethral lacs typically treated with Foley, complete urethral lacs require surgery</p>
<p>Interpretation of RUG for possible urethral injuries</p>	<p>Anterior Urethral Injury: distal to UG diaphragm, usually external signs of trauma, RUG: small extravasation with bladder filling Posterior Urethral Injury: proximal to UG diaphragm, usually normal external exam, RUG: large extravasation into pelvis</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Bladder rupture</p>	<p>SSx: pelvic fx, gross hematuria (most common) Dx: retrograde cystogram Tx: Intraperitoneal (contrast posterior to bladder) → OR (non-emergent), Extraperitoneal (flame pattern) → Foley (no OR)</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Renal injuries</p>	<p>SSx: blunt trauma causes 90% renal injuries, gross hematuria (microscopic hematuria is rare for significant injury) Dx: CT with IV contrast Tx: all ureteral injuries go to OR, most blunt renal injuries are nonoperative; Renal injury is rarely in isolation - look for other injuries</p>
<p>Management of severe head trauma</p>	<p>Intubate with GCS ≤ 8; remove c-collar + hold in-line cervical stabilization for intubation; HOB elevated; hyperventilation (temporary measure) → cerebral vasoconstriction → ↓ ICP; Hypertonic saline; consider AEDs; Reverse AC if bleeding</p>
<p>What are the NEXUS Criteria and what are they used for?</p>	<p>Used to clear c-spine; DOES NOT include "cervical PAIN": midline cervical ttp, distracting injury, AMS, intoxication, neuro deficits</p>
<p>How to read C-spine XR?</p>	<p>Lateral: Ant/Post spinal line & spinal laminar lines should be smooth; normal prevertebral space- 6mm at C2 & 22mm at C6; Open mouth: C1 and C2 lateral edges should align, look at odontoid for fx</p>

<p>Identify the unstable C-spine injuries and their associated mechanisms</p>	<p>"Jefferson Bit Off A Hangman's Thumb" Jefferson fx (C1 burst fracture 2/2 axial load) Bilateral facet joint dislocation (2/2 hyperflexion) Odontoid fx (Type I - tip [stable fx]; Type II - neck [most common and unstable]; Type III - body [unstable]) Atlantoaxial dislocation (C1/C2 dislocation) Hangman's fx (bilateral C2 pedicle fracture 2/2 hyperextension) Teardrop fx (anterior & inferior vertebral body fx with interspinous ligament rupture, 2/2 flexion > extension) **Mechanisms are very important for boards**</p>
<p>Identify landmarks for anterior, middle and posterior spinal column (Denis model)</p>	<p>Classification for thoracolumbar fx Anterior column: anterior half of vertebral body Middle column: posterior half of vertebral body Posterior column: posterior to vertebral body > 2 columns = unstable</p>
<p>Most common location of spinal fractures</p>	<p>T11-L2 (50%). **Spinal fractures often occur in multiples**</p>
<p>Describe wedge, burst and chance fractures</p>	<p>Wedge: compression of anterior column Burst: crush with multiple fragments involving anterior & middle columns Chance: fracture through all columns, associated with lap belt injuries</p>
<p>Identify the spinal cord syndromes and their associated neurologic findings: Central Cord, Anterior Cord, Brown-Séquard</p>	<p>Central cord: 2/2 hyperextension, usually elderly person hitting chin, sensory (cape distribution) & motor deficit, UE > LE Anterior cord: 2/2 hyperflexion, bilateral motor paralysis, loss of pain/temp, intact vibration & proprioception, worst prognosis (ischemia/infarction) Brown-Séquard: penetrating trauma to 1/2 spinal cord, ipsilateral loss of motor, vibration & proprioception, contralateral loss of pain/temp, best prognosis</p>
<p>Identify landmarks for dermatomes: C6/7/8, T4, T10, L1, L4/L5/S1, S3-5</p>	<p>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</p>
<p>Clinical Features, Treatment: Neurogenic Shock</p>	<p>Loss of vasomotor & sympathetic tone Classic features: hypotension (vasodilation), bradycardia (unopposed vagal tone), poikilothermia (peripheral vasodilation, "warm shock") Tx: IVF, pressors, Atropine</p>

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
Indication for perimortem C-section	gestation ≥ 24 weeks + loss of maternal vital signs; within 4 minutes of arrest; does not worsen maternal outcome
Describe the steps of perimortem c-section (resuscitative hysterotomy)	Indication: > 24 weeks and less than 4 minutes from time of maternal arrest. Vertical incision from uterine fundus to pubic symphysis. Dissect down to the uterus. 2 cm horizontal incision at base of uterus. Insert fingers into incision, push away fetal parts, and extend incision with scissors. Deliver neonate and clamp cord. Deliver placenta. Pack uterus with gauze. Staple close abdominal incision.
What population is at highest risk for intimate partner violence ?	Pregnant women
Leading non-OB causes of death in pregnant women	1. MVC 2. Intimate partner violence 3. Falls
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°: individual thrown by explosion (crush injuries, blunt trauma) 4°: environmental contamination (burns, inhalation injury, smoke, radiation)
What are 2 common concerning blast-related injuries	TM rupture: most common injury, CXR if (+) to look for blast lung Blast Lung: pulmonary barotrauma, most common cause of death Dx: CXR (patchy opacities in butterfly pattern); Others: delayed intra abdominal injuries, compartment syndrome
Immediate and delayed possible complications with myocardial contusion	Immediate: arrhythmia (sinus tach most common) Delayed: pericardial effusion (most common complication 2 weeks out), MI/CHF, valvular injuries (aortic valve + AR most common), ventricular wall rupture (rare; most common cause of death in non-penetrating cardiac injuries); Sternal fx = most common associated fx. Screen with 1. ECG 2. troponin
What is the most common cause of in-hospital death following near-hanging?	Pulmonary edema (post-obstructive response)

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 → asymmetric forehead
Diagnosis, Treatment: Patient presents 2 days after a femur fracture w/ AMS, hypotension, hypoxia and petechiae	Dx: Fat embolism to the lungs Tx: Supportive care. Intubate early because of the development of ARDS and AMS. High risk for DIC. 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.
Diagnosis: Fat protruding through an eyelid laceration	Globe injury Eyelids do not have fat therefore the presence of fat is concerning for deeper injury.
When should tourniquets be applied prehospital per ACEP practice guidelines?	Tourniquets should be used in the setting of significant extremity hemorrhage if direct pressure is not sufficient or impractical
What injury should you expect in pediatric trauma patient with paralysis on scene that resolves on arrival to ED ? Workup? Management?	SCIWORA - spinal cord injury without radiographic abnormalities Dx: MRI of spine Tx: Spine Immobilization for 12 weeks.
What location of a dog bite is a candidate for primary closure ?	Facial laceration (close approximation is appropriate and does not lead to increased infection rates)
Signs & Symptoms, Treatment: Patella tendon rupture	SSx: inability to extend knee, superior patellar displacement, tenderness inferior to patella Dx: straight leg raise exam, patella alta on XR (high riding patella) Tx: knee immobilizer, crutches, referral to orthopedics outpatient
How do you manage a high pressure injury (paint gun into finger)?	Splinting, IV antibiotics, admission and immediate surgical consultation (most important as needs debridement)
What medication can be used to reverse Dabigatran in a patient with traumatic intracranial hemorrhage?	Idarucizumab (Praxbind)
What laboratory test, if positive, raises suspicion for basilar skull fracture ?	Beta-transferrin , will help differentiate nasal secretions from CSF leak
Management: Avulsed tooth	Handle tooth by the crown (AVOID handling the root). Rinse (DON'T brush or debride) with normal saline. Extraoral time <20 min: gently rinse tooth and replace Extraoral time >60 min: soak in citric acid/fluoride and consult oral surgeon Do not reimplant primary teeth (6mo - 6yr)
What is the best transport medium for avulsed tooth ?	Hank's balanced salt solution. Others: milk > saliva > saline

<p>What are the general guidelines for administering tetanus vaccination for wounds?</p>	<p>Fully vaccinated: <u>Clean wound</u> → vax only if last dose >10 years ago; <u>Dirty wound</u> → vax only if last dose > 5 years ago Un/Incompletely vaccinated: <u>Clean wound</u> → vax; <u>Dirty wound</u> → vax and TIG</p>
<p>Diagnosis: Trauma patient receiving massive transfusion has tetanic muscle contractions and prolonged QTc</p>	<p>Hypocalcemia related to citrate in pRBC</p>
<p>What is the most appropriate transfusion strategy for patients with severe hemorrhage on the boards?</p>	<p>1:1:1; pRBC:FFP:platelets</p>

Procedures & Skills

Bizz	Buzz
Describe adequate CPR prior to placement of a definitive airway	Minimize interruptions, adequate rate (100-120/min), adequate depth (5cm 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 ?	Single rescuers: 30:2 compression-to-ventilation ratio 2 person rescuers: 15:2 compression-to-ventilation ratio
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 ?	H&Ts : 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 IV push (esp if torsades), Calcium chloride 1 amp (esp if possible hyperkalemia), Bicarbonate (esp if prolonged arrest)
Are pads or paddles better for defibrillation ?	Pads because better skin contact and safety
What rhythms during cardiac arrest should be defibrillated and at what dose?	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, doesn't follow commands), 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 temperature 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), facial hair, 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 bstruction, N eck mobility

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
Medications for pretreatment prior to intubation and the theoretical benefits of each	Lidocaine: blunt increased ICP, bronchospasm Fentanyl: thought to blunt sympathetic response to intubation Atropine: previously given in kids to prevent reflex bradycardia with intubation (only recommended in specific situations now, per PALS)
What complication of fentanyl cannot be reversed with Narcan?	Chest wall rigidity or " Wooden Chest Syndrome " (rare complication)
Succinylcholine contraindicated for which patients	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. 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 > 3 hr (may accept small clear liquids). Note: dislocation reduction and other urgent/emergent procedures should not be delayed to allow time for fasting.
Define: Minimal, Moderate, Deep sedation, 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 repetitive or forceful stimulus, likely depressed breathing General: no response, requires support of breathing and vitals
Side Effects for 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
Contraindication to the use of Ketamine	Schizophrenia (may increase psychosis)
Benefits of ultrasound guided central lines	Increases rate of success on initial attempt, decreases number of attempt. However, 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 SC 14cm, R IJ 15cm L SC 17cm, L IJ 18cm All +/- 2cm **Remember that SC is shorter distance than IJ and R is shorter than L**
Equipment for needle cricothyroidotomy	Syringe (3 or 5 ml), ETT (3.0 mm), over-the-needle catheter (14G), BVM

What is the appropriate intervention for a failed airway in a pediatric patient (< 8-10 year old)?	Surgical cricothyrotomy is contraindicated in this age group due to small membrane. Perform needle cric with transtracheal ventilation. 3.5 mm ETT cap can be attached to the angiocatheter and BVM can be attached to cap for ventilation
Steps for emergent cricothyroidotomy	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 tracheostomy tube and inflate OR bougie > 6.0 ETT
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 (lower risk), bacteremia
Location of arthrocentesis: ankle	Medial to the anterior tibial tendon and directed toward the anterior edge of the medial malleolus
Location of arthrocentesis: elbow	Directed medially in the groove between the lateral epicondyle, radial head, and olecranon
Location of arthrocentesis: knee	Midpoint or upper portion of patella and directed beneath the posterior surface of patella into joint
Location of arthrocentesis: shoulder	Inferior and lateral to the coracoid process and directed posteriorly toward the glenoid rim
Synovial fluid differences between inflammatory and infectious arthritis	Inflammatory: clear to opaque, low viscosity, WBC 2-50k, PMN ≥ 50%, negative culture, total protein 3-5, LDH high, glucose >25 (lower than serum) Infectious: opaque, variable viscosity, WBC >50k , PMN ≥ 75%, positive culture often, total protein 3-5, LDH variable, glucose <25 (MUCH lower than serum)
Location for escharotomies	Neck: incise from clavicle to mastoid process. Chest wall: incise along anterior axillary line from 2nd to 12th rib. Extremities: incise on medial and lateral aspects <u>1 cm proximal to 1 cm distal to burn</u>

Differences between amides and ester local anesthetics	Amides: lidocaine, bupivacaine (2 Is). Esters: tetracaine, benzocaine (1 I), allergenic 2° to (PABA). Benzocaine: cardiotoxicity, methemoglobinemia Lidocaine: seizures, hypotension Toxicity treatment: lipid emulsion
Describe the location of a lower leg saphenous vein cutdown	1-2 cm anterior and superior to the medial malleolus. Horizontal incision through the outer skin layers followed by blunt dissection and identification of the vein then cannulation of the vein.
Contraindication for tonometry	Suspected or confirmed globe rupture
Steps for emergent lateral canthotomy & cantholysis	1) Anesthetize the lateral canthus with lidocaine 2) Crush lateral canthus with a straight Kelly clamp x1-2 min 3) Remove clamp and cut the canthus horizontally 4) cut the inferior crus of the lateral canthal tendon and recheck IOP. **Note: If unsuccessful, cut superior crus of the lateral canthus**
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 Both make the space smaller
Management to epistaxis	Apply external pressure to the nasal bridge while leaning forward (often ineffective) → topical vasoconstrictors/anesthetics (oxymetazoline) → packing (anterior packs left in for 2-5 days; includes ribbon gauze, nasal sponges, tampons & balloon catheters). Consider cautery (will not work on actively bleeding vessels, NEVER bilaterally), TXA. If none of these work → IR or ENT
Approach to failed anterior nasal packing for epistaxis	Consider posterior bleed! Remove anterior pack and replace with either double or posterior pack. Can use commercial device or Foley catheter.
Location for intracavernosal aspiration/injection for priapism	Performed at 2 or 10 o'clock position on the proximal shaft at the dorsal surface This is the location of the corpus cavernosum
Treatment: Thrombosed hemorrhoid	Clot excision with elliptical incision **Do not do this if it has been present for >48 hours as they will heal spontaneously at this point and excision does not help**
When draining a peritonsillar abscess , what structure is at risk and how can it be avoided?	Internal carotid artery (2.5 cm posterolateral to tonsil), jugular vein. Keep the needle as medial as possible and cut the needle cap to make a needle guard. Ensure a maximum of 1 cm insertion depth.
What portion of a peritonsillar abscess is drained first ?	First aspiration attempt at the superior pole (where most abscesses are located) before moving to the middle and finally the inferior pole Superior → Middle → Inferior

Maneuvers to diagnose meningitis	Brudzinski's: bend the brain Kernig's: extend knees, jolt accentuation
Contraindications for an lumbar puncture	Increased ICP, bleeding diathesis, cardiopulmonary instability, soft tissue infection at LP site, vertebral trauma (e.g. fractured vertebra)
Indications for a CT brain PRIOR to an LP	Altered mental status, immunocompromised state, focal neuro deficit, increased ICP, h/o CNS lesion, new onset sz in previous week, suspected SAH
Anatomic landmarks for lumbar puncture in an adult and infant	Adult needle placement: L2-L3 to L5-S1 (spinal cord ends L1-L2 in adults); L3-L4 interspace is at the level of the iliac crest Infant needle placement: L4-L5 or L5-S1 (spinal cord ends at L3 in infants)
CSF finding that is pathognomonic for Subarachnoid hemorrhage	Xanthochromia (may have yellow tinge), can be found from a few hours post bleed up to 4 weeks
Signs & Symptoms, Treatment: Postdural puncture headache	Most common complication of LP. SSx: 24-48 hours after the procedure, bilateral frontal/occipital, worse when upright or improves/resolves when supine Tx: hydration, NSAIDs, caffeine, epidural blood patch (severe sx) Prevention: small caliber/blunt needle, replace stylet, bevel parallel to nerve fibers
Indications for a perimortem C-Section	>24wks, loss of vitals in ED, no worse outcome for mother, should be done within 4min of loss of pulses
Needle location for paracentesis	RLQ/LLQ entry: 4-5 cm superior and medial to ASIS Infraumbilical: midline 2 cm below the umbilicus Spinal needle in obese
Indications for thoracentesis	Diagnostic: suspected pleural space infection, new onset pleural effusion Therapeutic: relieve dyspnea
Needle location for thoracentesis	1-2 intercostal spaces below effusion, 5-10 cm lateral to spine DO NOT perform below level of 9th rib to avoid intra-abdominal injury
Steps for resuscitative hysterotomy	Midline vertical from the uterine fundus to pubic symphysis . Cut through skin and then open peritoneum with scissors. Make a small horizontal incision in the wall of the uterus with a scalpel. Lift the uterine wall off of the fetus with your fingers and extend the uterine incision with scissors. Deliver the infant, clamp the cord, and hand off to the neonatal team. Deliver placenta. Pack the uterus with sterile gauze. Close the abdomen with skin stapler.
Steps to remove insect from 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 old does a G-tube need to be for safe replacement in the ED ?	G-tubes take 3 weeks to make a mature tract. Before this, surgery needs to be consulted because of the risk of creating a false tract.
How long does it take a tracheostomy to mature ?	Two weeks
Steps for a bleeding tracheostomy	1) Attempt tamponade with cuff overinflation 2) Secure airway with endotracheal intubation and cuff overinflation 3) Remove trach tube >> digital compression of innominate artery.
Steps 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 (saves the sensate portion of the finger**
Treatment: Chronic paronychia	Topical corticosteroids
Telemetry/ ECG findings with successful placement of transvenous pacer in RV	Left bundle branch block with left axis deviation
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	Absence of respiratory fluctuation or a ↓ in drainage: indicates system is blocked or that the lung is fully expanded Air leak: indicates continuous bronchial injury or problem with the mechanics of the chest tube system. To test for air leak, have patient cough and bubbles will form in water seal chamber.
Signs & Symptoms, Diagnosis, Treatment: Re-expansion pulmonary edema after chest tube	Rare complication of chest tube insertion Patients at risk: > 30% pneumothorax, pneumothorax present for > 3 days SSx: worsening dyspnea, hypoxia Dx: CXR with pulmonary edema Tx: supportive. Turn off suction and keep chest tube to water seal only. Treat 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 - 540 deg).
Describe the RUSH protocol	Rapid ultrasound for shock and hypotension Bedside US exam looks at cardiac function, IVC dynamics, pulmonary congestion, abdominal free fluid, and abdominal aortic aneurysm
How do foreign bodies in the skin appear on US? How sensitive is bedside US for detecting soft tissue foreign bodies ?	Foreign bodies will usually appear as hyperechoic foci with acoustic shadowing extending distally Sensitivity is 90% for identifying foreign bodies such as wood, metal, plastic greater than 4-5 mm in length

Pediatrics

Bizz	Buzz
Etiology of neonatal jaundice within first 24 hours of life	BAD sign - ABO incompatibility, Rh incompatibility, TORCH infections, G6PD deficiency Next step: admit, hydrate, and order Coombs test
Etiology of neonatal jaundice 24 hour - 72 hours	Usually physiologic (if gaining weight, stooling, not anemic, and not direct hyperbili) Check bilirubin nomogram to determine need for phototherapy
Etiology of neonatal jaundice > 72 hours	DDx: Sepsis, breast milk jaundice, breastfeeding jaundice, Gilberts syndrome
What is the difference between breastfeeding and breastmilk jaundice ?	Breastfeeding jaundice: suboptimal supply of breast milk, requires hydration and supplementation Breast Milk jaundice: when the baby's liver is not developed enough to handle breaking down the supply of breast milk from mom
Diagnosis, Treatment: Baby age 1 month with jaundice and direct hyperbili	Dx: Biliary atresia (disease of intra and extrahepatic bile ducts leading to obstructive jaundice, cirrhosis, and death) Typically diagnosed before 2 months Tx: surgery w/ Kasai procedure.
What are the most concerning (and unique) causes of abdominal pain in the following age groups: 0-3 month, 3 month - 2 year, school aged kids	0-3 month: Necrotizing Enterocolitis, Hirschsprung's/Toxic Megacolon, Volvulus, Pyloric Stenosis 3 month - 2 year: Intussusception, Meckel's Diverticulum, Foreign Bodies School age: similar to adults appendicitis, pregnancy, ect.
Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Necrotizing Enterocolitis	Pathophys: Inflammation & necrosis of the bowel wall from translocation of gut bacteria; prematurity is greatest risk factor SSx: bilious emesis, bloody stools, abdominal wall erythema Dx: XR with pneumatosis intestinalis (pathognomonic), portal vein air (poor prognosis) Tx: IVF, broad spectrum antibiotics, NG tube (bowel rest), surgery consult, admit ICU
Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Hirschsprung Disease	Pathophys: Lack of ganglion cells in the rectosigmoid colon → lack of distal bowel motility SSx: Delayed passage of meconium (> 48 hours) → obstruction & bilious emesis (late finding) Complications: enterocolitis/toxic megacolon Dx: rectal suction biopsy (gold standard), contrast enema (shows transition zone) Tx: surgery, admit

<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Midgut Volvulus</p>	<p>Pathophys: 1st month of life; Congenital malrotation → volvulus → midgut ischemia SSx: bilious vomiting (always emergent), abd pain/distention, ± rectal bleeding/hematochezia (gut ischemia) Dx: XR "double bubble" can also be seen in duodenal atresia, upper GI series "corkscrew" sign (definitive) Tx: NGT, surgery consult. Associated conditions: congenital diaphragmatic hernia, congenital heart disease, omphalocele</p>
<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Intussusception</p>	<p>Pathophys: 6 month - 3 year; telescoping of bowel (ileocecal most common); typically has a lead point - Tumor, Meckel's, post-viral, HSP SSx: colicky abd pain w/ lethargy + abd mass (sausage-shape in RUQ; RLQ usually empty) + "currant jelly" stools Dx: XR - obstruction, Dance's sign (pathognomonic); US with "target sign" Tx: OR (sick), air/contrast enema (not sick)</p>
<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Meckel Diverticulum</p>	<p>Pathophys: 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, 2 feet proximal to terminal ileum, 2x more often in males, 2 year old most common Dx: Meckel (technetium-99m) scan Tx: surgical consult</p>
<p>At what anatomical levels do ingested foreign bodies usually get stuck?</p>	<p>Cricopharyngeus C6 (60-80%), GE junction T11 (10-20%), Aortic Arch T4 (5-20%) **Coin most common object swallowed</p>
<p>Describe the different appearance on CXR of a coin in the esophagus versus the trachea</p>	<p>If in the esophagus: coin will appear flat on the AP view If in the trachea: coin will appear flat on the lateral view</p>
<p>What are indications for emergent endoscopy for ingested foreign body?</p>	<p>High-grade obstruction, object in esophagus > 24 hours, object > 6 cm, sharp objects, multiple objects swallowed, button battery in esophagus, button battery in stomach > 48 hours or if symptomatic (earlier)</p>
<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Pyloric Stenosis</p>	<p>Pathophys: 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"; Exam: palpable "olive-shaped" mass Dx: Labs: hypoCl, hypoK, metabolic alkalosis (2/2 vomiting), dehydration; US (target sign), upper GI series "string sign" Tx: fluids and correction of electrolytes FIRST, then surgery</p>

What is the most likely location of traumatic C-spine injury in young children?	Age < 8 more susceptible to upper cervical spine injuries (C1-3) given the proportional size of the head relative to the rest of the body
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.
Common causes and presentations 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: 3.5 kg 1yr: 10 kg 5yr: 20 kg 10yr: 40 kg
How do you determine ETT size, depth, and blade size in young children?	Term Newborn: 3.5 ETT Otherwise ETT = Age/4 + 4 (minus 0.5 cm for cuffed) Depth = 3x tube size Blade = 1 for newborn up to 2 yr; 2 for 2-12 yr; 3 > 12 yr
ETT size for premature neonates or small neonates?	< 1 kg: 2.5 uncuffed 1-2 kg: 3.0 uncuffed
What are the general cutoffs for abnormal vitals in a newborn/infant?	Patient is SICK if SBP < 60, RR > 60, HR > 180
What is the equation to determine the lower limit of normal for a child's SBP ?	Normal SBP = (Age x 2) + 70
Signs & Symptoms, Treatment: Breath Holding Spells	Dx: 6 mo-6 yr; associated with pain/emotion, ± turn blue then pass out, but child returns to normal after this and is otherwise well Tx: reassurance. Rule out Fe deficiency anemia, otherwise patient will grow out of it
What characterizes Tic/Movement Disorders ? What are some associated red flags ?	More common in males, suppressible but involuntary movements/actions/verbalizations 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 are initial bolus doses for blood and IVF ?	pRBC: 10cc/kg Crystalloid: 20cc/kg

<p>Characteristics of the following viral exanthems: Measles/Rubeola, Rubella, Erythema Infectiosum/5th Disease, Varicella, Roseola, Hand/Foot/Mouth, Herpangina</p>	<p>Measles (Rubeola): Cough/Coryza/Conjunctivitis (3 C's), Koplik's spots, rash: maculopapular, head → feet; complications: AOM (most common), encephalitis Rubella: "3d Measles"; suboccipital/posterior auricular lymph nodes, petechiae on hard palate, rash: maculopapular: face → trunk</p> <p>Erythema Infectiosum (Fifth Disease): Parvovirus B19, URI sx (3-5 days) → rash: "slapped cheek" with circumoral pallor; aplastic crisis in sickle cell dz.</p> <p>Varicella: vesicles (dew drop on rose petal) in crops at different stages, spares palms/soles. Treat with Acyclovir for immunosuppressed/age > 12, encephalitis/pneumonitis</p> <p>Roseola: HIGH fever → rash (blanching maculopapular), HHV-6, associated with febrile sz, mimics sepsis/meningitis</p> <p>Hand/Foot/Mouth: Coxsackievirus, URI prodrome, vesiculopapular lesions (hands/feet), anterior mouth ulcers (most common: tongue/buccal mucosa)</p> <p>Herpangina: painful ulcers and vesicles in the posterior oropharynx + fever</p>
<p>Characterize, Treatment for tinea infections based upon location</p>	<p>T. capitis: head T. corporis: body T. cruris: groin T. pedis: foot T. unguium: nail (aka onychomycosis)</p> <p>Tx: topical antifungals UNLESS in hair (PO griseofulvin or terbinafine)</p>
<p>Diagnosis, Treatment: Kerion</p>	<p>Inflammatory head/hair fungal lesion on scalp Tx: PO griseofulvin or terbinafine Complication: scarring alopecia</p>
<p>Distinguish, Diagnose, and Treatment: staph/strep infections including Impetigo, Bullous Impetigo, Staph Scalded Skin Syndrome</p>	<p>Impetigo: age < 6, honey-crusted lesions on face, pruritic NOT painful; Tx: topical mupirocin; Complication: PSGN. Bullous Impetigo: bullae formation with honey crusts, Tx: topical mupirocin + PO Keflex SSSS: severe form of bullous impetigo (extensive bullae), infants/young children, rash: erythroderma (perioral is classic), NO mucosal involvement, (+) Nikolsky's, Tx: PCN (e.g. Dicloxacillin) ± MRSA coverage (e.g. Vancomycin), admit</p>
<p>Diagnosis, Treatment: Acute Rheumatic Fever</p>	<p>Child with recent hx of strep throat that has fever, migratory polyarthralgia, signs of pericarditis or myocarditis/CHF, subcutaneous nodules, sydenham's chorea (rare, but often tested). All parts of JONES criteria. Tx: Penicillin. ASA/NSAIDs for the arthritis.</p>
<p>Complication of untreated rheumatic fever</p>	<p>Mitral stenosis as an adult</p>

<p>Signs & Symptoms, Treatment: Scarlet Fever</p>	<p>Age 2-10. Group A Strep infxn SSx: pharyngitis, strawberry tongue, circumoral pallor, rash: sandpaper feel, groin/axilla → trunk/extremity, spares palms/soles, pastia lines (linear petechiae) Tx: PCN to reduce incidence of rheumatic fever (not glomerulonephritis)</p>
<p>Signs & Symptoms, Treatment: Erysipelas</p>	<p>Upper dermal infection with GAS SSx: erythematous plaque, sharp border (cellulitis is less discrete), ± ears Tx: Amoxicillin/Keflex (mild), Ceftriaxone (systemic dz)</p>
<p>Diagnosis, Treatment: Pityriasis Rosea</p>	<p>SSx: Herald patch → Christmas tree distribution of rash on back Tx: improved with sunlight, antihistamines (pruritus), otherwise self-resolves</p>
<p>Distinguish, Diagnosis, and Treatment: Scabies vs. Lice</p>	<p>Scabies: linear burrows (pathognomonic), pruritic rash (hand/feet/groin); Tx: permethrin (NOT lindane for peds/pregnant → seizures), Ivermectin Lice: nits (eggs attached to hairs) with extreme pruritus on head; Tx: permethrin (alt. malathion), scrape out nits, repeat treatment in 7-10 days</p>
<p>Diagnosis, Treatment: Bed bugs</p>	<p>Pain immediately after bite. Can develop papules, bullae, and wheals. Lines of bites. Tx: antihistamines for itching, consider topical steroids for inflammation, supportive care, hot water washing for bedding</p>
<p>Signs & Symptoms, Treatment, Complication: Kawasaki Disease</p>	<p>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) Tx: high-dose ASA, IVIG. Complication: coronary artery aneurysm</p>
<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment, Complication: Henoch-Schönlein Purpura</p>	<p>Pathophys: Post-infectious vasculitis (IgA deposition); most common vasculitis in Peds. Age 6 mo-5 yrs SSx: (TRIAD): palpable purpura + colicky abd pain + arthralgia Dx: hemolytic anemia, normal/high platelet count, AKI, lethargy Tx: 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)</p>
<p>What type of intussusception is typically seen in Henoch-Schonlein Purpura?</p>	<p>ileo-ileo **The most common type of intussusception outside of HSP is ileocecal</p>
<p>Bacterial cause, Signs & Symptoms, Treatment: Cat Scratch Disease</p>	<p>Bartonella henselae SSx: cat scratch/bite 1-3 weeks prior, causes regional LAD (up arm and into axilla) Tx: Doxycycline (Azithromycin in pregnancy)</p>

<p>Febrile Seizures: Simple vs Complex</p>	<p>Febrile sz criteria: convulsions + fever, 6 mo-5 yr (NOT < 6mo), no CNS infxn/inflammation, no metabolic abnormalities, no h/o non-febrile sz Simple: 6 mo-5 yrs, single episode/24hr, <15 min, generalized (tonic/clonic); no neuro hx and normal exam; no special workup or tx needed Complex: anything else</p>
<p>What are the most common midline and lateral congenital neck masses?</p>	<p>Midline: thyroglossal duct cyst - moves up and down with tongue protrusion Lateral: branchial cleft cyst (vs. cystic hemangioma)</p>
<p>Most common objects, Signs & Symptoms, Diagnosis, Treatment: Peds Respiratory Foreign Body</p>	<p>Objects: coins (most common), peanuts, beans SSx: high suspicion if sudden choking or coughing ± wheezing, stridor. Often unsupervised child Dx: CXR: obstructive emphysema (FB obstructs bronchus on expiration, hypodense), CT if in doubt Tx: bronch (gold standard for Dx & Tx)</p>
<p>What is the PALS approach to resuscitation in a choking child <1 yr?</p>	<p>5 back blows, 5 chest compressions (no abd compressions)</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Croup</p>	<p>Age: 6 mo - 3 yr Pathogen: Parainfluenza virus SSx: URI sx with barking, seal-like cough, inspiratory stridor, low grade fever, non-toxic appearance Dx: mostly clinical diagnosis, XR: steeple sign Tx: dexamethasone (0.6mg/kg), racemic epinephrine for stridor at rest (before steroids if given, monitor for rebound x4hr); Dispo: admit if sick, hypoxic, or with persistent stridor. Consider bacterial superinfection</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Epiglottitis</p>	<p>Age: 3 - 7 yr Pathogen: <i>H. influenzae</i> (less since vaccine, now more adults), Strep. spp. (most common), <i>S. aureus</i> SSx: toxic appearance, rapid progression of high fever, dysphagia, leaning forward/"tripod" position, dysphonia, drooling, inspiratory stridor Dx: XR: thumbprint sign Tx: airway management (OR for eval, BVM ok, avoid RSI), IV abx (CTX)</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Bacterial Tracheitis</p>	<p>Age 3 - 5 yr Pathogen: <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. breathe better when they lay flat. **Croup but TOXIC appearing Dx: XR: subglottic narrowing, hazy tracheal lumen Tx: airway management, IV abx. They do not respond to racemic epi or steroids.</p>

<p>Signs & Symptoms, Diagnosis, Treatment: Retropharyngeal Abscess</p>	<p>Age: 6 m - 4 yr Pathogen: 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 Dx: XR: widened prevertebral space 7 mm at C2, CT neck w contrast (imaging study of choice) Tx: Amp+sulbactam, ENT/OR</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Bronchiolitis</p>	<p>Age < 2 yrs Pathogen: RSV (most common) Lower airway inflammation SSx: URI prodrome → fever, tachypnea, wheeze, apnea (< 1 mo), lasts 1-2 wks Dx: CXR: diffuse infiltrates Tx: mild → nasal suctioning, hydration; severe → trial of nebs (controversial), suctioning, humidified HFNC Dispo: admit if persistently hypoxemic or < 3 mo for apnea monitoring</p>
<p>Most common causes of pneumonia by age group: < 3 mo, 3 mo - 5 yr, > 5 yr. What are the treatments by age?</p>	<p>0-3 wks: GBS, E. Coli, Listeria, Staph 3 wk - 3 mo: C. trachomatis, RSV, pertussis 1 mo - 5 yrs: RSV, numerous viruses, <i>S. Pneumo</i>, atypicals > 5 yrs: M. pneumoniae, atypicals Tx: Neonates: septic w/u, Amp + Gent or Cefotax, admit, 3 wk - 3 mo: Azithro ± Cefotax, 3 mo - 18 yrs: Vanc + CTX ± Azithro (ICU), CTX (inpt), Amox or Azithro (outpt)</p>
<p>Workup, Treatment: Fever in kids < 4 wks, 4-8 wks</p>	<p>< 4 wks: (GBS, <i>E. coli</i>, Listeria) blood & urine cultures, XR, LP, admit; Tx: ampicillin + cefotaxime or gentamycine, add acyclovir & vancomycin depending on risk 4-8 wks: use clinical decision rules (Philadelphia, Rochester, Boston - generally well appearing, full-term, WBC <15, bands <1.5, CSF wnl, UA WBC <10); LP, abx Dispo: home (low risk), admit (high risk). **Always send UA</p>
<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Myocarditis vs. Pericarditis in kids</p>	<p>Myocarditis: Most common cause of HF in kids. Viral infxn (MC Parvovirus) SSx: poor feeding & sweating, increased RR/HR, hepatomegaly (most specific finding or HF) Dx: nonspecific ECG, +trop Tx: diuretics, inotropes, IVIG, admit, consider ECMO Pericarditis: viral infection (Coxsackievirus) SSx: fever + dyspnea + chest pain (worse lying flat) Dx: ECG diffuse STE or PR depression, trop (-), get TTE r/o effusion Tx: NSAIDs</p>

<p>Signs & Symptoms, Treatment: PDA vs. ASD vs. VSD in kids</p>	<p>PDA: L→R shunt (Ao→PA) SSx: continuous machine murmur, wide PP, **may be worse with O2** Tx: indomethacin, surgery</p> <p>ASD: L→R shunt & R heart failure SSx: asymptomatic unless large (often delayed dx), fixed split S2</p> <p>VSD: most common congenital heart dz; L→R shunt R heart failure SSx: loud, harsh holosystolic murmur at LLSB (smaller the defect, the louder the murmur), usually presents @ 6 wks Tx: heart failure tx, peds cards consult ± surgery</p>
<p>What are the 5 cyanotic congenital heart lesions?</p>	<p>All R→L shunt (eaRLy cyanosis) Truncus Arteriosus, Transposition of Great Arteries, Tricuspid Atresia, Tetralogy of Fallot, Total Anomalous Pulmonary Venous Return</p>
<p>Diagnosis, Treatment: Cyanosis in a 2-10 day old neonate with a murmur</p>	<p>Dx: Cyanotic congenital heart disease (ductal dependent). Rapid decompensation with cyanosis at day 2-10 (when ductus closes) Lesions: coarctation of aorta, critical aortic stenosis, hypoplastic left heart, tricuspid atresia, tetralogy, transposition Tx: PGE1 to reopen duct (side effects: hypotension, apnea, cardiac arrest), admit to PICU</p>
<p>What is the hyperoxia test and what is it used for?</p>	<p>Test used to differentiate sick infants/neonates with cyanotic congenital heart disease from those with pulmonary disease. Place the patient on 100% oxygen for 10 minutes and check an ABG from the R radial artery. If the PaO2 is <100-150 after 10 minutes, this is more consistent with cyanotic CHD with R>L shunting.</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Tetralogy of Fallot</p>	<p>Key features: RVH, RVOT stenosis, VSD, overriding aorta SSx: cyanosis day 2-10 with duct closure, shock, little improvement with O2 Dx: XR boot-shaped heart, ECHO Tx: PGE1, bicarb, IVF, blood, sat to 70s is ok</p>
<p>Describe a typical tet spell and appropriate treatment</p>	<p>Hypercyanosis associated with feeding, straining, crying, or exertion Tx: increased SVR (knee to chest, squatting), O2, morphine (decreased PVR)</p>
<p>Describe the presentation of Coarctation of the Aorta based on age</p>	<p>Neonate: HF & shock @ day 2-10 2/2 ductus closure Infant/child: HTN UE>LE, UE pulse delay and HF sx Adults: HTN, delayed LE pulses XR: rib notching</p>

What is the appropriate administration of glucose for hypoglycemia in neonates and kids?	"Rule of 50" or "5-2-1" < 1 yr: D10 (5cc/kg) 1-8 yrs: D25 (2cc/kg) >8 yrs: D50 (1cc/kg)
Signs & Symptoms, Diagnosis, Treatment: Congenital Adrenal Hyperplasia	SSx: Virilization ("girls look like boys, boys not much different") & salt wasting. 21-hydroxylase most common Dx: adrenal crisis (↓ Na, ↑↑ K) Tx: glucose, IVF, IV steroids (Hydrocortisone, not dexamethasone, which only has glucocorticoid activity)
Signs & Symptoms, Diagnosis, Treatment: (Generic) Inborn Error of Metabolism	Ammonia & acid production Present at day 3-5 SSx: tachypnea, vomiting, AMS, seizures, odd smell Dx: hypoglycemia, metabolic acidosis, hyperammonemia Tx: stop protein breakdown (NPO, IVF), glucose (D10, not D5), remove ammonia (NH3 scavenging meds)
Diagnosis: 3 - 5 yo patient with painless abdominal mass	Wilms Tumor (nephroblastoma)
Age, Risk Factors, Protective Factors for SUDI/SIDS	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 (campaign was "Back to Bed"), loose bedding, soft sleeping surface, male, prematurity, +FHx Protective: breast feeding, pacifier, supine sleeping
Criteria, Diagnosis, Treatment: BRUE	Brief Resolved Unexplained Event; formerly ALTE Associations: Pertussis, RSV. Idiopathic = most common cause Criteria: Peak age: 1 wk-2 mo (must be < 12 mo). Sudden, brief, now resolved episode including: cyanosis or pallor, irregular breathing, change in tone, ALOC with return to baseline Dx: workup per h&p and high vs low risk. Pertussis screen and ECG Tx: low threshold to admit for board exam. Only dc select very low risk episodes
Diagnosis, Treatment: infant with seizures, financial insecurity	Hyponatremia from diluted feeds Tx: hypertonic saline (2 ml/kg 3% NaCl)
Diagnosis: AMS, kid with ETOH ingestion, hx of DM	Hypoglycemia (replete per rule of 50)
Diagnosis, Treatment: kid at grandma's house with AMS, pinpoint pupils	Clonidine ingestion Tx: narcan
Diagnosis: kid with lethargy, intermittent crying and abdominal pain	Intussusception
Diagnosis: kid with bloody diarrhea, lethargy	E Coli O157:H7 & possibly HUS **NO abx → increases chance of HUS
Diagnosis: child crying, boyfriend babysits	Non-accidental trauma/abuse
Diagnosis: Recurrent RML pneumonia in a child	Aspirated foreign body / Ball valve effect and post-obstructive PNA

<p>Diagnosis, Treatment: Phimosis vs. Paraphimosis</p>	<p>Phimosis: unable to retract foreskin, NOT an emergency if patient can urinate Tx: topical steroid cream, gentle retraction</p> <p>Paraphimosis: inability to reduce foreskin back to anatomic position, emergency ("call the paramedics") → causes ischemia Tx: manual reduction, Urology consult (dorsal slit procedure, circumcision)</p>
<p>Risks for UTI based on sex/age, criteria for sending urine culture, and dispo criteria for UTI in kids</p>	<p>Males: < 1 yr & uncircumcised < 2 yrs Females: ALL, esp < 2 yrs Ucx analysis: suprapubic aspiration (rare but gold standard), even if urine dip normal, >50K CFU+ Fever + UTI = Pyelonephritis requires admit for IV abx, otherwise home with abx</p>
<p>What is a common cause of UTI < 1 yr?</p>	<p>50% with vesicoureteral reflux or other structural abnormality</p>
<p>Diagnosis, Treatment: Reye's Syndrome</p>	<p>Kid takes aspirin for viral URI → AMS and fatty degeneration of the liver, cerebral edema Tx: supportive.</p>
<p>What is the most common cause for meningitis in a neonate?</p>	<p>Group B Strep, E. coli, or Listeria</p>
<p>Diagnosis: Rectal prolapse in a kid</p>	<p>Cystic Fibrosis</p>
<p>Common Pathogens, Treatment: neonatal conjunctivitis</p>	<p>Rule of 5's N. gonorrhoeae (d 0-5), lots of purulent discharge, Tx: IV cefotaxime C. trachomatis (5 d-5 wk), Tx: oral macrolide to prevent pneumonia Strep or H. flu (5 wk-5 yr) **Bonus: The eye is most common site of gonorrhea in newborns</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Legg-Calve-Perthes disease</p>	<p>Idiopathic avascular necrosis of one/both femoral heads Male 4-10 yrs SSx: limp + unilateral hip/thigh/knee pain, worse with activity. Limited hip abduction & internal rotation, ± limb length discrepancies Dx: XR "moth eaten", "crescent sign" Tx: non-weight bearing, Ortho referral</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment, Complications: Slipped Capital Femoral Epiphysis (SCFE)</p>	<p>Most common hip disorder in teens Obese males. Age 12-16 yrs SSx: limp (L>R), hip pain, ext. rotation deformity Dx: XR pelvis ("ice cream falling off of cone") Tx: non-weight bearing, surgery, admit Complication: avascular necrosis</p>

<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Transient Synovitis</p>	<p>Inflammation + hypertrophy of hip synovium. Most common cause of hip pain in 3-10 yrs SSx: URI hx, limp with decreased ROM. Hip held in flexion, ABduction, and ext. rotation. Dx: Normal labs. XR: normal, US: ± joint effusion. ±Arthrocentesis (normal). Tx: NSAIDs</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Septic Arthritis</p>	<p>Infection of the joint space (most commonly S. aureus). Male < 4yrs Knee (most common joint) > hip SSx: fever, irritability, pain, refusal to bear weight or move joint; hip, held in flexion, ABduction and external rotation Dx: WBC > 12k, ESR > 40, fever, refusal to bear weight (Kocher criteria). Synovial fluid will show WBC > 50,000 with > 75% PMNs Tx: IV abx, OR for washout</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Post-Streptococcal Glomerulonephritis</p>	<p>Follows a GAS infxn (pharyngitis > impetigo) SSx: HTN, hematuria, periorbital edema Dx: UA: proteinuria, RBC casts. Others: +ASO titer, low C3 level Tx: supportive. Water restriction and lasix. Abx do NOT prevent this dz.</p>
<p>What are the 3 most common disease associations with pediatric biliary colic?</p>	<ol style="list-style-type: none"> 1. Hemolytic anemia (e.,g. sickle cell dz) is most common. Hemolysis → pigmented stones 2. Cystic fibrosis 3. Obesity (less so)
<p>What is an apt test?</p>	<p>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 fetal blood it will stay pinkish, red. Maternal blood will degrade.</p>
<p>Diagnosis, Treatment: Infantile Spasms</p>	<p>Presents at 4-8 months of age Dx: triad of clusters of myoclonic seizures on awakening + hypsarrhythmia on EEG + developmental delay Tx: ACTH, prednisone and AEDs Association: tuberous sclerosis (10-20% of cases)</p>
<p>What medication is associated with development of pyloric stenosis?</p>	<p>Macrolides: Azithromycin, erythromycin</p>
<p>What bilirubin concentration puts neonate at risk of kernicterus?</p>	<p>Total Bilirubin > 25 Requires phototherapy</p>
<p>What are clinical features of kernicterus?</p>	<p>Lethargy, hypotonia, poor feeding, eventually develop choreoathetoid cerebral palsy, hearing loss, gaze abnormalities MRI imaging will find signals in globus pallidus</p>
<p>What is surgical airway of choice in children less than 10 years old?</p>	<p>Needle cricothyrotomy (attach 3.0 mm endotracheal tube adapter directly to angiocatheter of 14 to 16 gauge and bag ventilate)</p>

What are the I:E ratios used when using a BVM through a needle cric ?	Incomplete airway obstruction: 1:5 or 1:6 (10-12 breaths per minute) Complete airway obstruction: 1:10 to 1:12 (5-6 breaths per minute) **Major difficulty will be exhalation and avoiding barotrauma
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Gastrointestinal

Bizz	Buzz
What pain medication is best for biliary colic ?	NSAIDS , it is prostaglandin-mediated pain
Diagnosis, Treatment: RUQ US with gallstones and dilated common bile duct	Dx: Choledocolithiasis, ± Jaundice Tx: ERCP
Gold standard diagnostic test: choledocolithiasis	MRCP. ERCP and endoscopic US are good as well but they are invasive. HIDA scan not helpful.
What is the sensitivity of Murphy's sign for Acute Cholecystitis?	65%
US findings: Acute Cholecystitis	Gallstones, gallbladder wall thickening (> 3 mm), pericholecystic fluid, sonographic Murphy's
What is Acalculous Cholecystitis ? Who is at high risk?	Inflamed gallbladder but <u>NO</u> stone ; typically in the critically ill , post-operative or elderly. higher rates of gangrene, higher mortality
Diagnosis, Treatment: Fever + RUQ pain + Jaundice	These symptoms are Charcot's Triad . For Reynold's Pentad, add AMS and hypotension. Dx: Ascending Cholangitis - biliary obstruction with ascending bacterial infection; HIGH Mortality. Tx: broad spectrum antibiotics, ERCP vs surgery
What malignancy is associated with chronic RUQ abdominal 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
Diagnosis: abdominal pain with bruising around the flank and umbilicus	Hemorrhagic Pancreatitis. Ecchymosis of left flank (Grey-Turner sign), Peri-umbilical ecchymosis (Cullen sign)
Does lipase level correlate with severity of disease in Pancreatitis?	No, useful for diagnosis but not prognosis
What are the components of Ranson's Criteria in Acute Pancreatitis?	<u>At admission/In ED:</u> Age > 55, WBC > 16k, Glucose > 200, LDH > 350, AST > 250. Helps predict inpatient mortality
What are 2 potential long-term consequences of Chronic Pancreatitis ?	Malabsorption when 90% affected, late-onset type I diabetes
What malignancy is associated with painless jaundice and palpable gallbladder (Courvoisier sign)?	Pancreatic Cancer ; most common at head of pancreas, high mortality, high CA 19-9
What malignancy is associated with migratory thrombophlebitis ?	Pancreatic Cancer; also called "Trousseau's sign"
What is the difference between incarcerated and strangulated hernias?	Incarcerated: not reducible Strangulated: not reducible <u>and</u> ischemic (requires surgery)
What are the structures that make up Hesselbach's triangle ?	Inguinal ligament, inferior epigastric vessels, lateral border of rectus abdominis
What do you call an inguinal hernia <u>medial</u> to the inferior epigastric artery?	Direct inguinal hernia. Less likely to incarcerate. Do not typically descend into the scrotum.

What do you call an inguinal hernia <u>lateral</u> to the inferior epigastric artery?	Indirect inguinal hernia. most common inguinal hernia in men and women. involves the internal inguinal ring. Often descends into the scrotum.
What type of hernia appears on the medial thigh ? Who is at highest risk?	Obturator hernia. Elderly women are at highest risk.
Location and Treatment: femoral hernia	Location: anterior thigh below the inguinal ligament Tx: urgent surgery consult due to very high risk of strangulation
What is the underlying pathology in Achalasia ?	Impaired relaxation of the lower esophageal sphincter (LES), absence of peristalsis; most common esophageal motility disorder. Patients will present with dysphagia and they will “raise their arms above their heads” or “straighten their backs” after eating to increase intraesophageal pressure
Diagnosis, Signs and Symptoms, Diagnostic Test, Treatment: ill-appearing patient with chest pain after vomiting	Dx: Boerhaave's Syndrome - <u>full-thickness</u> perforation of esophagus causing mediastinitis. SSx: Mackler's Triad: SubQ emphysema + chest pain + vomiting; "Hamman's Crunch" (crunching sound around heart). Diagnostic Test: esophagram (water soluble) or CT w/ contrast. Tx: antibiotics, surgical consult
On what side of the esophagus is rupture most common?	Left side (distal posterolateral esophagus).
What condition predisposes to spontaneous esophageal rupture ? Treatment?	Esophageal Candidiasis (consider in HIV patient). Tx: oral fluconazole , IV fluconazole if septic or cannot tolerate PO.
Diagnosis: regurgitating food and recurrent aspiration pneumonia	Esophageal Diverticula (Zenker's is pharyngeal mucosa above UES)
Diagnosis: a 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 24 hours or more, airway compromised, or evidence of perforation.
Management: esophageal food impaction	EGD. You can try Glucagon 1 mg IV (relaxes LES and causes vomiting) while you wait for GI; if glucagon works, patients must follow up 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
Diagnosis: small volume of blood after frequent emesis	Mallory-Weiss Syndrome: longitudinal, <u>partial-thickness</u> esophageal tear in the distal esophagus and proximal stomach

Diagnosis: pediatric patient with respiratory distress with feeding and recurrent pneumonia	Tracheoesophageal Fistula Commonly presents on tests as a NG tube that enters the lungs
Diagnosis: pediatric patient who presents not being able to tolerate solid foods but can have liquids	Esophageal web Think of Plummer-Vinson syndrome and associated with iron deficiency anemia
Diagnosis, risk factors: malignancy with a history of smoking, chest pain, and dysphagia	Dx: Esophageal Cancer (most likely Squamous Cell). Risk factors: males, heavy alcohol use, smoking, chronic GERD/Barrett's esophagus.
Diagnosis, Treatment: HIV patient with chest pain and dysphagia	Dx: Candida Esophagitis (with risk of perforation). Tx: oral fluconazole or IV fluconazole if they cannot tolerate PO. (nystatin is only for oral disease)
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 patients getting treatment for acne, clinda), anti-inflammatories (NSAID/ASA), bisphosphonates , iron , vitamin c , potassium chloride . Tx: stop inciting medication; endoscopy if severe or persistent symptoms
What type of caustic ingestion is worse and why?	Alkali ingestions are worse (cause liquefactive necrosis and deeper burns) than Acid ingestions (cause coagulative necrosis and more superficial damage)
Management: caustic ingestion	Get upright CXR to r/o perforation, consult GI for early endoscopy , consult surgery as needed. Do NOT induce vomiting or attempt decontamination/neutralization.
What is the most common cause of Cirrhosis in the US and the rest of the world?	Alcohol in US; Hepatitis C outside the US
Most common complication of cirrhosis ?	Ascites
Most common cause, Management: upper GI bleed in Cirrhosis	Esophageal Varices 2/2 portal HTN; Tx: airway protection, blood transfusion, PPI , octreotide , ceftriaxone (mortality benefit!), GI consult for endoscopy vs IR for TIPS
What medication improves mortality when given for variceal bleeding ?	Ceftriaxone ; likely prevents 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
Diagnosis, Pathophysiology, Treatment: cirrhosis and new renal dysfunction	Dx: Hepatorenal syndrome (acute renal failure without other reversible cause). Pathophysiology: Cirrhosis → splanchnic vasodilation → decreased renal blood flow → activation of RAAS and increased sympathetic tone → further decreased renal blood flow → renal failure. Tx: Albumin decreases mortality (as it prevents large fluid shifts after large-volume paracentesis).

Diagnosis, Triggers, Treatment: patient with cirrhosis presents with altered mental status	Dx: Hepatic Encephalopathy - accumulation of nitrogenous waste (e.g. ammonia). Triggers: infection (SBP is most common), GI bleed, meds, or constipation. Tx: lactulose/Rifaximin and find/treat the underlying cause.
What are the symptoms associated with the 4 stages of hepatic encephalopathy ?	I: mild confusion and agitation. II: drowsiness, disorientation, inappropriate behavior. III: Somnolent, confused, slurred speech. IV: Coma.
What is the usual source of infection for Spontaneous Bacterial Peritonitis (SBP)?	bacterial translocation from gut
What are the diagnostic criteria for Spontaneous Bacterial Peritonitis (SBP)?	Paracentesis with PMN (neutrophils) > 250, pH < 7.34, low glucose, (+) gram stain/culture
What are the diagnostic criteria for Spontaneous Bacterial Peritonitis (SBP) in patients on peritoneal dialysis ?	Paracentesis with > 100 WBC and > 50% PMNs
What are the two main types of Liver Abscess and what is the correct treatment ?	Pyogenic: (80% in US), sepsis/RUQ pain/ JAUNDICE , mixed bacteria (staph/strep); Tx: broad spectrum antibiotics (Ceftriaxone, Ampicillin, metronidazole), surgical drainage. Amoebic: (10%) usually subacute presentation, 2/2 entamoeba histolytica, no jaundice . possible complication is amoebic dysentery; Tx: metronidazole, medical management. Note: BOTH may cause biliary obstruction.
What lab abnormalities are expected with Acute Viral Hepatitis ?	Elevated AST/ALT (to 1000s) , high conjugated and unconjugated bilirubin, high Alk Phos, Coagulopathy
Which is more common: Hep B or Hep C?	Hepatitis C - 85%. Hepatitis B - 15%
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
Antibodies and Antigens 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 HBV <u>HBeAg</u> : high infectivity <u>Anti-HBeAb</u> : low infectivity

<p>Diagnosis, Signs and Symptoms, Diagnostic Test, Treatment: Patient with a history of Afib presenting with severe abdominal pain</p>	<p>Dx: Mesenteric Ischemia - 2/2 embolism (50%). Most common location is jejunum via SMA occlusion. SSx: severe pain out of proportion to exam (nonfocal abd exam), lactic acidosis (late finding, high mortality). Diagnostic Test: Xray to rule out free air, CTA abdomen is gold standard. Tx: antibiotics, anticoagulation, surgery consult</p>
<p>What is the most commonly affected vessel in Mesenteric Ischemia?</p>	<p>Superior Mesenteric Artery</p>
<p>Describe Rovsing sign, Psoas sign, and Obturator sign associated with appendicitis</p>	<p><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)</p>
<p>What are diagnostic criteria for diagnosis of acute appendicitis on US?</p>	<p>Non-compressible, tubular structure with a diameter ≥ 6 mm must be visualized. Others: fluid, target sign, appendicolith</p>
<p>What is the most common type of bezoar?</p>	<p>Phytobezoar (food, fiber); Others: Trichobezoar (hair), Pharmacobezoar (antacids, aspirin)</p>
<p>What is the significance of an enlarged left supraclavicular lymph node?</p>	<p>Indicative of GI malignancy, "Virchow node"</p>
<p>What is a common contributing cause of Gastric Adenocarcinoma?</p>	<p>H. pylori. Most common cancer with H.pylori is Mucosa-associated lymphoid tissue lymphoma (MALToma)</p>
<p>What is the significance of an enlarged periumbilical lymph node?</p>	<p>Indicative of metastatic spread of malignancy to peritoneum, "Sister Mary Joseph node"</p>
<p>Describe the intestinal and extraintestinal manifestations of Crohn's Disease. Treatment?</p>	<p>Intestinal: terminal ileitis (classic) [remember yersinia can also have terminal ileitis], full thickness involvement, "skip lesions" of normal bowel between disease, can involve ANY part of the GI tract. Complications - abscess, fistula, stricture, toxic megacolon. Extraintestinal: arthritis (most common), uveitis, erythema nodosum Tx: steroids, immunosuppressive</p>
<p>Describe the intestinal and extraintestinal manifestations of Ulcerative Colitis. Treatment?</p>	<p>Intestinal: continuous disease (no skip lesions) of rectum and colon ONLY, partial thickness involvement. Complications - toxic megacolon, increased risk of cancer. Extraintestinal: arthritis, uveitis, erythema nodosum Tx: steroids, less often antibiotics</p>
<p>What are the 3 most common causes of Small Bowel Obstruction?</p>	<p>Adhesions (very common with prior surgery) > tumor/mass > hernia</p>
<p>Diagnosis, Signs and Symptoms, Treatment: History of AAA repair with massive GI bleed</p>	<p>Dx: Aortoenteric fistula. SSx: Triad - GI bleed ("herald bleed") + abdominal pain + palpable mass; rare but high mortality. Tx: blood, surgical consult Note: Duodenum is most commonly involved portion of the intestines.</p>

<p>Diagnosis, Diagnostic Test, Treatment: patient with diarrhea after recent antibiotic use</p>	<p>Dx: <u>C.difficile</u> (anaerobic gram positive bacillus) causing Pseudomembranous Colitis Diagnostic Test: stool antigen Tx: metronidazole or vancomycin PO vs stool transplant</p>
<p>What are two signs/symptoms that EXCLUDE a diagnosis of Irritable Bowel Syndrome?</p>	<p>Fever or blood in stool</p>
<p>Patient population, Diagnostic Test, Treatment: Sigmoid vs Cecal Volvulus</p>	<p>Sigmoid: most common, elderly/nursing home, immobilized, chronic constipation. Triad: abdominal pain + distension + constipation. Diagnostic Test: Xray with inverted "U". Tx: sigmoidoscopy with rectal tube decompression (stable), surgery (definitive). Cecal: younger, marathon runners. Diagnostic Test: Xray with kidney-shaped/coffee-bean (massively dilated cecum in the LUQ), comma sign. Tx: surgery, antibiotics for perforation</p>
<p>Diagnosis, Patient population, Treatment: Sudden severe abdominal pain, abdominal distension and inability to pass NG tube</p>	<p>Dx: <u>Gastric Volvulus</u> - closed loop obstruction, ischemia and perforation. Patient population: elderly or infants w/ congenital diaphragmatic defect. Tx: try to pass a NG tube (on an adult). Call surgery.</p>
<p>Diagnosis, Treatment: Patient with a history of Roux-en-Y gastric bypass presenting with dizziness, tachycardia, and syncope soon after a meal.</p>	<p>Dx: <u>Dumping Syndrome</u> - rapid stomach emptying (pylorus removed) and rapid fluid shifts in small bowel. Typically a clinical diagnosis. Tx: fluids, electrolyte replacement, dietary modifications, smaller meals</p>
<p>Diagnosis, Treatment: Abscess above gluteal cleft near midline</p>	<p>Dx: <u>Pilonidal cyst/abscess</u> Tx: I&D in ED, surgical removal (definitive). recurrent disease is the most common complication</p>
<p>Causes, Signs and symptoms, Diagnostic Test, Treatment: Proctitis</p>	<p>Causes: Inflammation of the lining of the rectal mucosa caused by STDs (most common is gonorrhea), radiation, Crohn's. SSx: tenesmus, rectal discharge Diagnostic Test: sigmoidoscopy Tx: treat underlying cause - antibiotics, anti-inflammatories, biologics</p>
<p>What is the most common location for an anal fissure? What should be considered for anal fissures NOT at this location?</p>	<p>Posterior midline (90%). If anal fissure is lateral, should consider systemic process: Crohn's, HIV, leukemia, tuberculosis or syphilis</p>
<p>Treatment: thrombosed hemorrhoid</p>	<p>Excision of clot with elliptical incision (only if present <72 hours), sitz baths, topical lidocaine</p>
<p>What are the classifications of internal hemorrhoid severity I-IV?</p>	<p>I: don't protrude through anus II: prolapse but spontaneously reduce III: prolapse but require manual reduction IV: prolapse and cannot be reduced (\pm strangulation)</p>

<p>Patient population, Signs and Symptoms, Treatment: Rectal Prolapse</p>	<p>Patient population: Seen in young and elderly (related to constipation), also with anal intercourse; Consider cystic fibrosis in Peds. SSx: red mass protruding from anus Tx: manual reduction (can use granulated sugar), surgery consultation pm (ischemia)</p>
<p>Diagnosis, Patient population, Treatment: patient with large bowel obstruction without identified obstructing lesion on CT</p>	<p>Dx: Ogilvie's Syndrome: colonic pseudo-obstruction, massive dilatation of the colon (>10cm), <u>absence of mechanical obstruction.</u> Patient population: elderly/bedridden patients with comorbidities Tx: colonic decompression and neostigmine</p>
<p>What is the most common cause of surgical and non-surgical abdominal pain in the elderly?</p>	<p>Surgical: Acute Cholecystitis (**present with milder symptoms); Non-surgical: Pancreatitis</p>
<p>What is the most common cause of acute pancreatitis?</p>	<p>Gallstones (45%) > Alcohol (35%). Rationally, there are more people with gallstones than alcoholics in the world.</p>
<p>Treatment: epiploic appendagitis</p>	<p>NSAIDs, supportive, likely discharge with outpatient follow up. Presents similarly to appendicitis.</p>
<p>Treatment: intussusception in children and adults</p>	<p>Children: barium or air enema to reduce if uncomplicated Adults: surgery as most are associated with a mechanical cause (most often tumor)</p>
<p>Most common lab findings in ischemic hepatitis?</p>	<p>AST and ALT >10,000. Much higher than with viral hepatitis</p>
<p>What lab findings are seen in Gilbert Syndrome?</p>	<p>Elevated indirect (unconjugated) bilirubin</p>
<p>When is a G tube tract considered mature?</p>	<p>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.</p>
<p>What is hallmark symptom of Irritable Bowel Syndrome?</p>	<p>Pain improved with defecation</p>

Respiratory

Bizz	Buzz
Diagnosis, Cause, Signs and Symptoms: most common infectious airway obstruction in children	Dx: Croup , i.e. "laryngotracheobronchitis" Cause: <i>Parainfluenza</i> SSx: barky, seal-like cough worse at night, inspiratory stridor
What is the characteristic CXR finding in Croup ?	" Steeple sign " (tapering of the upper airway on AP view)
Management: Croup	<u>Dexamethasone for all.</u> Mild: home tx, antipyretics, fluids, mist. Moderate-Severe (stridor at rest): Racemic Epi → if stridor goes away, observe for 3 hours and then okay to discharge home if stridor does not persist/return or if child is well appearing. Admit if needs 2nd treatment. Things that support admission for croup: age <6 months, stridor at rest, respiratory distress, hypoxemia, lack of good follow up.
Diagnosis, Cause: Sore throat, normal posterior oropharynx, ill-appearing, in tripod position	Dx: Epiglottitis. Cause: <i>H.flu</i> (unvaccinated), <i>Staph/Strep</i> (vaccinated). More common in adults now thanks to the HiB vaccine
What is the characteristic CXR finding in Epiglottitis ?	" Thumbprint sign " (enlarged epiglottis on lateral view)
Treatment: Epiglottitis	Emergent airway management , antibiotics, steroids with ENT consult. Unstable/ill-appearing: go to the OR with ENT for direct visualization/scope. Respiratory arrest = BVM. No RSI! Well-appearing/stable: consider CXR.
Diagnosis, Signs and Symptoms, Diagnostic Test: Inspiratory whoop between violent coughing spells.	Dx: Pertussis. <i>Bordetella pertussis</i> SSx: (3 phases) Catarrhal (1-2 weeks): URI **HIGHLY CONTAGIOUS during this time** Paroxysmal (2-3 months): violent " whooping " cough, inspiratory stridor Convalescent (1-2 weeks): gradual reduction in symptoms Diagnostic Test: nasopharyngeal culture, PCR Notes: most common cause of death = pneumonia. Associations: seizures. Infants can present ONLY with apnea. One third of adults do not have the "whooping" either.
Treatment: Pertussis	Azithromycin = first line. No Clarithro/Erythro for infants < 1 month (increased risk of pyloric stenosis). Alternative: TMP-SMX. Make sure to treat close contacts!

<p>Diagnosis, Signs and Symptoms, Treatment: Bleeding from trach site weeks after placement</p>	<p>Dx: Tracheo-innominate fistula SSx: often smaller sentinel bleed weeks after placement followed by massive hemorrhage. Tx: hyperinflate trach cuff, intubate to compress bleeding through trach site, compress with finger. ENT to OR STAT. Extremely high mortality.</p>
<p>Diagnosis: Child alone in room starts coughing</p>	<p>Inhaled foreign body. Can have hyperinflation of the ipsilateral lung on CXR due to ball-valve effect</p>
<p>Treatment: suspected hereditary angioedema</p>	<p>Fresh frozen plasma or Icatibant (bradykinin antagonist); epi and H1/H2 blockers don't work</p>
<p>Causes, Antibiotic options: community acquired pneumonia (CAP) as an outpatient</p>	<p>Outpatient CAP Cause: <i>S. Pneumo</i> or <i>atypicals</i> Tx: healthy patients - Amoxicillin, Doxycycline or Azithromycin; Patients with comorbidities - respiratory tract fluoroquinolone (RTF) or Augmentin + Macrolide/Doxycycline</p>
<p>Antibiotic options: Pneumonia as an inpatient (Floor and ICU)?</p>	<p>Floor: add Gram negative coverage. Combo tx of ceftriaxone + azithromycin vs fluoroquinolone monotherapy ICU: antipneumococcal β-lactam (CTX or cefotaxime) + either azithromycin or an RTF \pm MRSA coverage (Vancomycin or Linezolid)</p>
<p>Most Common Cause, Signs and Symptoms: community acquired pneumonia</p>	<p>Cause: <i>Strep pneumoniae</i>, Gram positive lancet-shaped encapsulated diplococcus Key features: rusty sputum, lobar pneumonia SSx: acute onset + rigors, follows URI or influenza</p>
<p>Diagnosis, Signs and Symptoms, Diagnostic Test, Treatment: Pneumonia + history of Cystic Fibrosis</p>	<p>Dx: <i>Pseudomonas</i>, Gram negative rod SSx: green sputum, cough, fevers Diagnostic Test: CXR with multilobar pneumonia Tx: antipseudomonal antibiotics (e.g. cefepime)</p>
<p>Most Common Cause: pneumonia in young children with cystic fibrosis</p>	<p>Staph aureus, Haemophilus influenzae. By the time they are 18 yo, 80% of pts with CF will be colonized by pseudomonas though.</p>
<p>Diagnosis, Diagnostic Test, Treatment: Pneumonia + alcoholic + currant jelly sputum</p>	<p>Dx: <i>Klebsiella</i>, encapsulated Gram negative bacilli in pairs Key features: higher risk in alcoholics, diabetics, nursing homes. Diagnostic Test: CXR with RUL infiltrate + "bulging" fissure, air-fluid level Tx: cephalosporin (e.g. CTX) + Gentamicin/Amikacin</p>
<p>Diagnosis, Diagnostic Test: Pneumonia after Influenza</p>	<p>Dx: <i>Staph aureus</i>, Gram positive cocci in clusters Associations: IVDU, hospitalization. Appear sick. Diagnostic Test: CXR with patchy/multilobar/cavitary/abscess</p>
<p>Diagnosis: Intermittent cough and episodic diaphoresis, CXR with lung mass</p>	<p>Pulmonary carcinoid</p>

What are risk factors for Health Care Associated Pneumonia?	Nursing homes, hospitalization in the last 90 days, Dialysis , home IV antibiotics. More likely to have drug-resistant bugs and thus require broad coverage including <i>Pseudomonas</i> and MRSA. Note: HCAP is now defunct, replaced by Hospital Acquired Pneumonia. Know HCAP criteria (test lags behind practice a few years)
Diagnosis, Diagnostic Test, Treatment: Immunocompromised with marked dyspnea and hypoxemia	Dx: PCP pneumonia. (HIV with CD4 < 200.) Diagnostic Test: ↑ LDH , BAL specimen, CXR with "batwing" sign. Tx: TMP-SMX (first line), Alternative: IV Pentamidine (± hypoglycemia, hypotension), PO Dapsone (± methemoglobinemia). Add steroids if PaO2 < 70 (~SaO2 < 93%) or A-a gradient > 35
Diagnosis, Signs and Symptoms, Diagnostic Test, Treatment: Mild pneumonia symptoms and ear pain	Dx: <i>Mycoplasma pneumoniae</i> SSx: Atypical " walking " pneumonia. Young adults, bullous myringitis , Faget sign (fever + relative bradycardia) Dx: CXR shows diffuse interstitial pattern , +cold agglutinin test. Tx: Azithromycin Note: bullous myringitis is most commonly caused by <i>S. Pneumo</i> , but the test often associated with <i>Mycoplasma</i>
What extrapulmonary complications are associated with <i>Mycoplasma pneumoniae</i> ?	Aseptic meningitis, hemolytic anemia, Guillain-Barré, erythema multiforme, bullous myringitis
Diagnosis, Signs and Symptoms, Diagnostic Test, Treatment: Pneumonia + Gram Positive Rods + Widened mediastinum on XR + occupation working with sheep/alpaca	Dx: Pulmonary anthrax SSx: viral prodrome, transient improvement, rapid decline with hypoxia, respiratory distress, shock, mediastinitis Diagnostic Test: CXR with widened mediastinum (typically because of hemorrhage) Tx: Ciprofloxacin OR Doxycycline and Clindamycin. Post-exposure prophylaxis for exposed contacts. Note: Cutaneous manifestations are often described as " eschar ." Consider bio-terrorism. Also called "wool sorters disease" - think about sheep, alpacas , and other wooly animals
Diagnosis: Infant with staccato cough	<i>Chlamydia pneumoniae</i>
Diagnosis, Treatment: Pneumonia and headache in a bird owner	Dx: Psittacosis, <i>Chlamydia psittaci</i>. Tx: Doxycycline (or Azithromycin)
Most Common Cause: <u>viral</u> pneumonia in adults	Influenza
Diagnosis, Treatment: Cough and respiratory failure after exposure to rodents	Dx: Hantavirus Tx: supportive care only Note: commonly progresses to ARDS

<p>Diagnosis, Signs and Symptoms, Diagnostic Test, Treatment: Pneumonia + diarrhea + hyponatremia</p>	<p>Dx: Legionnaires 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 symptoms (n/v/d), neurologic symptoms (confusion, seizure). Diagnostic Test: hyponatremia, CXR shows patchy alveolar infiltrates, urinary antigen testing. Tx: Azithromycin or fluoroquinolone (severe disease)</p>
<p>Diagnosis, Cause, Diagnostic Test, Treatment: Pneumonia + <u>sheep</u></p>	<p>Dx: Q fever Cause: <i>Coxiella burnetii</i>, obligate intracellular Gram negative bacterium Diagnostic Test: LFT abnormalities + proteinuria Tx: tetracycline</p>
<p>Diagnosis, Cause, Treatment: Pneumonia + high temp + hunter/butcher. Rabbit exposure.</p>	<p>Dx: Tularemia Cause: <i>Francisella tularensis</i>, a Gram negative coccobacillus Tx: Streptomycin Note: commonly referred to as a bioterrorism agent</p>
<p>Diagnosis, Diagnostic Test, Treatment: Pneumonia in alcoholic who passed out/vomiting</p>	<p>Dx: Aspiration pneumonia Diagnostic Test: CXR with RLL or RUL infiltrate. Tx: broad spectrum antibiotics (need Gram negative, anaerobes coverage)</p>
<p>What pathogen is associated with bullous myringitis accompanying pneumonia?</p>	<p>Classically <i>Mycoplasma pneumoniae</i>; newer studies suggest it is actually caused by <i>Strep pneumoniae</i>.</p>
<p>What is the underlying pathologic process in emphysema?</p>	<p>Irreversible destruction of alveolar septae. Airways collapse on exhalation, trapping air. Associated with smoking, certain jobs (e.g. ship-building), cystic fibrosis, <i>alpha</i>-1-antitrypsin</p>
<p>CXR findings: COPD</p>	<p>Hyperinflation, flat diaphragms, blebs/bullae</p>
<p>What is required for a diagnosis of chronic bronchitis?</p>	<p>A cough most days of the month, for 3 months each year, for at least 2 consecutive years</p>
<p>Differential: acute decompensation in COPD patient</p>	<p>Pneumothorax (high risk due to blebs), mucous plug, PE, MAT/arrhythmia, pneumonia</p>
<p>Review the approach to mechanical ventilation of a COPD patient</p>	<p>Goal is to avoid barotrauma. Minimize auto-PEEP by using lower RR (8-10/min) to lower I:E ratio, lower TV (5cc/kg), prolong expiration time, and tolerate respiratory acidosis/permissive hypercapnia</p>
<p>Review the approach to supplemental O2 in a COPD patient</p>	<p>They are chronically hypercapneic, 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 pre oxygenating</p>
<p>What are the typical pulmonary function test abnormalities in Asthma and COPD patients?</p>	<p>↓ FEV1, ↓FEV1/FVC, ↓ PEFr (peak expiratory flow rate). Means they can't exhale effectively.</p>

Review the approach to treatment of COPD patients	Supplemental O2 pm for target SpO2 88-92%, antibiotics for change in sputum or obvious infection, steroids, albuterol/ipratropium, BiPAP, epinephrine, terbutaline, intubate if all else fails.
What are the most likely causes/triggers for COPD and asthma ?	COPD: infection (virus) = most common cause of exacerbation. Always get CXR. Asthma: more likely 2/2 meds, exercise, allergens.
What is the underlying pathologic process in asthma ?	Triad of lower airway inflammation + bronchoconstriction due to hyperreactivity + reversible airflow obstruction
Diagnosis: Persistent cough in patient with atopic history	Cough-variant asthma
Management: 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). PEFR > 70% predicted has high likelihood of successful discharge. PEFR < 40% should be admitted.
Review the approach to treatment of asthma patient	Albuterol/ipratropium, steroids, supplemental O2 (if hypoxic), Magnesium if sick, Epi if sick, Terbutaline, 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, and decreases leukotriene and prostaglandin production. Note: these effects are delayed (onset 1-2 hours, peak 24 hours). No real immediate effect in ED.
Review the approach to intubation and mechanical ventilation of an asthma patient	Intubation indications: cardiac or respiratory arrest, physical exhaustion, AMS. Intubation: IVF before (PPV decreases preload and may cause hypotension). RSI: consider Ketamine. Ventilation: Goal is to avoid barotrauma. Minimize auto-PEEP by using lower RR (8-10/min) to lower I:E ratio, lower TV (5cc/kg), prolong expiration time, and tolerate respiratory acidosis/permissive hypercapnia
What is meant by delayed sequence intubation ?	Procedural sedation (typically with ketamine to preserve airway reflexes and respiratory drive) to help facilitate pre-oxygenation before RSI
What is the best measurement of airway compliance in a patient on a ventilator ?	Plateau pressure (i.e. alveolar pressure). Calculated via inspiratory hold . High plateau = poor compliance, Low plateau = good compliance. <u>Keep plateau pressure < 30 in asthma/COPD.</u> Peak pressure measures flow resistance in larger airways.
Diagnosis, Treatment: PEA arrest after intubation of asthma patient	Dx: Tension pneumothorax Tx: disconnect from vent, squeeze chest, place bilateral chest tubes, give IVF.

<p>Definition, Signs and Symptoms, Diagnostic Test, Treatment: Bronchiectasis</p>	<p>Definition: 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 with dilated, tortuous airways. Tx: antibiotics (cover <i>Pseudomonas</i>), albuterol.</p>
<p>Child or teenager with pancreatitis should raise suspicion for what condition?</p>	<p>Suspect cystic fibrosis (GI variant)</p>
<p>What is the pathophysiology of cystic fibrosis?</p>	<p>Autosomal recessive. Leads to mutations affecting Na/Cl exchange channel. Results in abnormally viscous mucous secretions. Multiple organ systems affected: recurrent pulmonary infxns (<i>Pseudomonas</i>), meconium ileus & intussusception, pancreatic insufficiency & pancreatitis</p>
<p>Diagnostic Test: cystic fibrosis</p>	<p>Elevated quantitative sweat chloride test or DNA testing</p>
<p>What is the infectious complication associated with esophageal rupture?</p>	<p>Mediastinitis (2/2 Boerhaave syndrome)</p>
<p>Most Common Cause: pleural effusion in elderly patients</p>	<p>Transudative causes</p>
<p>Pathophysiology, Management: exudative vs. transudative effusions</p>	<p>Exudative: Pathophysiology: damaged capillaries leak thick fluid usually 2/2 inflammation Management: effusion must be removed</p> <p>Transudative: Pathophysiology: intact capillaries leak thin fluid 2/2 increased hydrostatic or decreased oncotic pressure Management: treat the underlying cause</p>
<p>Common causes: exudative vs. transudative pleural effusions</p>	<p>Exudative: pulmonary infections (pneumonia) > malignancy (most common cause of massive effusions), PE (most common cause of isolated, unexplained effusion). Transudative: CHF (most common cause in US), renal failure, liver failure.</p>
<p>Review Light's Criteria to distinguish exudative from transudative pleural effusion</p>	<p>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.</p>
<p>Diagnosis: PCP/PJP pneumonia with sudden worsening shortness of breath</p>	<p>Pneumothorax (strong association PCP/PJP and PTX)</p>
<p>What is the most sensitive bedside test for possible pneumothorax? What indicates a positive test?</p>	<p>Bedside ultrasound (better than CXR). Normal lung will show "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.</p>

What are possible CXR findings in a supine patient with pneumothorax ?	"Deep sulcus" sign. Air rises anteriorly and tracks along the anterior diaphragm
Treatment: tension pneumothorax	Immediate needle decompression (2nd intercostal space at midclavicular line) followed by chest tube. Do NOT take the time to get CXR.
Diagnostic Test, Treatment: Empyema	Empyema = pus in pleural space. Dx: CXR with effusion +/- loculations, thoracentesis (pH < 7.20, WBC > 50k, glucose < 60, pus). Tx: ultimately requires fluid drainage (thoracentesis vs. tube thoracostomy vs. 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
Treatment: 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. After initial stabilization, the patient will need bronch or angio to identify source of bleeding.
Diagnosis, Treatment: Young person, massive hemoptysis, bilateral white out on CXR	Dx: Diffuse Alveolar Hemorrhage. Cause is usually inflammatory or autoimmune. Tx: high dose steroids, airway management, supportive care
Diagnosis, Risk Factors, Treatment: altered mental status + vomiting + patchy dependent consolidation	Dx: Aspiration pneumonitis , chemical pulmonary inflammation. Risk factors: alcoholism, seizure, neuromuscular disorder, dysphagia. Tx: supportive, monitor for development of aspiration pneumonia, no antibiotics unless true PNA.
Diagnosis, Most Common Cause, Treatment: Alcoholic with foul breath, cough and CXR with air fluid level	Dx: Lung abscess. Cause: Polymicrobial. Anaerobes are most common cause (e.g. Peptostreptococcus), <i>Staph</i> (post-influenza). CXR: consolidation + cavity/air-fluid level; aerobes/TB (upper lobe), anaerobes (lower lobe). Tx: antibiotics (ampicillin+sulbactam, zosyn, carbapenem), surgery if severe.
CXR findings: primary, reactivation and miliary TB	Primary: <u>lower</u> lobes, looks like pneumonia. Children: pronounced hilar adenopathy. Elderly: isolated pleural effusion. Reactivation: <u>upper</u> lobe granuloma ± cavitation . Miliary: scattered nodules (millet seeds) throughout lung fields

How is TB diagnosed?	<p><u>Sputum stain</u> (for AFB, suggestive, faster)</p> <p><u>Sputum culture</u> (gold standard, confirmatory test, takes weeks).</p> <p><u>Quantiferon Gold</u> (possible alternative, expensive).</p> <p><u>Tuberculin skin testing</u> (i.e. PPD, used to screen, but positive tests require follow up with CXR, Quant Gold, etc.).</p>
What defines a positive TB skin test ?	<p>Assessed for induration (not erythema).</p> <p>≥ 5 mm: HIV, immunosuppressed (e.g. organ transplant), close contact with active TB, abnormal CXR.</p> <p>≥ 10 mm: h/o IVDU, exposure to high risk setting (immigrant from TB-endemic area, jail, healthcare worker), children < 4 years old.</p> <p>≥ 15 mm: everyone else</p>
Treatment: latent and active TB	<p>Latent: Isoniazid (+ B6) for 6-9 months.</p> <p>Active: Rifampin, Isoniazid, Pyrazinamide, Ethambutol (alternative: Streptomycin) for 9-12 months</p>
What are potential side effects of the different drugs in RIPE therapy?	<p>Rifampin: <u>orange body fluids</u>, hepatitis, low platelets.</p> <p>Isoniazid: neuropathy (<u>B6 deficiency</u>), seizures (B6 for refractory cases), <u>hepatitis</u> (most common), drug-induced lupus.</p> <p>Pyrazinamide: hepatitis, high uric acid → gout, teratogenic.</p> <p>Ethambutol: <u>optic</u> neuritis, red-green blindness.</p> <p>Streptomycin: vestibular nerve damage, renal injury (contraindicated in pregnancy → congenital deafness)</p>
ECG changes: pulmonary embolism	<p>Sinus tachycardia (most common), nonspecific ST-T changes, precordial T wave inversions.</p> <p>R heart strain (e.g. RAD, new RBBB, p-pulmonale, S1Q3T3 = classic but rare).</p>
What are some echocardiographic signs of right heart strain ?	<p>RV dilation, RV hypokinesis, septal shift to the <u>LEFT</u>, tricuspid regurgitation, elevated pulmonary artery pressure, decreased LV filling (due to septal bowing), and impediment of LV output.</p>
What is McConnell's Sign on echo? What does it represent?	<p>Akinesis of the RV free wall that spares the apex. It indicates right heart strain and is highly specific for acute pulmonary embolism.</p>
Signs and Symptoms: pulmonary embolism	<p>Dyspnea (73%) and tachypnea (54%).</p> <p>Other "classic" symptoms are less common, such as calf pain/swelling (44%), pleuritic pain (44%), cough (37%), tachycardia (24%), hemoptysis (15%).</p>
CXR findings: pulmonary embolism	<p>CXR: nonspecific abnormalities, Hampton's hump (pleural-based wedge infarct), Westermark's sign (vascular cut-off sign)</p>
What is the appropriate workup for patients with clinical symptoms and multiple risk factors for DVT and PE?	<p>DVT: need negative D-dimer and Doppler US (repeat Doppler US if high risk and initial study negative) to exclude.</p> <p>PE: need negative imaging (CTA or VQ scan) to exclude</p>

When should thrombolytics be given and when should they be considered a patient with PE ?	Indication for Thrombolytics: hypotension/shock (sustained x 15 minutes), cardiac arrest. Indication to consider thrombolytics in PE: RV enlargement or dysfunction (ECG, TTE, or CT proven), extensive clot burden, severe hypoxemia, RA/RV thrombus, PFO
Diagnosis: IVDU + multiple infiltrates on CXR	Septic pulmonary emboli (raising concern for endocarditis)
Signs and Symptoms, Diagnostic Test, Treatment: pulmonary arterial hypertension	SSx: <u>SOB</u> (most common), chest pain, hypoxia, lower extremity and abdominal swelling. Diagnostic Test: TTE, cath, CT, CXR with enlarged pulmonary arteries, ECG with R heart strain. Tx: vasodilators (prostacyclins like Remodulin), optimizing fluid balance, pressors, <u>NO NITRATES</u> (preload dependent).
Causes: acute decompensation in patients with pulmonary hypertension	PE or Vasodilator IV pump failure
Causes: acute decompensation in patients with pulmonary fibrosis	Progression of disease vs. acute pneumonia
Diagnosis, Treatment: Non-caseating granulomas in lungs with bilateral hilar adenopathy	Dx: Sarcoidosis (associated with erythema nodosum) Tx: steroids
Definition, Signs and Symptoms, Diagnostic Test: Pneumoconiosis	Definition: lung disease caused by inhalation of organic or inorganic dust SSx: SOB, cough (non-productive), hypoxia Diagnostic Test: CXR and CT shows interstitial fibrosis
What are the associated risk factors for each example of pneumoconiosis? (Asbestosis, Berylliosis, Byssinosis, Coal worker's lung, Siderosis, Silicosis, Stannosis)	Asbestosis: shipping, roofing, plumbing (from the roof, but affects the base [lower lobes]) Berylliosis: aerospace , fluorescent bulbs Byssinosis: cotton Coal worker's lung: coal (from the base [earth], but affect the roof [upper lobes]) Silicosis: foundries, sandblasting, mines , increases risk for TB. (from the base [earth], but affect the roof [upper lobes]) Siderosis: arc welding (iron) Stannosis: tin welding
What are classically abnormal labs in patients with sarcoidosis ?	Hypercalcemia (reaction associated with granulomas), high ACE
What are the criteria for diagnosis of ARDS ?	1) acute onset (symptoms within 1 week of causative insult) 2) bilateral opacities on CXR/CT (pulm edema) 3) no cardiac cause 4) impaired O2 exchange (PaO2/FiO2 < 300)
Clinical features, Causes: ARDS	Features: Poor lung compliance, pulmonary edema, severe hypoxemia unresponsive to supplemental O2. Causes: shock states (gram negative sepsis = most common), trauma, almost anything EXCEPT heart failure.

What is the approach to "lung protection" in ventilated ARDS patients?	Low tidal volume (6-8 cc/kg), high PEEP (5-20), permissive hypercapnia but can increase RR as needed, supplemental O2, Plateau < 30, (PEEP & FiO2 should be titrated up together [ARDSnet trial])
What is the expected PCWP in ARDS vs. CHF?	Remember PCWP approximates LA Pressure. ARDS: <u>low/normal</u> PCWP (diagnosis requires it to not be caused by cardiac congestion) CHF: <u>high</u> PCWP
What mechanisms of hypoxemia cause an increase in the A-a gradient (>15)?	Right to left shunt, Diffusion impairment, V-Q mismatch
Diagnosis, CXR findings: Cough and ulnar neuropathy	Dx: Pancoast tumor CXR: mass at lung apex, causes brachial plexus compression
What size spontaneous pneumothorax can be managed with O2 and observation alone?	20% or less. Pneumothoraces resorb 1-2% per 24 hrs in healthy lungs. DOES NOT apply to secondary PTX (i.e. COPD, ILD, asthma, penetrating trauma)
What is the signs and symptoms and 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: right 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)
Signs and Symptoms, Diagnostic Test, Treatment: COVID-19	SSx: Generalized viral syndrome symptoms including fevers, chills, body aches, diarrhea, headache, loss of taste and smell. Dx: SARS-CoV2 PCR testing, CXR reveals bilateral interstitial infiltrates. Tx: Corticosteroids for patients with hypoxia , symptomatic treatment. Monoclonal antibodies (not tested).
What are important vent settings in intubated asthmatic patients to reduce "breath stacking" and barotrauma?	Reduce minute ventilation, adjust I:E ratio (inspiratory to expiratory ratio) to allow for longer expiration. ideally 1:4 or greater.
Treatment: refractory hiccups (i.e., > 48 hours)	Thorazine (FDA approved), baclofen, reglan, antacids; Always investigate for serious cause of hiccups prior to symptomatic treatment.

Infectious Disease

Bizz	Buzz
Diagnosis, Treatment: fish tank granuloma	Dx: <i>Mycobacterium marinum</i> , spreads on lymphatic channels - looks like sporotrichosis Tx: Clarithromycin, Doxycycline, TMP-SMX, or Ciprofloxacin
Diagnosis, Treatment: rose thorn injury and rash spreading up the arm	Dx: <i>Sporothrix schenckii</i> Tx: Itraconazole (amphotericin B if systemic)
Diagnosis, Treatment: dog and cat bite with rapid infection	Dx: <i>Pasteurella multocida</i> Tx: Amoxicillin-Clavulanate (Augmentin → “dog-mentin”), second line: doxycycline (“Dogs-ycycline”)
Diagnosis: reptile bites and infection	Dx: <i>Salmonella</i>
Diagnosis: sickle cell disease and joint pain/infection	Dx: <i>Salmonella osteomyelitis</i> . * <i>Staph aureus</i> is still most common cause of osteomyelitis in HbSS but <i>Salmonella</i> is very characteristic and testable
Diagnosis, Treatment: cat scratch, tender axillary lymphadenopathy two weeks later	Dx: <i>Bartonella henselae</i> Tx: Azithromycin
Diagnosis, Treatment: human bite and infection	Dx: <i>Eikenella corrodens</i> Tx: amoxicillin-clavulanate
Diagnosis: gastroenteritis on a cruise ship	Dx: Norovirus/Norwalk virus
Diagnosis: gastroenteritis at a daycare	Dx: Rotavirus *Remember, there is a vaccine now; also has an association with intussusception.
Compare general diagnosis and treatment of toxin-mediated vs. invasive bacterial diarrheal illness	Toxin: abrupt onset, watery, non-bloody Tx: IVF, ± loperamide, ± ciprofloxacin if prolonged or severe symptoms Invasive: gradual onset, bloody, systemic symptoms Tx: IVF, ± ciprofloxacin UNLESS pediatric or elderly patients with possible <i>E. coli</i> O157:H7 (can increase risk of HUS)
Diagnosis: watery diarrhea in the evening after eating eggs/mayonnaise at a picnic that day	Staph. aureus; toxin-mediated with rapid onset
Diagnosis: watery diarrhea after eating reheated rice	Bacillus cereus; toxin-mediated with rapid onset
Diagnosis, Treatment: diarrhea + flatulence + recent hiking and drinking from a freshwater stream	Dx: <i>Giardia lamblia</i> . Parasitic infection. Test for it with a stool antigen, not ova and parasite. Tx: Metronidazole. People who are risk: hikers, children at daycare, and oral-anal sexual conduct.
Diagnosis, Treatment: watery diarrhea + travel with unfiltered drinking water	Dx: Enterotoxigenic E. coli (toxin, ETEC) Tx: if no blood in diarrhea, give one dose of ciprofloxacin 750 mg. If patient traveled to southeast Asia, it is likely <i>Campylobacter</i> , so give 1,000 mg of azithromycin.
Diagnosis: watery diarrhea + meat/poultry	Dx: <i>Campylobacter</i> or <i>Clostridium perfringens</i> (toxin-mediated)

Diagnosis, Treatment: watery diarrhea + dark meat fish + rash/flushing/itching	Dx: Scombroid (histamine fish toxicity). Dark fleshed, peppery tasting fish such as tuna, mahi-mahi, and mackerel. Excess histidine on fish is broken down by bacteria to histamine which causes anxiety, flushing, headache, palpitations, and vomiting. Tx: antihistamines
Diagnosis: watery diarrhea + carnivorous fish + neurologic symptoms	Dx: Ciguatera. Toxin causes neurologic symptoms such as hot cold sensory reversal, paresthesias, nerve palsies, ataxia, and vertigo.
Diagnosis: bloody diarrhea + undercooked eggs/chicken + relative bradycardia	Dx: Salmonella typhi (invasive), cafeteria outbreaks, classically with high fever and relative bradycardia; can cause osteomyelitis in sickle cell patients.
Diagnosis: bloody diarrhea (severe) + high fever + institutionalized	Dx: Shigella (invasive)
Diagnosis: bloody diarrhea + fever + seizure in pediatric patient	Shigella
Diagnosis, Treatment: bloody diarrhea followed by ascending weakness	Dx: Campylobacter (invasive) associated with appendicitis mimic and can cause Guillain-Barré Tx: Azithromycin or erythromycin (resistance to ciprofloxacin)
Diagnosis: bloody diarrhea + RLQ pain + farm animal exposure	Dx: Yersinia (invasive); pseudoappendicitis (appendicitis mimic), can cause terminal ileitis
Diagnosis, Treatment: bloody diarrhea + undercooked seafood + alcoholic who gets very sick	Dx: Vibrio parahaemolyticus (invasive) Tx: ciprofloxacin or doxycycline
Diagnosis: bloody diarrhea + poorly cooked ground beef/raw milk	Dx: E. coli O157:H7 , associated with TTP (adults) and HUS (kids); NO ANTIBIOTICS
Diagnosis, Treatment: rice-water stools + contaminated water	Dx: Vibrio cholerae (toxin). Tx: oral rehydration tablets.
Diagnosis, Treatment: profuse diarrhea after recent antibiotics	Dx: Clostridium difficile (invasive) Tx: PO vancomycin is first line, fidaxomicin
Diagnosis: diarrhea + AKI ± low platelets	Dx: E. coli O157:H7 causing TTP/HUS
Diagnosis: food-borne illness associated with dairy that can lead to premature delivery in pregnant patients	Dx: Listeria monocytogenes
What is the most common cause of bacterial diarrhea in the US ?	Salmonella ; second most common is <i>Campylobacter</i> .
What factors of a patient history prompt you to obtain stools studies for patients with diarrhea ?	<ol style="list-style-type: none"> 1. Fever > 38.5C 2. Symptoms for greater than one week 3. Concerns for <i>C. difficile</i> 4. Immunocompromised or advanced age 5. Underlying IBD

<p>Signs and Symptoms, Treatment: Botulism</p>	<p>Paralytic illness caused by <i>Clostridium botulinum</i>, a neurotoxin that blocks acetylcholine release at the neuromuscular junction causing 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 (most common finding), parasympathetic blockade (dry mouth/sore throat, urinary retention) Tx: supportive care, respiratory monitoring (respiratory failure is most common cause of death), antitoxin (infants: BabyBIG; age > 1 year: antitoxin; antibiotics (for wounds))</p>
<p>Leading infectious cause of infertility?</p>	<p>Chlamydia trachomatis</p>
<p>Diagnosis, Treatment: painless vesicular lesions or ulcers to groin + buboes (huge tender lymphnodes)</p>	<p>Dx: Lymphogranuloma venereum (LGV). Caused by <i>Chlamydia trachomatis</i>. Tx: Doxycycline or Azithromycin (and treat partners), drain abscesses</p>
<p>Diagnosis, Treatment: painful ulcer with irregular borders to groin + buboes (huge LNs)</p>	<p>Dx: Chancroid. Caused by <i>Haemophilus ducreyi</i>. Looks like syphilis but the lesion is painful. Tx: ceftriaxone, azithromycin or ciprofloxacin, drain abscesses</p>
<p>Diagnosis, Treatment: neonate with copious purulent discharge from eyes</p>	<p>Dx: Neisseria gonorrhoeae conjunctivitis Tx: IV cefotaxime</p>
<p>Review the timing of the various causes of neonatal conjunctivitis</p>	<p>Chemical: first 24 hours Gonococcal: first 2-5 days Chlamydial: five days to two weeks</p>
<p>Treatment: neonatal chlamydial conjunctivitis</p>	<p>Tx: systemic treatment with oral azithromycin or erythromycin (both increase risk for pyloric stenosis); Patients must be admitted to evaluate for pneumonia as well.</p>
<p>Diagnosis, Treatment: "gunmetal grey" pustules to hands/skin, septic arthritis ± tenosynovitis</p>	<p>Dx: disseminated gonococcus; arthritis-dermatitis syndrome; gram-negative intracellular diplococci Tx: IV ceftriaxone</p>
<p>Diagnosis, Treatment: contact with armadillos, red patches of skin, paresthesias</p>	<p>Dx: Leprosy, caused by <i>Mycobacterium leprae</i> Tx: dapsone + rifampin (+ clofazimine for lepromatous disease)</p>
<p>Diagnosis, Treatment: contact with prairie dogs, eschar, buboes, sepsis</p>	<p>Dx: bubonic plague, caused by <i>Yersinia pestis</i> Tx: gentamicin, ciprofloxacin, or doxycycline.</p>
<p>Most common cause of viral pneumonia in adults?</p>	<p>Influenza</p>
<p>Diagnosis, Treatment: HIV + lung disease + pancytopenia</p>	<p>Dx: Mycobacterium avium intracellulare (MAI). Highest risk with CD4 count < 50. Tx: rifampin + ethambutol + azithromycin</p>

<p>Diagnosis, Treatment: primary tuberculosis</p>	<p>Dx: <i>Mycobacterium tuberculosis</i>. Transmission via inhalation of droplets. Often asymptomatic. Can progress to latent or reactivation TB. Tx: isoniazid (INH) + pyridoxine (Vitamin B6) for nine months</p>
<p>Most common chest x-ray finding in primary TB?</p>	<p>Most common overall: a single lobar infiltrate associated with hilar adenopathy Children: pronounced hilar adenopathy Elderly: isolated pleural effusion Ghon complex: calcified lung lesion, ± calcified lymph nodes [a.k.a. Ranke complex] representing HEALED infection Immunocompromised patients often cannot form a Ghon complex</p>
<p>Signs and Symptoms, Diagnosis, Treatment: reactivation TB</p>	<p>SSx: often occurs if immunocompromised, other stressor; cough, fever, night sweats, weight loss, hemoptysis Dx: XR with <u>upper lobe</u> apical lesions ± cavitation; 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</p>
<p>Immunocompetent person undergoes a high risk exposure to TB, what should you do?</p>	<ol style="list-style-type: none"> 1. Test them now with PPD skin test or interferon gamma and test them again in three months for conversion. 2. If either of these tests are positive obtain a CXR and call an ID specialist.
<p>What are the common side effects of TB treatment with RIPE?</p>	<p>Rifampin: orange body fluids, hepatotoxicity Isoniazid: neuropathy, hepatotoxicity, seizures (in overdose, treat with vitamin B6) Pyrazinamide: hepatotoxicity, gout, teratogenic (pregnancy) Ethambutol: optic neuritis (red-green color blindness)</p>
<p>What is the most common treatment regimen in pregnant women with active TB?</p>	<p>Rifampin, isoniazid (+B6), and ethambutol. No pyrazinamide because it is teratogenic.</p>
<p>Signs and Symptoms, Diagnosis, Treatment: rapidly progressive skin infection, unusually high heart rate, and indifferent patient</p>	<p>Gas gangrene/myonecrosis. Clostridial myonecrosis (usually <i>C. perfringens</i>). SSx: similar presentation to necrotizing fasciitis, tachycardia out of proportion to fever, la belle indifference Dx: subcutaneous/intramuscular gas, incision with foul-smelling "dishwater" fluid and dead muscle Tx: antibiotics (vancomycin + zosyn + clindamycin) + wide surgical debridement (don't delay antibiotics). Clindamycin crucial to reduce toxin formation.</p>

<p>Review the definitions of SIRS, sepsis, severe sepsis, and septic shock</p>	<p>SIRS: At least two of: (1) Temperature < 36.0 (96.8) or > 38.0 (100.4), (2) HR > 90, (3) RR > 20, (4) WBC < 4k or > 12k or > 10% bands Sepsis: SIRS + source of infection Severe Sepsis: sepsis + end organ damage Septic Shock: sepsis + <u>refractory hypotension</u> *Note: ITE/boards still test these concepts</p>
<p>Review the key components of Early Goal-Directed Therapy for sepsis.</p>	<p>Early IVF (30 mL/kg crystalloid), early empiric antibiotics, MAP > 65 (IVF or pressors), SvO2 > 70%, CVP 8-12, transfuse pRBCs if hematocrit < 30% Note: strict adherence to this regimen has been debunked by several recent trials, but the necessity of adequate fluid resuscitation (30 mL/kg) and early antibiotics remains well-supported.</p>
<p>Give examples of empiric antibiotic regimens for sepsis by suspected infectious source.</p>	<p>CAP: ceftriaxone + azithromycin HAP: vancomycin + zosyn Urinary: ceftriaxone (if not culture-guided) Intra-abdominal: ceftriaxone/ciprofloxacin + metronidazole Biliary: zosyn Device related: vancomycin + gentamicin Skin/Soft tissue: vancomycin</p>
<p>Diagnosis, Treatment: young woman with high fever + rash + shock and organ failure</p>	<p>Toxic Shock Syndrome. Causes: tampon, surgical or nasal packing or other foreign body; bacterial superantigen. Staph (TSS): more common; erythematous rash with desquamation, hypotension, fever, and associated with foreign body Strep (STSS): fever, but less rash, often with existing wound Tx: remove foreign bodies FIRST, supportive care, and antibiotics (clindamycin first to combat toxin, then empiric broad-spectrum for sepsis coverage), IVIG for refractory cases</p>
<p>Signs and Symptoms, Diagnosis, Treatment: primary syphilis</p>	<p>Treponema pallidum (spirochete) SSx: painless genital ulcer (<u>chancre</u>), regional lymphadenopathy Dx: VDRL/RPR are nonspecific and often negative at this stage Tx: penicillin G benzathine 2.4 million U IM x1</p>
<p>Signs and Symptoms, Diagnosis, Treatment: secondary syphilis</p>	<p>Onset 5-8 weeks after primary syphilis. SSx: rash (papulovesicular) trunk → palms/soles, condyloma lata Dx: VDRL or RPR, confirm with FTA-ABS Tx: penicillin G benzathine 2.4 million U IM x1 (if late disease three weekly doses)</p>

<p>Signs and Symptoms, Diagnosis, Treatment: tertiary syphilis</p>	<p>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 penicillin every 4 hours for two weeks</p>
<p>Diagnosis, Treatment: worsened rash and toxicity shortly after treatment of syphilis</p>	<p>Jarisch-Herxheimer reaction due to endotoxin release from dying spirochetes. Tx: Supportive care</p>
<p>Care plan if patient with syphilis is allergic to penicillin?</p>	<p>Preferred: admit for desensitization, they need penicillin. Alternative: doxycycline for early stages and ceftriaxone for neurosyphilis.</p>
<p>Pathophysiology, Signs and Symptoms, Treatment: tetanus</p>	<p><i>Clostridium tetani</i> spores which enter into wounds. Sources: dust, soil, feces Pathophysiology: 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 (benzodiazepines, opioids, ± paralytics), wound care, antibiotics (metronidazole > penicillin), Tdap vaccine (prevention) + tetanus IG (unimmunized + high risk wounds)</p>
<p>Review indications for tetanus prophylaxis</p>	<p><u>≥ 3 vaccine doses + low risk wound:</u> dT if > 10 years since last dose <u>≥3 vaccine doses + high risk wound:</u> dT if > 5 years 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: > 6 hours old, contaminated (dirt, saliva, feces), puncture/crush/avulsion wounds, foreign body, frostbite</p>
<p>Diagnosis, Treatment: red rash to diaper area with satellite lesions</p>	<p>Dx: <i>Candida</i> Tx: topical antifungals (also occurs in moist areas/skin folds especially in patients with diabetes)</p>
<p>Diagnosis, Treatment: immunocompromised + odynophagia/dysphagia</p>	<p>Dx: <i>Candida esophagitis</i> Tx: oral fluconazole, low threshold for IV for those who can't tolerate PO *Nystatin swish and spit does not cover</p>
<p>Diagnosis, Treatment: indwelling catheter + yeast on blood cultures</p>	<p>Dx: <i>Candida fungemia</i> Tx: amphotericin B</p>
<p>Common organisms implicated in diarrhea in patients with AIDS?</p>	<p><i>Cryptosporidium, Isospora, CMV, M. avium</i>; often unclear cause</p>
<p>Diagnosis, Treatment: immunocompromised + painless brown/black skin lesions</p>	<p>Dx: Kaposi sarcoma (classically on face, chest, oral cavity), AIDS-defining illness caused by HHV-8 Tx: HAART, chemo, radiation; steroids can worsen disease</p>

<p>Diagnosis, Treatment: AIDS + white plaque on oropharynx</p>	<p>Dx: <u>Candida/Thrush:</u> plaques scrape off. <u>Oral Hairy Leukoplakia:</u> lateral tongue, can't scrape off, caused by EBV, very specific for HIV, NOT precancerous Tx: clotrimazole or nystatin for candida; supportive care for oral hairy leukoplakia</p>
<p>Signs and Symptoms, Diagnosis, Treatment: meningitis and focal neurologic findings in patient with AIDS</p>	<p><i>Cryptococcus neoformans</i> (encapsulated yeast in soil with pigeon poop), most common cause of meningitis in patients with AIDS SSx: headache, neck pain, fever, altered mental status or cranial nerve abnormalities Dx: CSF cryptococcal antigen, LP with high opening pressure, + india ink stain Tx: amphotericin B + flucytosine</p>
<p>Signs and Symptoms, Treatment: histoplasmosis</p>	<p>Dimorphic fungus. Found in spelunkers, caves, bird/bat droppings; can cause epidemics if soil upturned. Endemic to <u>Ohio and Mississippi River valleys.</u> SSx: flu-like symptoms, disseminated disease or chronic progressive pulmonary disease (diffuse infiltrates and calcified nodes). Tx: itraconazole, amphotericin B.</p>
<p>Diagnosis, Treatment: immunocompromised + encephalitis + ring-enhancing lesions on CT</p>	<p>Dx: <i>Toxoplasmosis gondii</i> (protozoan); associated with cat feces; bad for fetus if infection occurs during pregnancy (TORCH) Tx: pyrimethamine, sulfadiazine</p>
<p>Signs and Symptoms, Diagnosis: travel + cyclical fever + splenomegaly</p>	<p>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 disease: profound hypoglycemia, hemolytic anemia, seizures, coma Dx: thick + thin blood smears (ring forms) ± Giemsa or Wright stain</p>
<p>Treatment: malaria</p>	<p><u>Uncomplicated + chloroquine-sensitive</u> (Central America, Caribbean): chloroquine <u>Uncomplicated + chloroquine-resistant</u> (South America, South Asia, Africa): quinine (hypoglycemia) + doxycycline <u>Complicated or <i>P. Falciparum</i></u>: artesunate and quinidine IV (QT prolongation)</p>
<p>What is the most dangerous/severe strain that causes malaria? Treatment?</p>	<p><i>P. falciparum</i>: cerebral malaria, "blackwater" fever, death Tx: artesunate, IV quinidine</p>
<p>Signs and Symptoms, Diagnosis, Treatment: traveler + myalgias and fever. Worse symptoms on second infection</p>	<p>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 Tx: supportive</p>

What is the cause, vector, and treatment of Lyme disease?	Cause/vecotr: <i>Ixodes tick (deer tick)</i> carrying <i>Borrelia burgdorferi</i> , primarily in northeast US & Wisconsin; tick bite history is often absent Tx: doxycycline (<u>adults and children</u>), CNS/cardiac involvement: IV ceftriaxone. If pregnant: amoxicillin
What are the typical stages of Lyme disease? Treatment?	Stage 1: 1 week; erythema migrans ("bull's eye") rash Stage 2: days to weeks; neurologic changes (meningoencephalitis is the most common neurologic finding with cranial nerve palsies - bilateral Bell's palsy), cardiac changes (variable AV block is most common cardiac finding) Stage 3: months to years; arthritis, neurologic symptoms Tx: doxycycline for everything other than severe carditis or neurological manifestations - use IV ceftriaxone
Describe the criteria and medications for prophylactic treatment of Lyme disease	Criteria: tick attached for ≥ 36 hours, prophylaxis started within 72 hours of tick removal, engorged tick at removal Tx: single dose doxycycline
Signs and Symptoms, Diagnosis, Treatment: 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 (most common symptom), centripetal (towards trunk) rash (palms + soles), calf tenderness Dx: low platelets, hyponatremia Tx: ALWAYS doxycycline (even children)
Signs and Symptoms, Diagnosis, Treatment: ehrlichiosis	<i>Ehrlichia</i> spp. with tick vector. South central & south Atlantic US SSx: <u>abrupt onset fevers/chills/myalgias/rigors</u> (differentiates from other tick-borne illnesses), conjunctival injection \pm rash Dx: blood smear with intracellular parasites (poor sensitivity), PCR, <u>leukopenia, thrombocytopenia, and elevated LFTs</u> Tx: doxycycline; rifampin if pregnant
Diagnosis, Treatment: fever, malaise, exudative pharyngitis, posterior lymphadenopathy, \pm splenomegaly	Dx: Infectious mononucleosis. Epstein-Barr virus (EBV); Tx: supportive; warn against contact sports for 1-2 months due to risk of splenic rupture
Diagnosis: mononucleosis + amoxicillin for presumed strep	90% develop maculopapular rash (NOT an allergy)
What are associated lab abnormalities found in patients with mononucleosis?	Atypical lymphocytes , + heterophile antibodies (monospot test), hemolytic anemia, thrombocytopenia, elevated LFTs, false positive RPR or VDRL
What is the difference between antigenic drift and antigenic shift?	Antigenic drift: minor mutation Antigenic shift: major mutation Often used in context of influenza (orthomyxovirus) and HA/NA surface antigens
Who is at high risk for death with influenza and what is the most common cause of death?	Extremes of age and pregnant women are highest risk; most common cause of death is secondary bacterial pneumonia.

Describe the indications for and side effects of neuraminidase inhibitors when used for influenza .	Can reduce symptom duration by ~24 hours if within 48 hours of symptom onset OR hospitalized. Oseltamivir/Tamiflu causes GI symptoms, and Zanamivir/Relenza causes bronchospasm/wheezing.
Diagnosis, Signs and Symptoms, Treatment: exposure to rat feces, ARDS	Dx: hantavirus. Transmitted via aerosolized rodent excretions. SSx: hantavirus pulmonary syndrome - ARDS, thrombocytopenia. Starts with a flu-like syndrome and is then rapidly progressive (and therefore many patients are discharged and return critically ill). Tx: 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 and treated?	HSV-1: mouth, stomatitis, keratitis (possible corneal ulcer), vesicles on digits (Whitlow) HSV-2: anus, genital, & neonatal (C-section if pregnant and in labor) Dx: Tzanck smear (multinucleated giant cells), viral culture Tx: acyclovir or valacyclovir
Compare the presentation and treatment of chickenpox vs. shingles (Varicella Zoster Virus)	Primary varicella (chickenpox): highly contagious, incubation ~ 2 weeks SSx: crops of vesicles in <u>various stages of healing</u> . Rash: "dew drop rash on a rose petal", starts on hairline → chest, palms/soles (+ mucous membranes) Tx: <u>healthy/age <12:</u> supportive, monitor for bacterial superinfection; <u>immunocompromised, age >12:</u> acyclovir Shingles: reactivation of dormant VZV SSx: prodrome (itching, burning), rash- painful vesicular eruption (usually unilaterally in a single dermatome) Tx: acyclovir only started if rash present for < 72 hours, steroids (controversial), pain control. Complication: post-herpetic neuralgia
Diagnosis: Bell's palsy and vesicle on ear	Ramsay-Hunt syndrome/Zoster Oticus (VZV of CN VIII)
Diagnosis: vesicle on tip of nose	Hutchinson's sign, V1 zoster, predicts corneal involvement/ulceration (Zoster Ophthalmicus)
Treatment: herpes zoster ophthalmicus	Tx: oral acyclovir or trifluridine drops. This happens when the nasociliary branch of CN V is infected with VZV. Emergent ophthalmology follow up is needed.
Treatment: pregnant or immunocompromised patient after exposure to chicken pox	Send titers to check for immunity; if negative give varicella zoster IG.
Diagnosis, Treatment: pregnant woman presents with dyspnea, infiltrate on CXR, and widespread itchy vesicular rash in various stages of development.	Dx: varicella pneumonia Tx: admit and treat with IV acyclovir and CAP coverage. VZIG IS NOT ENOUGH treatment.
What is the definition of disseminated zoster?	Involvement of three or more dermatomes.

What domestic and wild animals are high risk for rabies transmission?	Domestic animals: cats > dogs (dogs in developing countries are HIGH risk) Wild animals: (account for ~90% cases in US): bats (most common cause in US) > raccoons > skunks > foxes > coyotes, mongooses; NOT rabbits or rodents
What are symptoms of rabies infection ?	Incubation 3-7 weeks, 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 Need Ig and full vaccine series for close proximity with a bat and exposure/bite cannot be ruled out (e.g. awakening to find bat in room, unattended children)
What defines AIDS ?	HIV with CD4 < 200 OR AIDS defining illness such as esophageal candidiasis, cryptococcus, CMV, Kaposi, PJP, toxoplasmosis, TB (in an non-endemic area)
Signs and Symptoms: acute HIV infection?	Acute Retroviral Syndrome. Often missed, occurs 2-4 weeks post-exposure SSx: non-specific viral syndrome (fever, rash, lymphadenopathy, myalgias). Most infectious stage of HIV (high viral load + shedding), but antibody testing will be negative as seroconversion takes 3-12 weeks post-exposure
How is HIV diagnosed?	ELISA: to screen (sensitive), delayed (+) weeks to months HIV-1/2 immunoassay & Western blot: to confirm (sensitive + specific), blood test
Which opportunistic infections are more likely below the following CD4 counts: < 500, < 200, < 100, < 50?	< 500: TB, HSV, VZV, Kaposi's sarcoma < 200: PJP, HIV encephalopathy, candidiasis, PML < 100: toxoplasmosis, histoplasmosis, cryptococcus < 50: CMV (GI, pulm, retina), MAC avium, CNS lymphoma *NOTE: HIV patients get all usual infections as well, but have increased risk of opportunistic as CD4 count 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 72 hours. However, the earlier the better.
Signs and Symptoms, Diagnosis, Treatment: 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). Elevated LDH. BAL for diagnosis Tx: TMP-SMX, steroids (indications: children, PaO2 < 70 mm Hg [~SaO2 ~93%], A-a gradient > 35)
What are possible side effects of pentamidine (treatment for PJP pneumonia)?	Hypoglycemia, hypotension, pneumothorax

What are the appropriate outpatient treatments for PJP 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.
What are the CT findings on non-contrast and contrasted CT head in toxoplasmosis ? Treatment ?	Non-contrast CT : multiple subcortical lesions in basal ganglia Contrast CT : ring-enhancing lesions with surrounding edema Tx : pyrimethamine, sulfadiazine and leucovorin
Diagnosis : HIV + CD4 < 200, focal neurologic deficits with nonenhancing white matter lesions	Dx : PML (JC virus) - if ring-enhancing on CT, think toxoplasmosis or primary CNS lymphoma
Diagnosis, Treatment : progressive blindness in AIDS patient	Dx : CMV retinitis . Exam shows "fluffy white perivascular lesions (cotton wool spots) with areas of hemorrhage." Tx : IV ganciclovir
Which 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 < 72 hours: HAART for 28 days; if low risk and > 72 hours no treatment is necessary All others per clinical judgement.
What are potential oral antibiotic options for community acquired MRSA ?	Clindamycin, TMP-SMX, doxycycline, or linezolid. IV vancomycin required if hospital-acquired.
Diagnosis : skin lesion, gram positive rod	Anthrax
Diagnosis, Treatment : cutaneous vs. pulmonary anthrax	<i>B. anthracis</i> (Gram positive rod) Cutaneous : pruritic, black eschar + painful lymphadenopathy over 1-2 weeks Pulmonary : due to inhaled spores (not contagious), flu-like sx. CXR shows wide mediastinum ; rapid progression to sepsis + death Tx : ciprofloxacin
Diagnosis, Treatment : pneumonic and bubonic plague	Dx : <i>Yersinia pestis</i> Pneumonic : inhaled aerosolized rat droppings, very contagious, severe pneumonia, bioterrorism agent Bubonic : transmitted via flea bite, causes buboes + acral necrosis (black/dead distal extremities), may travel to lungs (contagious at this stage) Tx : streptomycin, gentamicin, doxycycline.
What is the appropriate management of a patient with a tick bite, targetoid rash, and Bell's palsy ?	CT and LP followed by ceftriaxone due to concern for disseminated Lyme
What is the most infectious blood-borne pathogen ?	Hepatitis B, followed by Hepatitis C and HIV

Diagnosis, Treatment, Complications: military recruit or college student with fever, headache, petechial rash	Dx: Meningococemia. <i>Neisseria meningitidis</i> (aerobic, gram-negative diplococcus) Tx: ceftriaxone + vancomycin Complications: myocarditis with CHF or conduction abnormalities; <u>Waterhouse-Friderichsen syndrome</u> : bilateral adrenal hemorrhage + meningococemia
Signs and Symptoms, Diagnosis, Treatment: relative bradycardia in the setting of fever after travel to southeast Asia when patient presents with fever, malaise, relative anemia, and elevated LFTs.	Dx: typhoid fever; caused by salmonella typhi. Oral ingestion of contaminated food or water. SSx: fever, malaise, abdominal pain, fatigue, diarrhea in children, constipation in adults Tx: fluoroquinolones in adults, third-generation cephalosporin in children. Vaccine only gives 55% immunity.
Which infections are most common with puncture wounds to the plantar surface of the foot, either barefoot or through a shoe?	Barefoot: lower risk for infection, if infection occurs typically staph or strep Through shoe: pseudomonas
What antibiotics can be used as chemoprophylaxis against meningococcal disease?	Rifampin first line, other medications include ciprofloxacin, ceftriaxone
Signs and Symptoms, Treatment: leptospirosis	Comes from contaminated fresh water (urine of rodents/livestock) SSx: fevers, rigors, myalgia, CONJUNCTIVAL SUFFUSION (redness without exudates), jaundice, acute renal failure Tx: doxycycline or penicillin G (may precipitate Jarish-Herxheimer reaction)
Treatment of which infections can be associated with the Jarisch-Herxheimer Reaction ?	Syphilis, Leptospirosis, Lyme Disease
Signs and Symptoms, Treatment: tularemia	History will discuss handling rabbits . Bioterrorism agent. SSx: skin ulcers, lymphadenopathy, fever. Tx: streptomycin. Alternative: ciprofloxacin, doxycycline.
Most common infection in a patient with an indwelling urinary catheter	Pseudomonas ; human + plastic (ETT/foley/tracheostomy) = <i>Pseudomonas</i>
Diagnosis: AIDS + chronic watery diarrhea	Cryptosporidium
Diagnosis: AIDS (CD4 < 200) + pneumonia	PJP or TB (may have negative CXR/PPD)
Diagnosis: AIDS + ring-enhancing intracranial lesions + focal neurologic deficits	Toxoplasma gondii
Diagnosis: AIDS + ring-enhancing intracranial lesions + altered mental status	Primary CNS lymphoma
Diagnosis: AIDS (CD4 < 100) + meningitis	Cryptococcus
Diagnosis: AIDS (CD4 < 50) + focal neurologic deficits + nonenhancing white matter lesions	PML (JC virus)
Diagnosis: AIDS (CD4 < 50) + retinitis + cotton-wool spots	CMV
Diagnosis: AIDS + dark purple skin/mouth nodules	Kaposi's sarcoma
Diagnosis: AIDS + vesicular rash	HSV, zoster reactivation

HIV medication side effect: didanosine	Pancreatitis
HIV medication side effect: efavirenz	Vivid dreams, headache, severe rash (rarely SJS), dizziness
HIV medication side effect: indinavir	Nephrolithiasis (radiolucent), hyperbilirubinemia, hepatitis
HIV medication side effect: lopinavir	Nausea, vomiting, diarrhea, hepatitis
HIV medication side effect: nucleoside/nucleotide reverse transcriptase inhibitors	Lactic acidosis
HIV medication side effect: ritonavir	Paresthesias
HIV medication side effect: zidovudine	Bone marrow suppression

Neurology

Bizz	Buzz
Diagnosis: Eye down and out	CN III palsy; consider CVA, PCOM aneurysm, uncal herniation if with blown pupil
Diagnosis: Bilateral internuclear ophthalmoplegia	Combined 3rd & 6th nerve palsy; usually multiple sclerosis
Diagnosis, Treatment: Urinary incontinence, altered mental status, ataxia	Dx: Normal pressure hydrocephalus; "wet, wacky, wobbly"; will have normal opening pressure on LP Tx: high volume CSF removal vs shunt
Diagnosis: Young obese woman, headaches, vision changes, CN VI palsy	Dx: Idiopathic intracranial hypertension (pseudotumor cerebri); CT normal (may show empty sella); LP (↑ opening pressure) Tx: LP (drain to <20 cm H2O), acetazolamide Complication: permanent vision loss
Mechanism, Signs & Symptoms, Treatment: Neuroleptic Malignant Syndrome	Mechanism: Anti-dopaminergic/ antipsychotic use or dopamine withdrawal SSx: "FEVER"; fever , encephalopathy, vital instability, elevated CK, rigidity , ***no clonus/DTR changes*** Tx: Supportive (IVF, benzodiazepines, cooling), ± dantrolene (direct skeletal-muscle relaxant), ± bromocriptine (dopamine agonist)
Mechanism, Signs & Symptoms, Treatment: Serotonin Syndrome	Mechanism: Serotonergic agent use (combo 2+ SSRIs) or multi-drug overdose SSx: Fever , autonomic instability, encephalopathy, clonus + hyperreflexia (LE > UE) Tx: Supportive (IVF, benzodiazepines, cooling), ± cyproheptadine (antihistamine w/ antiserotonergic properties); withdraw offending agent
Diagnosis: CNS mass lesion in AIDS	1) Toxoplasmosis (multiple ring-enhancing lesions w/ edema) 2) CNS lymphoma (hyperdense, round enhancing lesions) **Know that these are both differentials but treat as toxo on the boards.
Treatment: Intraparenchymal hemorrhage	"BRAINS": BP control (~160/90), Reverse coagulopathy , Airway mgmt , ICP control if herniating (hyperventilate, elevate HOB 30°, mannitol), Neurosurgery consult (craniotomy for cerebellar vs. ventriculostomy), Seizure ppx (controversial)
Diagnosis: Contralateral hemiparesis/hemiplegia, contralateral sensory loss, homonymous hemianopia	Putamen hemorrhage (most common type of ICH)
Diagnosis: Ataxia, headache/vomiting, gaze palsy, facial weakness	Cerebellar hemorrhage
Diagnosis: Hemiparesis or hemisensory loss, upward gaze palsy, miotic pupils	Thalamic hemorrhage
Diagnosis: Deep coma, total paralysis, pinpoint pupils	Pontine hemorrhage

Presentation: ACA vs. MCA vs. PCA stroke	ACA: frontal lobe dysfunction, apraxia, contralateral paralysis (LE > UE) MCA: contralateral paralysis (upper > lower), ipsilateral hemianopsia, aphasia PCA: LOC, nausea/vomiting, CN dysfunction, ataxia, vertigo, visual agnosia
Diagnosis: Unilateral CN deficits + contralateral hemiparesis or hemisensory loss	Brainstem stroke (this presentation is known as “crossed signs”)
Treatment: Sickle cell patient with CVA	Exchange transfusion
Indications for tPA in CVA	Age ≥18, dx of ischemic stroke + neuro deficits, symptoms < 4.5 hr , CTH negative for bleed, no clear reversible cause.
Absolute contraindications to tPA in CVA	Stroke/neurosurgery/head trauma within 3 mo, ANY ICH (current or previous), known intracranial neoplasm/AVM/aneurysm, BP >185/110 after reduction attempted, reversible cause, active bleeding or coagulopathy (Plt <100k, INR >1.7, PT>15 seconds), multilobar infarcts, glucose < 50
Relative contraindications to tPA in CVA	Pregnancy, seizure at onset with postictal period, major surgery in last 14 days, GI or GU bleeding in last 21 days, MI in last 3 mo, endocarditis (tested often)
Dosing: tPA/alteplase vs TNK/tenecteplase in ischemic stroke	tPA: 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 TNK: 0.25 mg/kg or 0.50 mg/kg with a maximum dose of 50 mg given as a bolus dose without follow-up infusion
What is the major difference between lacunar and cortical infarcts?	Cortical: Large artery, cortical dysfunction (aphasia, neglect, ALOC), motor AND sensory sx, deficits to contralateral side Lacunar: Small artery, pure motor OR sensory sx, related to HTN
Localize the lesion: Neglect or “hemi-inattention”	Non-dominant parietal lobe (right hemisphere for most people)
Diagnosis, Treatment: Transient episode of slurred speech and unilateral arm weakness, now resolved	Dx: Transient ischemic attack (TIA) = episode of neurological ischemia without infarction Tx: Aspirin ± clopidogrel (without treatment 10% of TIA patients will go on to have a stroke within 90 days)
What is the further workup and management is needed for a patient after transient ischemic attack?	Vessel imaging (MRI/MRA head & neck), ECG, echocardiogram Start dual anti-platelet therapy (DAPT) for ABCD2 Score >3 (vs ASA alone)

How do you calculate an ABCD2 Score and how is it used?	Used to determine disposition for patients with TIA; predicts likelihood of subsequent stroke within 2 days. Age >60 (1), BP >140/90 (1), Clinical features : unilateral focal weakness (2) speech disturbance w/o weakness (1), Duration of symptoms : >60 min (2), 10-59 min (1), <10 (0), Diabetes (1) Hospitalize for score >2 (vs expedited outpatient workup)
What is the best study to diagnose venous sinus thrombosis?	Magnetic resonance venography (MRV)
Treatment : Suspected bacterial meningitis	ABCs, Antibiotics: CTX + Vancomycin ± Ampicillin (age >50, alcoholics) ± Acyclovir , steroids, supportive (MAP/temperature mgmt, sz control PRN)
Diagnosis : Suspected bacterial meningitis	Obtain CT head first, then perform lumbar puncture (send CSF studies) Do NOT delay antibiotics for diagnosis/lumbar puncture
Diagnosis, Treatment : Bloody, necrotizing encephalitis	Dx : Herpes encephalitis Tx : IV acyclovir
Physical exam findings : Upper motor neuron lesion	Spastic paralysis, ↑ DTRs, positive (upgoing) Babinski, ↑ tone
Physical exam findings : Lower motor neuron lesion	Flaccid paralysis, ↓ DTRs, muscle wasting, fasciculations/twitching
What distinguishes Conus Medullaris Syndrome (CMS) from Cauda Equina Syndrome (CES)?	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).
Signs & Symptoms, Diagnosis, Treatment : Guillain-Barré Syndrome (GBS)	SSx : preceding respiratory (mycoplasma) or GI illness (campylobacter = MCC), progressive, ascending, symmetric motor weakness , ↓ DTRs , normal rectal tone GBS = most common peripheral neuropathy caused by autoimmune demyelination Dx : LP shows ↑ CSF protein + mild pleocytosis Tx : plasmapheresis, IVIG, measure NIF (intubate if worse than -30 cm H2O), vital capacity (respiratory monitoring)
What is the Miller Fisher variant of Guillain-Barré Syndrome?	Descending motor weakness (whereas GBS is ascending) + ataxia + ophthalmoplegia and bulbar symptoms May present similarly to botulism.
What is the difference between Guillain-Barré Syndrome and tick paralysis ?	GBS : ascending motor weakness, post-infectious autoimmune disorder Tick paralysis : symmetric ascending flaccid paralysis + ataxia + fixed dilated pupils; caused by neurotoxin in tick saliva (Tx: remove tick)
Mechanism : Botulism	Clostridium botulinum neurotoxins → blocks ACh release at neuromuscular junction → flaccid paralysis Forms: food borne (canned foods, honey), wound, infantile (most common)

<p>Signs & Symptoms, Treatment: Botulism</p>	<p>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) Antidote: <1 year-old gets botulism Ig; otherwise antitoxin</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Syringomyelia</p>	<p>SSx: headache (2/2 Chiari malformation), "cape-like" loss of pain/temp sensation to UE, preserved proprioception/vibratory sense Cyst or cavity in upper spinal cord (cervical spine = MC location) Dx: MRI Tx: neurosurgery consultation, monitor vs. drain</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Myasthenia gravis</p>	<p>SSx: repeated muscle use = ↑ weakness/fatigue, proximal muscle weakness, ocular sx (CNIII palsy, ptosis, dysphagia, diplopia) Autoantibodies to nicotinic ACh receptors (25% with associated thymoma) Dx: Tensilon Test (edrophonium) or Ice Pack test: ↓ sx Tx: Pyridostigmine Myasthenic crisis: triggered by infxn, meds; resp. failure → mechanical ventilation (follow vital capacity, NIF); treated with IVIG and/or plasmapheresis</p>
<p>What is the difference between Myasthenia Gravis (MG) and Lambert-Eaton Myasthenic Syndrome (LEMS)?</p>	<p>MG: fatigue with repeated movement LEMS: improvement repeated movement LEMS is often paraneoplastic, so look for underlying cancer if not already diagnosed.</p>
<p>Diagnosis, Treatment: Young man presents with lower extremity paralysis, hyperthyroidism, and hypokalemia</p>	<p>Dx: Thyrotoxic periodic paralysis Hallmark of periodic paralysis = painless weakness precipitated by heavy exercise, high carb meals Caused by transmembrane shift of K into cells Tx: K repletion (not aggressive) and beta blockers</p>
<p>Diagnosis, Treatment: Status epilepticus</p>	<p>Dx: Seizure lasting > 5 minutes or > 2 discrete seizures without return to baseline Common causes: AED discontinuation, medication noncompliance Tx: benzodiazepines (IV lorazepam, IM midazolam), phenytoin/levetiracetam/divalproex (second line), intubation + phenobarbital/propropofol (third line)</p>
<p>Treatment: Eclamptic seizure</p>	<p>Magnesium sulfate (4-6 g IV over 15 min followed by 2-3 g/hr) Side effects: loss of DTRs, dysrhythmia, resp. failure</p>
<p>Treatment: Respiratory depression due to hypermagnesemia</p>	<p>IV Calcium</p>
<p>Treatment: Seizure related to isoniazid overdose</p>	<p>Vitamin B6/Pyridoxine (1 gm per 1 gm of INH toxicity)</p>

<p>Physical exam findings: Meningismus</p>	<p><u>Jolt accentuation</u>: baseline HA ↑ when the patient turns head horizontally 2-3 rotations/sec <u>Brudzinski's sign</u> (Bend the brain): flexing the neck causes the hips and knees to flex <u>Kernig's sign</u> (extend knees): knees/hips flexed to 90 degrees, knee extension causes pain</p>
<p>What is appropriate chemoprophylaxis for those exposed to <i>N. meningitidis</i>? Who needs prophylaxis?</p>	<p>Ppx: Rifampin 600 mg BID x2d, Ceftriaxone 250 mg IM x1, OR Ciprofloxacin 500 mg PO x1 Indications: household contacts, sleeping in the same room, school or daycare contacts within past 7d, direct exposure to secretions (toothbrush, kissing, shared utensils), travel for >8 hours with the patient, intubation without facemask</p>
<p>Signs & Symptoms, Diagnosis, Treatment: HSV Encephalitis</p>	<p>SSx: fever + headache + AMS; neuro deficits, psych sx Dx: CSF with ↑ RBCs and no bacteria, usually focal at <u>temporal lobes</u> (may see hemorrhage on CT) Tx: IV acyclovir</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Neurocysticercosis</p>	<p>SSx: new onset sz in adult Pt will be immigrant from tropical areas, Mexico. Infxn acquired by eating pork containing larval cysts of Taenia solium (tapeworm). Dx: CT shows multiple ring-enhancing lesions Tx: Albendazole Complication: obstructive hydrocephalus</p>
<p>What is the most common cause of meningitis in an adult?</p>	<p><i>Streptococcus pneumoniae</i> (Most common cause of MOPS meningitis, otitis media, pneumonia, and sinusitis)</p>
<p>What are the expected cerebrospinal fluid findings for viral meningitis?</p>	<p>Opening pressure: normal WBCs: ↑ (< 300; lymphocyte predominance) Protein: normal to ↓ Glucose: normal Culture: negative</p>
<p>What are the expected cerebrospinal fluid findings for bacterial meningitis?</p>	<p>Opening pressure: ↑ WBCs: ↑↑ (>1000; neutrophil predominance) Protein: ↑ Glucose: ↓ Culture: positive</p>
<p>What are the expected cerebrospinal fluid findings for fungal meningitis?</p>	<p>Opening pressure: ↑ WBCs: ↑ (but < 500; lymphocyte predominance) Protein: ↑↑ Glucose: normal to slightly ↓ Culture: positive (fungal)</p>
<p>Treatment: Brain abscess</p>	<p>3rd generation cephalosporin (e.g. ceftriaxone or cefotaxime) + anaerobic bacterial coverage (e.g. metronidazole) + neurosurgical consultation</p>

How is caloric testing used? What direction would you expect nystagmus with normal caloric testing ?	Used to test vestibulo-ocular reflex; intact brainstem elicits nystagmus. Direction of fast beat of nystagmus depends on temperature of H2O used. "COWS": <u>C</u> old- <u>O</u> pposite, <u>W</u> arm- <u>S</u> ame
Signs & Symptoms, Diagnosis, Treatment: Posterior Reversible Encephalopathy Syndrome (PRES)	SSx: HTN emergency w/ neuro sx (severe headache, AMS, seizure, vision loss); often due to vasogenic edema of the brain Associated with preeclampsia/eclampsia . Most often in BILATERAL occipital and posterior parietal lobes Dx: mainly clinical; CT may show edema; MRI is more specific Tx: supportive, BP reduction (goal ~25% MAP reduction in ED)
Diagnosis: Diplopia on lateral gaze due to impaired adduction of the contralateral eye	Internuclear ophthalmoplegia (associated with MS)
Diagnosis: Diplopia on lateral gaze due to impaired adduction of the ipsilateral eye	CN VI Palsy (obtain brain imaging; neuro/ophtho consults)
What diagnoses typically present with ascending vs descending weakness ?	Ascending: GBS, tick paralysis Descending: Botulism, myasthenia gravis, Miller Fisher variant GBS, Lambert-Eaton myasthenic syndrome
What are potential causes of peripheral and central vertigo ?	Peripheral vertigo: BPPV, acute otitis media, labyrinthitis, Meniere's dz, vestibular neuronitis Central vertigo: brainstem or cerebellar lesion
What are the differences between peripheral and central vertigo ?	Peripheral vertigo: sudden onset, short duration, severe intensity (often with vomiting), worsened by position, fatigueable unilateral nystagmus, otherwise normal neuro exam . Central vertigo: often gradual, constant sx, less likely positional, nystagmus (multidirectional, non-fatigable), +additional neuro deficits
Diagnosis, Treatment: Woman with shock-like unilateral lower face pain, worse with chewing or brushing teeth	Dx: Trigeminal neuralgia Tx: Carbamazepine
Signs & Symptoms, Diagnosis, Treatment: Temporal arteritis (Giant Cell Arteritis)	SSx: monocular vision loss , unilateral headache, jaw claudication; usually woman >50 years old; associated with polymyalgia rheumatica Dx: temporal artery tenderness on exam, ↑ ESR and ↑ CRP , temporal artery biopsy (gold standard) Tx: high-dose IV steroids ASAP (don't wait or refer if visual sx)
What causes Bell's Palsy ?	CN VII palsy Causes: idiopathic , infection (HSV = MCC), malignancy

<p>Signs & Symptoms, Diagnosis, Treatment: Bell's Palsy</p>	<p>SSx: ipsilateral weakness of UPPER AND LOWER face, lip droop/drooling, hyperacusis, loss of taste sensation Dx: clinical (head imaging <u>not</u> necessary) Tx: prednisone, antivirals (determined by House-Brackmann score), artificial tears (complication: keratitis) *Bilateral palsy: consider Lyme, HIV, botulism, infectious mono*</p>
<p>Diagnosis, Treatment: Recurrent episodes of severe unilateral headaches w/ ipsilateral lacrimation, rhinorrhea, nasal congestion, and conjunctival injection</p>	<p>Dx: cluster headache, often men in their late 20s or early 30s Attacks last 45-90 minutes, happen 1-3x per day. Cluster period is 6-12 wks w/ remission for 12 months. Tx: oxygen via NRB (NC not effective)</p>
<p>Who needs a CT head prior to undergoing lumbar puncture?</p>	<ol style="list-style-type: none"> 1. Evidence of head trauma 2. AMS 3. Seizure 4. Focal neurological deficits 5. Papilledema 6. Immunocompromised pts
<p>Diagnosis: 5 year-old boy with wide based gait, poor tone, and horizontal nystagmus that started suddenly two weeks after a URI</p>	<p>Dx: Acute post-infectious cerebellar ataxia (must rule out other causes)</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Transverse myelitis</p>	<p>SSx: Young person with acute or subacute presentation of back pain that rapidly progresses to sensory and then motor loss at a spinal level Typically occurs after viral infection, but may be idiopathic. Dx: MRI Tx: manage ABCs, steroids, neurology consult</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Cerebral Vein Thrombosis</p>	<p>SSx: headache, seizure, encephalopathy Typically female gender, prothrombotic state (pregnancy, malignancy) Dx: MRV (gold standard), CT may show DELTA sign (hyperdensity at superior sagittal sinus) Tx: heparin gtt</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Slit Ventricle Syndrome</p>	<p>SSx: patient with a VP shunt presenting for positional headache (worse with standing) due to over-drainage of CSF after VP shunt placement or revision (usually weeks/months) Dx: history and CT Tx: neurosurgery consult</p>
<p>What is a rare but pathognomonic finding on noncontrast head CT that is seen in some acute strokes?</p>	<p>Dense MCA sign</p>

Endocrine & Metabolic

Bizz	Buzz
Review expected bicarbonate and pCO2 levels for: Metabolic Acidosis Metabolic Alkalosis Respiratory Acidosis Respiratory Alkalosis	Metabolic Acidosis: ↓ HCO ₃ , ↓ pCO ₂ (hyperventilation) Metabolic Alkalosis: ↑ HCO ₃ , ↑ pCO ₂ (hypoventilation) Respiratory Acidosis: ↑ pCO ₂ , ↑ HCO ₃ (↑ renal reabsorption) Respiratory Alkalosis: ↓ 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 <u>respiratory acidosis</u> and <u>alkalosis</u> ?	Respiratory acidosis: for every ↑ of pCO ₂ by 10, HCO ₃ should ↑ by 1 (Acute) and 3 (Chronic) Respiratory alkalosis: for every ↓ in pCO ₂ by 10, HCO ₃ should ↓ by 2 (Acute) and 5 (Chronic) Mnemonic is "1325" If NOT true then a mixed disorder is present
What is the appropriate respiratory compensation for <u>metabolic acidosis</u> and <u>alkalosis</u> ?	Metabolic acidosis: 1.5 x HCO ₃ + 8 (±2) = appropriate PCO ₂ Metabolic alkalosis: 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. Mnemonic is HAARDUPS: <u>H</u> yperalimentation (TPN) <u>A</u> cetazolamide, <u>A</u> mpho B <u>R</u> enal tubular acidosis <u>D</u> iarrhea <u>U</u> retersigmoidostomy <u>P</u> ost-hypocapneic state <u>S</u> ulfamylon
What are the most common causes of metabolic alkalosis ?	Vomiting, diuretics, and hypochloremia
What is the primary difference between Type I and Type II diabetes ?	Type I: insulin deficiency (auto-immune) Type II: insulin resistance (acquired)

What are the criteria for diagnosis of diabetes ?	Fasting blood sugar >126 (2 separate occasions) Random glucose >200 with signs and symptoms of diabetes Glucose >200 after oral glucose tolerance test HbA1c > 6.5%
Diagnosis: Suspected DKA and coffee-ground emesis	Erosive esophagitis & hemorrhagic gastritis in up to 9% of DKA, rarely need treatment/endoscopy
Signs and symptoms, Diagnosis: DKA	SSx: Polyuria, polydipsia, abdominal pain, vomiting, acetone (fruity) smell, ± unstable vitals/shock, altered mental status, possible coffee-ground emesis Dx: Labs: glucose > 250, pH < 7.3 (VBG acceptable), HCO ₃ <18, AG >10, +Serum/urine ketones Note: workup should include <u>evaluation for cause of DKA</u> (infection rule out) and ECG due to electrolyte abnormalities.
What lab value is critical to know prior to giving insulin for DKA ?	Serum potassium: patients have an overall deficiency of potassium (initial labs may show high K). Risking profound hypokalemia if started on insulin drip without potassium supplementation.
What is the appropriate approach to fluid resuscitation in DKA ?	2 liters normal saline IVF bolus (kids: 10-20 mL/kg), when glucose < 250, start glucose-containing fluids (D51/2 NS)
First intervention to start in DKA ?	IV fluids. Often asked on tests. Give fluids before insulin or potassium.
What is the appropriate approach to electrolyte repletion in DKA ?	Key = POTASSIUM! Know level before giving insulin. K < 3.3: HOLD Insulin & give potassium K = 3.3-5.2: can start insulin but supplement potassium and give potassium in each liter of IVF K > 5.2: Start insulin & normal saline, no supplemental potassium needed Bicarbonate: controversial, only start if severe DKA + intubated Mg: replete with K Na: pseudohyponatremia, abnormal Na will typically correct with fluids
What is the appropriate approach to insulin administration in DKA ?	Evaluate potassium level first, then give 0.1 U/kg/hr drip (double if glucose not decrease by 50 after first hour) Initial bolus not necessary; can also follow SQ regimen *Transition to regular insulin subcutaneous when gap closed and pH > 7.3, stop insulin drip 1 hour after patient given subcutaneous insulin
How do you correct sodium for hyperglycemia (pseudohyponatremia)?	Add 1.6 to sodium for each glucose value of 100 over 100 mg/dL Ex: If glucose is 600 and Na is 125, corrected Na is 125 + (5*1.6) = 132

<p>Diagnosis, Treatment: DKA followed by new altered mental status or seizure</p>	<p>Cerebral edema, more common in kids and new onset type I diabetes, more common with insulin bolus before drip Dx: STAT CT head, frequent neuro checks, airway management as needed Tx: mannitol (1-2g/kg)</p>
<p>What is the mechanism of action and possible adverse effect of Sulfonylureas (Glipizide, glyburide)</p>	<p>Stimulates insulin release from the pancreas; can cause prolonged hypoglycemia in overdose.</p>
<p>What is the mechanism of action and possible adverse effect of Biguanides (Metformin)</p>	<p>Suppress hepatic gluconeogenesis. Adverse effects: diarrhea, lactic acidosis No hypoglycemia</p>
<p>What is the mechanism of action and possible adverse effect of Thiazolidinediones (TZDs- Actos, Avandia)</p>	<p>Increased sensitivity to insulin (muscle & fat). Adverse effects: hepatitis & edema No hypoglycemia</p>
<p>Signs and Symptoms, Diagnosis, Treatment: Hyperosmolar hyperglycemic state</p>	<p>Occurs in Type II DM. SSx: CNS symptoms (stupor/coma) Dx: Labs: glucose > 600, pH >7.3, HCO₃ >18, ± urine ketones, AG <12, severe dehydration (8-12L deficit) Tx: <u>IVF</u>, insulin</p>
<p>Which lab test can help identify factitious hypoglycemia (exogenous administration)?</p>	<p>C-peptide. Only elevated when endogenous insulin is released from the pancreas, not with exogenous use.</p>
<p>What is the rule to calculate maintenance IVF rate?</p>	<p>4 mL/kg for first 10 kg, 2 mL/kg for second 10 kg, 1 mL for each additional kg to max of ~120 mL/hr total</p>
<p>What are typical causes and appropriate treatment for Hypervolemic, Euvolemic and Hypovolemic Hyponatremia?</p>	<p>Hypervolemic: CHF, ESRD, cirrhosis Tx: water restrict + diuretics Euvolemic: SIADH, psychogenic polydipsia Tx: water restrict Hypovolemic: vomiting, diarrhea, third spacing, diuretics Tx: NS vs 1/2 NS</p>
<p>What is the approach to correction of hyponatremia?</p>	<p>Overall goal correction rate 0.3 mEq/hr or 8-10 mEq/day. **Note: rapid correction risks central pontine myelinolysis/demyelination.** Tx: Na > 120 and asymptomatic = no emergent treatment Na < 120 and neurologic symptoms, give 3% NaCl (100 mL over 10 minutes, additional 100 mL over 50 minutes)</p>
<p>How and when does central pontine myelinolysis present?</p>	<p>How: Altered mental status (lethargy and coma) with spastic quadriplegia after treatment for hyponatremia. When: 1-6 days after treatment for hyponatremia. Patients with chronic hyponatremia (e.g. alcoholics) are most at risk for this.</p>

What is the approach to treatment of hypernatremia ?	Overall goal correction rate 1-2 mEq/hr **Note: rapid correction risks cerebral edema** Free Water Deficit = $(0.6 \times \text{weight (kg)} \times (\text{Current Na} - 140)/140)$ and replace with NS until euvolemic, then D5W vs D5 1/2 NS Give 50% over 12 hours, remainder over the next 24 hours
What is the most common cause of hyperkalemia ?	Lab error, resend lab
What ECG changes are seen in hyperkalemia ?	Peaked T waves > PR prolonged > loss of P wave > wide QRS > sine wave > VT/VF
What is the general approach to treatment of hyperkalemia ?	Cardioprotection: calcium gluconate or calcium chloride ONLY if ECG changes K shifters: Insulin/glucose, bicarbonate if acidotic, albuterol K excretion: lasix, kayexalate, lokelma, hemodialysis
Signs and Symptoms, Treatment: hypokalemia	Most common electrolyte disturbance, often secondary to GI or diuretic losses. SSx: cramps, weakness, arrhythmias, cardiac arrhythmias, rhabdo Tx: K repletion 10 mEq ~ $\uparrow 0.1\text{mEq/L}$, give 10-20 mEq/hr; *Note: supplement with Magnesium (\uparrow absorption)*
What is the most specific ECG change associated with hypokalemia ?	Flattened or inverted T wave, U waves = specific, prolonged QT, ST depression
Signs and Symptoms, Diagnosis, Treatment: Hypercalcemia	Ca > 10.5. Causes: hyperparathyroid (overall most common cause), malignancy (most common inpatient cause), dehydration SSx: BONES (bone pain), STONES (renal, biliary), GROANS (abdominal pain, nausea/vomiting), THRONES (polyuria) and PSYCHIATRIC OVERTONES (depression, anxiety, insomnia) Dx: labs: serum Ca, iCa; ECG with short QT Tx: immediate if Ca > 14 (12-14 if signs/symptoms) with IVF (first step), Calcitonin (\uparrow excretion, inhibits osteoclasts), Bisphosphonates (inhibits osteoclasts, requires days to work), Steroids (\downarrow GI absorption), Lasix (if volume overload)
Signs and Symptoms, Diagnosis, Treatment: Hypocalcemia	Ca < 8.5. Causes: hypoparathyroidism (thyroidectomy = most common cause), Vitamin D deficiency, high phosphorous, low/high magnesium SSx: paresthesias, tetany, Chvostek's sign, Trousseau's sign, seizure Dx: labs: serum Ca, iCa; ECG with long QT Tx: IV calcium (if <7.5 and severe signs/symptoms), give Vitamin D and Mg as needed
What are the significant differences between calcium gluconate and calcium chloride ?	Calcium chloride contains three times as much calcium as calcium gluconate . Calcium chloride must be given via central access (scleroses veins and causes tissue damage if leaks from vessels), while calcium gluconate can be given through peripheral IV.

Signs and Symptoms, Treatment: Hypermagnesemia	On tests, this is seen most often when treating eclampsia SSx: weakness, <u>loss of reflexes (first sign)</u> , dysrhythmias, <u>respiratory depression</u> Tx: IV calcium
Diagnosis, Treatment: patient with alcohol use disorder with altered mental status, ataxia, visual symptoms	Wernicke's Encephalopathy; Thiamine (B1) deficiency Tx: Thiamine 500 mg IV, improvement in hours
Diagnosis: patient with alcohol use disorder with short term memory loss	Korsakoff's Psychosis; Thiamine (B1) deficiency, irreversible
Diagnosis, Treatment: poor nutrition and high output cardiac failure (dyspnea, peripheral edema)	"Wet" Beriberi due chronic thiamine deficiency Tx: thiamine 100 mg IV
Diagnosis: diarrhea, dermatitis, dementia	Pellagra (Niacin (B3) deficiency). Think about populations who eat untreated corn/commeal.
Diagnosis: Crohn's patient with macrocytic anemia and paresthesias	Cobalamin (B12) deficiency; high risk include Crohn's (B12 absorbed in ileum), vegans, alcohol use disorder, PPIs, pernicious anemia (antibody to intrinsic factor) Causes megaloblastic anemia + neurologic deficits, hypersegmented neutrophils
Diagnosis: patient with alcohol use disorder with macrocytic anemia	Folic Acid deficiency High risk: alcohol use disorder, elderly, phenytoin use; no neurologic changes Hypersegmented neutrophils seen as well
Diagnosis: child with poor diet and bowed legs	Rickets, Vitamin D deficiency >> poor calcium absorption Osteomalacia: adult equivalent, normal height
Diganosis: bad skin, bleeding gums, perifollicular hemorrhages, and poor wound healing	Scurvy, Vitamin C deficiency >> poor collagen formation
Which vitamins are toxic in overdose?	Fat soluble vitamins, ADEK: A: bear liver consumption, skin changes, pseudotumor cerebri D: hypercalcemia, hypercalciuria E: ↑ bleeding K: hemolytic anemia, jaundice in newborns
What are the fat-soluble vitamins ?	A, D, E, K
Which hormones are secreted from the pituitary gland ?	GOAT FLAP: G rowth Hormone O xytocin, A DH T SH F SH L H A CTH P rolactin ALL but Oxytocin and ADH are from anterior pituitary
What are the most common causes of hypopituitarism ?	Mass lesions, bleeds (pituitary apoplexy), hypothalamic disease, Sheehan's syndrome

Diagnosis: low cortisol level but normal aldosterone	ACTH deficiency , causes 2° adrenal insufficiency. Aldosterone production means that the adrenal glands are working.
Diagnosis: fatigue and inability to lactate post-partum	Post-partum hemorrhage >> ischemia and necrosis of the pituitary gland >> Sheehan's syndrome causing prolactin deficiency (and panhypopituitarism).
Diagnosis, Treatment: visual field deficits, headache, hormonal abnormalities	Pituitary adenoma (macro if > 1 cm) Tx: transsphenoidal surgery
Treatment: prolactinoma	Most common pituitary tumor. Tx: bromocriptine or cabergoline (dopamine receptor agonists > decrease prolactin levels), typically doesn't require surgery
Signs and symptoms, Treatment: Cushing's syndrome	Cortisol excess. ACTH secreting pituitary adenoma or exogenous steroids (most common cause) SSx: ('CUSHING') <u>C</u> entral obesity 24 hr <u>U</u> rinary cortisol ↑ or ACTH level <u>S</u> triae <u>H</u> ypertension/ <u>H</u> yperglycemia/ <u>H</u> irsutism <u>I</u> atrogenic <u>N</u> eoplasms <u>G</u> lucose intolerance Tx: surgery (tumor)
Diagnosis, Treatment: Headache and tunnel vision in oversized person	Growth Hormone secreting pituitary adenoma Children: gigantism Adults: acromegaly Labs: ↑ GH & IGF 1 Tx: surgery
Which hormones are produced by the adrenal glands ?	Medulla: epinephrine and norepinephrine Cortex: cortisol, androgens, aldosterone
Identify the key differences between primary and secondary adrenal insufficiency .	Both: deficiency of adrenal gland hormone production Primary (adrenal disease): ↑ CRH & ACTH , Addison's disease (autoimmune) = most common cause, rapid withdrawal of steroids = most common cause in US SSx: shock, hypoglycemia, ↓ mineralocorticoid = ↓ Na/glucose, ↑ K, <u>HYPERPigmentation</u> (buccal, due to ↑ ACTH) Secondary (pituitary disease): ↑ CRH, ↓ ACTH: ↓ Na/glucose, normal K, NO hyperpigmentation; Tx: IVF, glucocorticoids, vasopressors
Signs and Symptoms, Diagnosis, Treatment: adrenal crisis	SSx: shock (refractory to fluids and pressors), fatigue, abdominal pain, AMS Dx: hypoglycemia, hyponatremia, hyperkalemia Tx: IVF, dextrose, replacement of GC and MC with hydrocortisone
What is the strategy for stress dosing steroids in ill patients on chronic steroids ?	Double (minor symptoms) or triple (more severe symptoms) their home dose for 2-3 days. 1-2 mg/kg hydrocortisone if in adrenal crisis with hypotension.

Diagnosis: young child with abdominal mass and hypertension	Neuroblastoma (adrenal medulla tumor)
Diagnosis: hypertension, headache, palpitations, elevated catecholamines	Pheochromocytoma (adrenal medulla tumor)
Review the hormone cascade and general function of thyroid hormones	Thyroid Releasing Hormone (hypothalamus) → Thyroid Stimulating Hormone (anterior 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 Disease (most common cause, young person) Toxic nodular goiter (elderly) Iodine-induced (amiodarone) Thyroiditis (amiodarone)
Signs and Symptoms, Diagnosis: thyrotoxicosis	SSx: heat intolerance, palpitations, wt loss, tachycardia, anxiety, hyperreflexia, goiter, exophthalmos, pretibial edema Dx: ↓ 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 (and sequence) for Thyroid storm ?	1) Beta-blockers (Propranolol): ↓ sympathetic activity + blocks peripheral conversion of T4 → T3 2) Antihormone medication: PTU (if Pregnant) or Methimazole (blocks new hormone synthesis) 3) Potassium Iodide (AFTER above, blocks release of preformed hormone) 4) Steroids (blocks peripheral conversion of T4 → T3) 5) Treat precipitant & prevent decompensation (IVF, tylenol, cool as needed) The order is controversial. Just know that PTU comes BEFORE iodine as this is typically tested.
What are common causes of hypothyroidism ?	Hashimoto's (most common cause in US, autoimmune), medications, postpartum, iodine deficiency (most common cause worldwide)
Signs and Symptoms, Diagnosis, Treatment: hypothyroidism	SSx: fatigue, weight gain, cold intolerance, brittle hair and nails, constipation, periorbital edema, slow reflexes, edema Dx: TSH (↑ with 1°, ↓ with 2°), ↓ free T3 / T4 Tx: levothyroxine
Signs and Symptoms, Treatment: Myxedema Coma	SSx/Dx: AMS, hypoglycemia, hypothermia, bradycardia, hypotension, edema Tx: hydrocortisone, IV levothyroxine, supportive care (warming)

<p>Diagnosis, Treatment: Thyroid Cancer</p>	<p>Five percent of thyroid nodules are cancerous, common cancer overall but low mortality Dx: fine-needle aspiration (biopsy) Tx: thyroidectomy, radioactive iodine-131, thyroid supplementation</p>
<p>Signs and Symptoms, Diagnosis, Treatment: Hyperparathyroidism</p>	<p>SSx: those of hypercalcemia - stones, bones, groans, psych overtones Dx: \uparrow PTH \rightarrow \uparrow Ca, \downarrow Phos Tx: lower Ca with IVF (first line), calcitonin, bisphosphonates, steroids, surgery</p>
<p>Signs and Symptoms, Diagnosis, Treatment: hypoparathyroidism</p>	<p>May be secondary to thyroid surgery SSx: same as hypocalcemia (paresthesias, tetany, Chvostek's sign, Trousseau's sign, seizure) Dx: \downarrow PTH \rightarrow \downarrow Ca, \uparrow Phos Tx: replacement of Ca, Vit D</p>
<p>Diagnosis, Treatment: Patient with a history of anorexia presents with signs of heart failure after starting an outpatient refeeding program.</p>	<p>Refeeding syndrome. Occurs when refeeding begins before correcting electrolyte abnormalities. Dx: hypophosphatemia, hypokalemia, hypomagnesemia, and ultimately volume overload and CHF. Tx: stop refeeding, correct electrolyte abnormalities.</p>
<p>How do you manage sulfonylurea overdose?</p>	<p>Glucose supplementation, octreotide (decrease insulin release from pancreas), and observation admission for at least 24 hours.</p>
<p>Signs and Symptoms, Treatment: Thyroid Storm</p>	<p>SSx: profound tachycardia, GI symptoms (nausea/vomiting/diarrhea), CNS dysfunction (anxiety, confusion, apathy, coma), goiter and multiorgan dysfunction. Tx: order is important! 1. Beta blockers 2. Thionamides (PTU, methimazole) 3. Iodine (only given 1 hour after methimazole) 4. Glucocorticoid (hydrocortisone) 5. Bile acid sequestrants (cholestyramine) *Dispo to ICU.</p>

Toxicology

Bizz	Buzz
<p>What is the mechanism of activated charcoal?</p> <p>What is the dosing and route of administration?</p>	<p>Activated charcoal has a high surface area to bind toxin in GI tract and prevent systemic absorption</p> <p>Administered orally: 10 g activated charcoal per 1 g drug</p>
<p>When is activated charcoal contraindicated or ineffective?</p>	<p>Contraindicated: AMS/obtunded patient (aspiration risk), intestinal obstruction/ileus</p> <p>Ineffective: Cyanide, <u>Hydrocarbons</u>, <u>Ethanol/alcohols</u>, <u>Metals</u>, <u>Iron</u>, Caustics, <u>Lithium</u>, CAMphor, Potassium (CHEMICaL CAMP)</p>
<p>What is the proposed mechanism of whole bowel irrigation?</p> <p>How should it be administered?</p>	<p>Proposed mechanism: Iso-osmotic agent taken in large volume will hasten toxin's progress through intestines and prevent absorption</p> <p>Administration: Dose 1-2 L/hr (adults) or 500 mL/hr (pediatrics), consider giving by NGT</p> <p>Continue until rectal effluent is clear (typically collected via rectal tube).</p> <p>Consider for "drug packers" and sustained-release BBs.</p>
<p>For what types of ingestion is whole bowel irrigation most effective?</p>	<p>Metals (iron or lithium), sustained-release formulations, enteric-coated medications, "body packers"</p>
<p>What toxins are cleared by hemodialysis?</p>	<p>"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</p>
<p>Pathophysiology: Acetaminophen (APAP) overdose</p>	<p>At therapeutic levels, APAP is metabolized mostly via sulfation & glucuronide conjugation with a small component of CYP450.</p> <p>CYP450 metabolism → toxic NAPQI metabolite</p> <p>In overdose, sulfation and glucuronidation is overloaded → excess NAPQI accumulates → liver toxicity</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Acetaminophen (APAP) overdose</p>	<p>SSx: intentional or unintentional ingestion, delayed presentation can present with abdominal pain, nausea/vomiting, AMS</p> <p>Dx: can use Rumack-Matthew nomogram for SINGLE, ACUTE ingestions; get APAP level at ≥ 4 hours (< 4 hours NOT useful, unless ZERO)</p> <p>High risk of toxicity: > 150 mg/kg (acute) or >4 g/day (chronic)</p> <p>Tx: N-acetylcysteine (NAC) → restores glutathione</p> <p>Best if given w/n 8 hours of ingestion</p> <p>Dose: PO (140 mg/kg load, 70 mg/kg q4hr) or IV (150 mg/kg load, 50 mg/kg over 4hr, 100 mg/kg over 16 hr)</p> <p>Safe for pregnant women and children.</p> <p>Side effect: anaphylactoid reaction</p>

When can you use the Rumack-Matthew nomogram for acetaminophen toxicity?	For single acute ingestions (not for chronic ingestions); serum APAP level must be drawn at least 4 hours after ingestion.
Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: NSAID overdose	Pathophysiology: COX inhibitor → decreased prostaglandin production SSx: AMS, coma, ataxia, metabolic acidosis, seizure (massive overdose) Tx: supportive
Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Aspirin overdose	Pathophysiology: Uncouples oxidative phosphorylation → increased metabolic rate, stimulates respiratory centers, hyperthermia SSx: ↑ RR, ↑ temp, ↑ HR (sinus tach = MC sign), tinnitus, vertigo, AMS, seizure Dx: Primary metabolic alkalosis (EARLY), anion gap metabolic acidosis (LATER) Serum salicyte levels: 10-30 therapeutic, >40-50 toxicity Tx: GI decon; activated charcoal if within 1-2 hrs of ingestion; urine alkalinization with bicarb (+K, +Mg) infusion (enhances urinary excretion of salicylate, also prevents CNS distribution); dialysis (acute level >100, chronic level >60, OR +renal failure, severe acidemia, pulmonary/cerebral edema) Avoid intubation if possible; set high respiratory rate if intubated.
Signs & Symptoms, Treatment: Opioid overdose	SSx: decreased LOC + respiratory depression + miosis (pinpoint pupils) Tx: naloxone (titrate to RESPIRATORY RATE)
What are the unique clinical complications of meperidine, tramadol and methadone?	Meperidine: seizures, serotonin syndrome, often dilated pupils (different from other opioids!) Tramadol: seizures, serotonin syndrome, anticholinergic effects (mydriasis) Methadone: QT prolongation (& TdP), hypoglycemia
Which opioids are NOT seen on urine toxicology screen ?	Will NOT detect synthetics including fentanyl, hydromorphone, buprenorphine, meperidine . Natural derivatives will show up (heroin, morphine, codeine).
What is the potential risk of using meperidine, tramadol, or dextromethorphan in the setting of antidepressant use?	Serotonin syndrome
Mechanism, Signs & Symptoms, Treatment: Clonidine toxicity	Mechanism: alpha-2 agonist SSx: AMS + miosis + respiratory depression; similar to opioid toxidrome but causes bradycardia and hypotension Tx: supportive, atropine, pressors, naloxone

What common substances are associated with methanol, ethylene glycol, and isopropyl alcohol ingestions?	Methanol: windshield washer fluid, wood alcohol, moonshine, paint solvent Ethylene glycol: antifreeze, radiator coolant, aircraft de-icing Isopropyl alcohol: rubbing alcohol, hand sanitizer
Clinical manifestations of ethylene glycol and methanol toxicities?	Comatose , mild hypothermia, tachypnea, 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)
Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Methanol toxicity	Pathophysiology: metabolized to formic acid SSx: altered mental status, optic neuropathy ("snowy" field of vision or blindness), and basal ganglia injury Dx: anion gap metabolic acidosis and increased osmolar gap Tx: fomepizole , ethanol, dialysis
Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Ethylene glycol toxicity	Pathophysiology: metabolized to glycolic and oxalic acids SSx: altered mental status, hematuria, oliguria , renal failure Dx: hypocalcemia, anion gap metabolic acidosis , and increased osmolar gap Tx: fomepizole , ethanol, dialysis
Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Isopropyl alcohol toxicity	Pathophysiology: metabolized to acetone (uncharged ketone) SSx: CNS depression similar to EtOH toxicity Dx: increased osmolar gap and ketosis WITHOUT acidosis or anion gap Tx: supportive
What is the biggest lab difference between ethylene glycol and methanol ingestions as compared to isopropyl alcohol ingestion?	Ethylene glycol and methanol will lead to an anion gap metabolic acidosis with ↑ osmolar gap. Isopropanol will cause osmolar gap and ketosis but no acidosis .
How do you calculate the osmolar gap ?	Serum Osmolality = $2xNa + Glucose/18 + BUN/2.8 + EtOH/4.6$ Gap = Calculated - Measured Normal is less than 10.
What cofactors are required to treat ethylene glycol ingestions? Methanol ingestions?	Ethylene glycol: thiamine & pyridoxine Methanol: folinic acid
What are potential adverse effects of ethanol intoxication?	"4 Hs": Hypotension , Hypoventilation , Hypothermia , Hypoglycemia ; atrial tachycardias ("holiday heart")
What is the time course of symptoms in alcohol withdrawal?	Alcohol withdrawal syndrome: cessation/reduction of etoh + etoh withdrawal sx Symptoms begin within 6-24 hours: Tremulousness (6-12 hrs) → hallucinations (12-48 hrs) → seizures (12-48 hrs) → delirium tremens (>48hrs)

<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Lidocaine toxicity</p>	<p>Pathophysiology: Na channel blockade → nerve conduction delays Toxic dose: >4 mg/kg plain or >7 mg/kg with epi SSx: CNS toxicity (perioral numbness, slurred speech, seizure), CV toxicity (VT/VF, AV block, AVNRT), methemoglobinemia, allergic rxn Tx: benzodiazepines for seizure, bicarb for arrhythmia or QRS widening, methylene blue for methemoglobinemia, epi for anaphylaxis, consider intralipid for CV collapse</p>
<p>What is the difference between ester versus amide anesthetics?</p>	<p>Esthers (one 'i' in name); cocaine, procaine, benzocaine; shorter acting; higher risk of allergic reaction 2/2 preservative or PABA Amides (two i's in name): lidocaine, mepivacaine, bupivacaine; longer acting; can use amides if allergic to ester/preservative</p>
<p>What are reasonable local anesthetic options for a patient with lidocaine allergy?</p>	<p>Allergy is usually to the <u>preservative</u> in lidocaine. Can use crash cart lido (preservative free) or locally injected diphenhydramine.</p>
<p>Causes, Signs & Symptoms, Treatment: Anticholinergic toxidrome</p>	<p>Causes: tricyclic antidepressants, atropine, antihistamines (MCC), belladonna (nightshade), jimsonweed, phenothiazines. SSx: "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 (anhidrosis), bloated as a toad (stool or urinary retention), the heart runs alone (tachycardia)" Tx: supportive, benzodiazepines (seizures), sodium bicarb (wide complex dysrhythmias) ± physostigmine (cholinesterase inhibitor; avoid if known TCA overdose, QRS widening, seizure)</p>
<p>How might you identify tricyclic antidepressant overdose in a patient with anticholinergic toxidrome?</p>	<p>Get an ECG! TCA overdose is suggested by wide QRS (threshold is >100 ms) or terminal R wave in aVR.</p>
<p>How can you distinguish an anticholinergic toxidrome versus sympathomimetic toxidrome?</p>	<p>Anticholinergic: dry skin Sympathomimetic: diaphoresis</p>
<p>Signs & Symptoms, Treatment: Cholinergic toxidrome</p>	<p>Causes: insecticides, organophosphates, chemical warfare SSx: Clinically WET, "DUMBBBELLS" (Diarrrhea, Uriination, Miosis, Bradycardia, Bronchorrhea, Bronchospasm, Emesis, Lacrimation, Lethargy, Salivation) Tx: atropine (often high doses, titrate to dry secretions), 2-PAM (pralidoxime) if administered early Do not intubate w/ succinylcholine (prolonged paralysis).</p>

<p>Pathophysiology, Diagnosis, Treatment: Heparin-induced thrombocytopenia (HIT)</p>	<p>Pathophysiology: Formation of antibodies → platelet inactivation Dx: +HIT antibody, thrombocytopenia, time of onset (5-10 d), thrombosis, no other cause (4 T's) Tx: STOP heparin or LMWH; can change to DTI (argatroban)</p>
<p>What is the mechanism of clopidogrel? How is it reversed?</p>	<p>Mechanism: antiplatelet agent monitored with P2Y12 level Reversal: platelet transfusion</p>
<p>Pathophysiology, Signs & Symptoms, Treatment: Phenothiazines (e.g., prochlorperazine, promethazine, chlorpromazine) toxicity</p>	<p>Pathophysiology: dopamine receptors blockade (also block ACh receptors, ion channels) SSx: CNS effects (sedation, seizures, extrapyramidal symptoms, dystonia); cardiovascular effects (long QTc, hypotension); miosis; NMS Tx: supportive (IVF, benzodiazepines, Mg)</p>
<p>Signs & Symptoms, Treatment: 5HT3 (serotonin) antagonists (i.e. ondansetron)</p>	<p>SSx: palpitations and hemodynamic instability related to prolonged QT & Torsades De Pointes Tx: Mg 2g (IV push), cardioversion/pacing</p>
<p>What drugs increase the risk of prolonged QT/Torsades and what is the treatment?</p>	<p>Causes: Class 1A & 1C antiarrhythmics, TCAs, antipsychotics, abx (macrolides, fluoroquinolones), antiemetics Tx: Mg 2 g IV push, overdrive pacing, cardioversion (unstable)</p>
<p>Pathophysiology, Signs & Symptoms, Treatment: Cocaine intoxication</p>	<p>Pathophysiology: inhibits neuronal reuptake of catecholamines (e.g. norepinephrine), Na⁺ channel blockade SSx: HTN, hyperthermia, tachycardia, rhabdomyolysis, MI 2/2 coronary vasospasm, seizure, VT Tx: benzodiazepines, cooling, nitrates, nicardipine, phentolamine for tachycardia AVOID β-blockers d/t risk of unopposed α activity.</p>
<p>Pathophysiology, Signs & Symptoms, Treatment: Amphetamine intoxication</p>	<p>Pathophysiology: ↑ catecholamine release SSx: HTN, tachycardia, hyperthermia, rhabdomyolysis, hypertensive intracranial hemorrhage Tx: benzodiazepines, cooling, nitrates, nicardipine (avoid beta blockers)</p>
<p>Signs & Symptoms, Treatment: Synthetic cannabinoid ("K2," "Spice," "Herbal Marijuana") intoxication</p>	<p>SSx: anxiety, paranoia, tachycardia, diaphoresis, psychosis, seizures Tx: supportive (IVF, benzodiazepines for agitation & seizures)</p>
<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Hallucinogenic amphetamines (e.g. MDMA/ecstasy)</p>	<p>Pathophysiology: ↑ catecholamine and serotonin release SSx: appears like amphetamine OD with serotonergic properties (hyperthermia, rhabdo, bruxism, hyponatremia). Dx: elevated CK, hyponatremia Tx: supportive (IVF, benzodiazepines, cooling, intubation/paralysis PRN)</p>

<p>Signs & Symptoms, Diagnosis, Treatment: Gamma-hydroxybutyrate (GHB) intoxication</p>	<p>SSx: "date rape drug," bradycardia, ↓ RR, poor coordination, hypotension, coma, rapid awakening after metabolism Dx: lab testing not useful (in blood 6hr, urine 12hr) Tx: supportive, intubate PRN</p>
<p>Signs & Symptoms, Treatment: Irritant gas (e.g. chlorine, ammonia, hydrogen chloride) exposure</p>	<p>SSx: respiratory tract and mucosal irritation, cough, SOB, pulmonary edema, and conjunctivitis Tx: ABCs, O₂, bronchodilators, supportive care</p>
<p>Compare alkaline versus acidic ingestion. How are they treated?</p>	<p>Alkaline: liquefactive necrosis → deep injuries, perforation (4-7 d), subsequent stricture Acidic: coagulative necrosis → limited injury, perforation risk (3-4 d), gastric outlet obstruction (2-4 w) Tx: NPO, do NOT attempt to induce emesis or neutralize, early endoscopy, supportive care</p>
<p>Common associations, Signs & Symptoms, Diagnosis, Treatment: Hydrofluoric acid burns</p>	<p>Common associations: glass etching, electronic manufacturing, rust removal, metal cleaning SSx: painful skin burns leading to eschar formation, also binds Ca & Mg Dx: severe hypocalcemia & hyperkalemia (muscle spasms, arrhythmia) Tx: copious low-pressure irrigation, calcium gluconate gel and injection **can cause systemic effects**</p>
<p>Why are button battery ingestions so dangerous? How are they treated?</p>	<p>Mechanism: generation of electrical current against mucosal surface; severe burns; high perforation risk If found in nose, ear, or esophagus → requires emergent removal Do not use vasoconstrictive agents in the nose (e.g. oxymetazoline). If in stomach and pt asymptomatic → can monitor</p>
<p>Signs & Symptoms, Treatment: Beta blocker overdose</p>	<p>SSx: Bradycardia (most common), hypotension, HYPOglycemia (or normoglycemia), HYPERkalemia, AV blockade, QT prolongation, seizures (propranolol) Tx: GI decontamination, atropine, glucagon, high-dose insulin (1 U/kg/hr) + glucose, calcium, dialysis, epinephrine bolus/drip, intralipid (if crashing), pacing (minimally effective)</p>
<p>Signs & Symptoms, Treatment: Calcium channel blocker overdose</p>	<p>SSx: Bradycardia, hypotension (most common), HYPERglycemia (often refractory to even high-dose insulin), AV blockade, any bradyarrhythmia, warm extremities Tx: GI decontamination, atropine, calcium, high-dose insulin (1 U/kg/hr) + glucose (may not be necessary given refractory hyperglycemia), high-dose pressors, intralipid (if crashing), pacing (minimally effective)</p>

<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Cardiac glycoside intoxication (e.g., digoxin, foxglove, oleander)</p>	<p>Pathophysiology: Blocks Na-K-ATPase → ↑ Ca in cell → ↑ contractility SSx: ANY dysrhythmia possible (PVCs MC, bidirectional VT and slow A fib = pathognomonic), agitation, yellow-tinted vision; Dx: hyperkalemia (correlates with severity of toxicity), serum digoxin levels. Tx: activated charcoal, Fab fragments (e.g. Digifab, Digibind), IVF, atropine, pressors, transcutaneous pacing (minimally effective), AVOID Ca for HyperK (stone heart - occurs on the test but not in real life)</p>
<p>What are the indications to give Dig-Fab in digoxin overdose?</p>	<p>Ventricular dysrhythmias, symptomatic bradycardias, hyperkalemia >5, elevated Dig level > 10 ng/dl in adults or > 4 ng/dl in children</p>
<p>What characteristic ECG changes may be seen with digoxin effect vs digoxin toxicity?</p>	<p>Dig effect: short QT, downsloping or "scooped" ST segment ("Salvador Dali mustache"), biphasic T wave Dig toxicity: can do anything to the ECG including PVCs, AV block, atrial tachycardia with block, polymorphic and bidirectional VT (pathognomonic but rare)</p>
<p>Name 4 common medications that cause bradycardia and hypotension ("Brady Bunch") in overdose.</p>	<p>Beta blockers, calcium channel blockers, digoxin, clonidine</p>
<p>Diagnosis: Major depression + new seizure + wide QRS</p>	<p>Tricyclic antidepressant (TCA) overdose</p>
<p>Pathophysiology, Signs & Symptoms, Treatment: TCA overdose (amitriptyline, nortriptyline, doxepin)</p>	<p>Pathophysiology: inhibits reuptake of bioamines (serotonin, norepinephrine, and dopamine), SSx: anticholinergic effects, sodium channel blockade (wide QRS), α-1 blockade (vasodilation, hypotension), antihistamine effects (sedation), GABA antagonism (seizures) Tx: supportive, sodium bicarb for wide QRS (threshold >100) or dysrhythmia, benzodiazepines for seizures</p>
<p>What common medications are associated with Na channel blockade in overdose?</p>	<p>TCAs (most common), diphenhydramine, propranolol, procainamide, cocaine</p>
<p>Pathophysiology, Triggers, Signs & Symptoms, Treatment: MAOI toxicity (phenelzine, selegiline)</p>	<p>Pathophysiology: inhibition of monoamine (dopamine, norepinephrine, serotonin) metabolism/degradation Triggers: tyramine-containing foods (salami, red wine, aged cheese) and drugs (meperidine, cocaine, dextromethorphan, SSRIs, lithium) SSx: sympathomimetic toxidrome, hypertensive crisis Tx: supportive, IVF, phentolamine, nitroprusside PRN, cooling, benzodiazepines for seizures</p>
<p>What are primary concerns in selective serotonin reuptake inhibitor (SSRI) overdose? What management is indicated even in asymptomatic patients?</p>	<p>Serotonin syndrome (AMS, autonomic instability, hyperthermia, clonus), delayed seizures, arrhythmia (QTc prolongation) Telemetry monitoring indicated even in asymptomatic patients.</p>

<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Carbon monoxide toxicity</p>	<p>Pathophysiology: binds to Hgb with high affinity (higher than O₂) → causes left shift of the O₂ dissociation curve (poor O₂ delivery) SSx: flu-like symptoms (multiple people with same SSx), cherry-red skin (with severe toxicity or when dead), O₂ sat inaccurate Dx: co-oximetry, carboxyhemoglobin level (on ABG) Tx: supplemental O₂, hyperbaric oxygen therapy (HBOT)</p>
<p>What are the indications for hyperbaric oxygen therapy for carbon monoxide (CO) poisoning?</p>	<p>End-organ damage at any COHb level, LOC/coma/seizure, CNS effects (neuro findings, AMS), COHb >25% (15% in pregnant women)</p>
<p>What is the half-life of carboxyhemoglobin on 1) room air, 2) 100% NRB, and 3) hyperbaric O₂?</p>	<p>1) Room air: 4-6 hours 2) NRB: 60-90 minutes 3) Hyperbaric O₂: 30 minutes</p>
<p>What are the expected O₂ sat, PCO₂, and PO₂ values in carbon monoxide toxicity?</p>	<p>O₂ sat likely to appear normal (most pulse oximeters cannot distinguish COHb from oxyhemoglobin). PCO₂ is unaffected. PO₂ is dissolved O₂ (not bound) and is unaffected. Co-oximetry will be abnormal.</p>
<p>Sources, Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Cyanide toxicity</p>	<p>Sources: burning of wool, silk, plastics, smoke inhalation Pathophysiology: inhibits oxidative phosphorylation, blocks ATP production SSx: "bitter almond" smell, bradycardia, hypotension, confusion, seizure, coma Dx: SEVERE lactic acidosis Tx: hydroxocobalamin (forms carboxy-B12) OR amyl nitrate (converts to MetHgb → binds CN) AND Na thiosulfate (converts CN → thiocyanate → excreted in urine)</p>
<p>What is the appropriate treatment for combined carbon monoxide (CO) and cyanide (CN) toxicity?</p>	<p>Oxygen, Na thiosulfate or hydroxocobalamin Do NOT give amyl nitrate & Na nitrate → can cause methemoglobinemia → will worsen oxygen carrying capacity (already compromised by CO)</p>
<p>Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Methemoglobinemia</p>	<p>Pathophysiology: state in which Hgb exists in ferric form (Fe³⁺) → can't transport O₂ (Hgb only binds in its Fe²⁺ form) and ↓ oxygen delivery (O₂ sat classically 85% regardless of degree of toxicity) SSx: "chocolate" brown blood, central cyanosis Dx: abnormal co-oximetry Tx: oxygen, methylene blue (except if G6PD → hemolysis)</p>
<p>When do you give methylene blue for methemoglobinemia?</p>	<p>Cyanotic but otherwise asymptomatic w/ methemoglobin levels >20% OR Symptomatic w/ methemoglobin levels >10%</p>

<p>What are potential causes of methemoglobinemia?</p>	<p>Dapsone, nitrates/nitrites, antimalarials, local anesthetics (**teething baby acting normally but has cyanosis d/t benzocaine-containing gel on gums **), aniline dyes, phenazopyridine (Pyridium), benzodiazepines, well water</p>
<p>How can you determine if a baby is cyanotic because of a congenital heart defect or because of acquired methemoglobinemia?</p>	<p>Congenital heart defect: on 100% O₂ → still has low PO₂ on ABG; baby becomes cyanotic when crying Acquired methemoglobinemia: on 100% O₂ → PO₂ improves; baby is always cyanotic</p>
<p>Sources, Pathophysiology, Signs & Symptoms, Treatment: Hydrogen sulfide toxicity</p>	<p>Sources: decay of sulfur material (industrial sources, volcanoes, sulfur springs, septic tanks) Pathophysiology: similar to CN; inhibits oxidative phosphorylation; blocks ATP production SSx: "rotten egg smell", industrial worker with unknown cause of LOC, dyspnea, cyanosis, headache, conjunctivitis, GI upset, bradycardia, AMS Dx: ABG with metabolic acidosis and normal SpO₂/PaO₂; elevated lactate Tx: remove from source; 100% oxygen; hydroxocobalamin; amyl nitrate (induce MetHgb); HBOT</p>
<p>Signs & Symptoms, Diagnosis, Treatment: "Metal fume fever"</p>	<p>SSx: welder with flu-like illness; symptoms worst on Monday and improve by repeat exposure through the week (tachyphylaxis); aka "Monday morning fever" Dx: normal CXR Tx: supportive</p>
<p>Associations, Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Arsenic toxicity</p>	<p>Associations: wood preservatives, garlic taste/smell after ingestion Pathophysiology: decouples oxidative phosphorylation → interferes with ATP production → multisystem organ failure SSx: GI sx (TRIAD of abd pain, hematuria, jaundice), heme (massive RBC hemolysis), renal failure, shock, arrhythmia, CNS (seizure, ascending flaccid paralysis) Dx: URINE arsenic level (spot and 24hr); serum arsenic not useful because it clears too quickly Tx: chelation therapy with dimercaprol (preferred) or dimercaptosuccinic acid (DMSA)</p>
<p>Presentation, Treatment: Hydrocarbon intoxication (paint thinners, gasoline, chloral hydrate, lighter fluid)</p>	<p>Presentation: Commonly sniffed or huffed; can cause ARDS if aspirated; can also cause VF/VT (sudden sniffing death) Tx: beta blockers, supportive care</p>
<p>What is the difference between sniffing, huffing, and bagging?</p>	<p>Sniffing: from container into nose Huffing: from impregnated cloth into mouth/nose Bagging: from plastic bag into nose/mouth</p>

<p>Pathophysiology, Signs & Symptoms, Treatment: Iron overdose</p>	<p>Pathophysiology: mucosal corrosive; inhibits oxidative phosphorylation; impairs ATP synthesis Toxicity: >20 mg/kg is toxic, >60 mg/kg is lethal Stages of toxicity: I: GI ssx (0-6 hrs) II: latent; asymptomatic (6-24 hrs) III: shock & lactic acidosis (6-72 hrs) IV: hepatotoxicity/necrosis (12-96 hrs) V: GI scarring & gatric outlet obstruction (2-8 wks) Tx: whole bowel irrigation, IVF, deferoxamine (indications: level >500 mcg/dL OR >300 mcg/dL and symptomatic)</p>
<p>Sources, Pathophysiology, Signs & Symptoms, Diagnosis, Treatment: Lead poisoning</p>	<p>Sources: paint, old batteries, occupational exposure SSx: microcytic anemia with basophilic stippling, abd pain, AMS, seizure, encephalopathy Dx: peripheral smear, whole blood lead level, xray showing lead lines Tx: chelation with PO succimer (only used in kids) or IM dimercaprol followed by IV EDTA Chelate levels >45 if symptomatic or >70 if asymptomatic.</p>
<p>How and when to treat a child with lead toxicity?</p>	<p>No level is safe! ASYMPTOMATIC child w/ lead level: - 5 to 44 mcg/dl: remove the source, education, get public health involved - 45 to 69 mcg/dl: oral chelation therapy - 70 mcg/dl or higher: hospitalize for chelation therapy OR 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**</p>
<p>Pathophysiology, Signs & Symptoms, Treatment: Isoniazid (INH) overdose</p>	<p>Pathophysiology: inhibits pyridoxine (B6) SSx: hepatotoxicity, metabolic acidosis, seizures/status epilepticus Tx: IV pyridoxine (1 g of B6 for every 1 g of INH)</p>
<p>Cause, Signs & Symptoms, Treatment: Lithium toxicity</p>	<p>Cause: often occurs 2/2 interactions with medications that affect renal function (NSAIDs, diuretics, ACEi) SSx: GI sx (acute), neuro sx (chronic), nephrogenic diabetes insipidus Tx: IVF, whole bowel irrigation, hemodialysis (indications: renal failure, level >5, severe neuro sx)</p>

What rare but severe condition can be precipitated by abruptly stopping lithium ?	Thyroid storm: 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 general timing of symptoms in mushroom ingestions help predict prognosis?	If symptoms (n/v/d) start within 6 hr of ingestion → likely non-toxic If symptoms start after 6 hr → hepatotoxicity may occur
Signs & Symptoms, Treatment: Cyclopeptide mushroom ingestion (<i>Amanita</i> , <i>Galerina</i> , <i>Lepiota</i>)	SSx: delayed GI sx (usually > 6 hrs) followed by liver failure , renal failure, AMS, and death Tx: supportive care, multidose charcoal during first 24 hours (speeds up enterohepatic clearance), GI decontamination, NAC
Signs & Symptoms, Treatment: Monomethylhydrazine mushroom ingestion (<i>Gyromitra</i> - "false morel")	SSx: delayed GI sx (> 6 hrs) followed by seizures (think 'gri' like brain), hepatorenal failure. Most have full recovery. Tx: supportive, benzodiazepines, B6 for seizures (can be refractory)
Signs & Symptoms, Treatment: Muscarine mushroom ingestion (<i>Inocybe</i> , <i>Clitocybe</i>)	SSx: muscarinic symptoms (cholinergic/"DUMBBBELLS") Tx: atropine, 2-PAM
Signs & Symptoms, Treatment: Psilocybin mushrooms ("magic mushrooms" - <i>Psilocybe</i> , <i>Conocybe</i> , <i>Gymnopilus</i> , <i>Panaeolus</i>)	SSx: hallucinations , euphoria, agitation Tx: benzodiazepines
What is the key clinical effect of Coprin mushrooms (inky caps)?	Disulfiram-like reaction
Signs & Symptoms: Phenytoin toxicity (PO and IV)	PO: gingival hyperplasia, seizure uncommonly, no cardiac effect with PO IV: hypotension (2/2 propylene glycol) , can give fosphenytoin instead
Pathophysiology, Signs & Symptoms, Treatment: Carbamazepine toxicity	Pathophysiology: Na channel blockade , anticholinergic effects SSx: ataxia, GI sx, QRS widening, seizure at high doses Tx: supportive, sodium bicarb if wide QRS
Pathophysiology, Signs & Symptoms, Treatment: Benzodiazepine overdose	Pathophysiology: GABA agonist , increases <u>frequency</u> of channel opening SSx: ataxia (MC sign), lethargy, respiratory depression Tx: supportive, intubate PRN Flumazenil is the reversal agent, but generally not given; precipitate withdrawal in patients with benzo or EtOH dependence.
Pathophysiology, Signs & Symptoms, Treatment: Barbiturate overdose	Pathophysiology: GABA agonist , increases <u>duration</u> of channel opening SSx: hypotension , bradycardia, respiratory depression, rhabdo ("barb blisters") Tx: supportive, intubate PRN
What ingestions are radiopaque on X-ray?	CHIPES: C hloral hydrate, H heavy metals, I ron/Iodine, P henothiazine, E nteric-coated, S olvents

Signs & Symptoms, Treatment: Antipsychotic use	SSx: AMS, lead pipe rigidity , hyperthermia , autonomic instability. Tx: supportive (IVF, benzodiazepines, cooling), Bromocriptine (dopamine agonist)
Signs & Symptoms, Treatment: Serotonin Syndrome	SSx: AMS, clonus/hyperreflexia , hyperthermia Tx: supportive (IVF, benzodiazepines, cooling) ± Cyproheptadine
Association, Pathophysiology, Signs & Symptoms, Treatment: Strychnine poisoning	Associations: gopher poison, adulterant in heroin Pathophysiology: inhibits glycine (similar to tetanus) SSx: agitation, myoclonus, severe and painful muscle contractions, rhabdo, seizures, “awake seizures” Tx: IVF, benzodiazepines, paralysis PRN
Pathophysiology, Signs & Symptoms, Treatment: Sulfonylurea overdose (e.g., glipizide, glyburide)	Pathophysiology: stimulation of pancreatic insulin release SSx: sulfonylureas are long-acting → severe recurrent hypoglycemia Tx: dextrose (IVP ± drip), octreotide (inhibits release of insulin), admit for monitoring
Signs & Symptoms, Treatment: Insulin overdose	SSx: hypoglycemia Note: Duration of effect and timeframe for monitoring are determined by the specific insulin formulation and its half life. Tx: glucagon, dextrose PRN
What are common toxic causes of hypoglycemia ?	Ethanol (especially in kids) , insulin/ hypoglycemics (NOT metformin), beta blockers, salicylates, quinine
Pathophysiology, Treatment: Metformin overdose	Pathophysiology: inhibition of gluconeogenesis → reduces hepatic glucose output → converts glucose to lactic acid → lactic acidosis Tx: bicarb, lasix (increases excretion), hemodialysis PRN (clears metformin and acidosis)
Pathophysiology, Signs & Symptoms, Treatment: Theophylline toxicity	Pathophysiology: Methylxanthine derivative (like caffeine) & beta agonist, metabolized by hepatic CYP450 enzymes (many drug interactions) SSx: hypotension, dysrhythmia (MAT = classic) , seizures Tx: IVF, beta blocker, consider hemodialysis in severe intoxication
Signs & Symptoms, Treatment: Hydrogen peroxide ingestion	SSx: stroke-like sx (cerebral gas embolism) Tx: hyperbaric oxygen
Antidote for acetaminophen toxicity?	N-acetylcysteine (NAC)
Antidote for aspirin toxicity?	Bicarb, hemodialysis
Antidote for beta blocker toxicity?	Glucagon, high dose insulin + dextrose
Antidote for calcium channel blocker toxicity?	Calcium, insulin + dextrose, high-dose pressors
Antidote for carbon monoxide toxicity?	Oxygen (hyperbaric O2 for severe toxicity)
Antidote for cyanide toxicity?	Hydroxocobalamin OR Dual therapy (sodium nitrite/amyl nitrate and sodium thiosulfate)

Antidote for digoxin toxicity?	Digoxin Fab Fragments
Antidote for ethylene glycol and methanol toxicity?	Ethanol or fomepizole , hemodialysis
Antidote for benzodiazepine toxicity?	Flumazenil (rarely used; can precipitate withdrawal sz)
Antidote for opioid toxicity?	Naloxone
Antidote for malignant hyperthermia ?	Dantrolene, benzodiazepines, bicarb
Antidote for serotonin syndrome?	Cyproheptadine, benzodiazepines
Antidote for neuroleptic malignant syndrome ?	Dantrolene, bromocriptine, benzodiazepines
Antidote for anticholinergic syndrome ?	Physostigmine, benzodiazepines
Antidote for iron toxicity?	Deferoxamine
Antidote for mercury toxicity?	Dimercaprol OR Dimercaptosuccinic acid (succimer)
Antidote for lead toxicity?	PO dimercaptosuccinic acid (succimer) OR Dimercaprol (IM) and EDTA (IV)
Antidote for isoniazid toxicity?	Vitamin B6 (pyridoxine)
Antidote for organophosphate toxicity?	Atropine, 2-PAM
Antidote for valproic acid overdose?	L-Carnitine
What treatments for (1) hyperkalemia and (2) bradycardia are classically contraindicated in digoxin toxicity ?	(1) Hyperkalemia: don't give calcium - rare risk of "stone heart" (this is debunked but still on boards). (2) 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
Signs & Symptoms, Treatment: Opioid withdrawal	SSx: tachycardia, HTN, abd pain, N/V/D, sweating, agitation, dilated pupils, piloerection, yawning Tx: antiemetics, clonidine, fluids, buprenorphine (more complex decision) Note: opioid withdrawal is NOT life-threatening in adults.
Signs & Symptoms, Diagnosis, Treatment: Valproic acid toxicity	SSx: GI distress, AMS Dx: high valproic acid level, high serum ammonia Tx: activated charcoal, L-carnitine , hemodialysis (if renal failure)
What is body packing ? How do you treat these patients?	Body packing - intentionally swallowing prepared packets of recreational drugs Tx (stable/asymptomatic): cardiac monitoring + polyethylene glycol Tx (unstable): surgery and supportive care
Toxin that smells like garlic ?	Arsenic or organophosphates
Toxin that smells like burnt almonds ?	Cyanide
Toxin that smells like fish ?	Zinc
Toxin that smells like fruit ?	Isopropanol, ethanol
Toxin that smells like rotten eggs ?	Sulfur -containing compounds
Treatment: Severe hydrofluoric acid exposure	Topical and intra-ARTERIAL calcium gluconate

<p>Associations, Signs & Symptoms, Treatment: Phosgene toxicity</p>	<p>Phosgene is a colorless gas associated with plastics, dyes, pesticides. SSx: minimal conjunctival and pulmonary irritation; leads to DELAYED noncardiogenic pulmonary edema Tx: admission and supportive care (intubation, ventilation); no antidote</p>
<p>Toxin that smells like freshly cut hay or grass?</p>	<p>Phosgene</p>
<p>Diagnosis: Chemical ingestion associated with severe hypocalcemia and hypomagnesemia</p>	<p>Hydrofluoric acid</p>
<p>Diagnosis, Treatment: Cyanosis and hypoxia after using "poppers" at a club</p>	<p>Dx: "poppers" commonly contain amyl nitrite, which induces Methb Tx: methylene blue, 100% O₂</p>

Eye, Ear, Nose & Throat

Bizz	Buzz
Diagnosis, Treatment: Blepharitis	Dx: inflammation of the eyelid Tx: wash with gentle soap, topical antibiotics (<i>Strep/Staph</i>)
What is a hordeolum ? How is it treated?	Dx: acute painful blockage and infection of gland (Zeis = sebaceous or Moll = sweat) at or near the eyelash follicle Tx: warm compresses
What is a chalazion ? How is it treated?	Dx: chronic or gradual-onset nontender granuloma due to blockage of meibomian gland (above eyelash or on upper lid) Tx: warm compresses , ophtho excision (if recurrent/persistent)
What is the difference a hordeolum and a stye ?	None (they are the same)
Cause, Signs & Symptoms, Diagnosis, Treatment: Iritis, Uveitis, Choroiditis	Cause: inflammation of the iris, ciliary body, choroid (respectively) SSx: painful red eye, photophobia, decreased visual acuity Dx: cell and flare or ciliary flush on slit lamp exam Tx: ophtho consult, dilate, steroids, pain meds
Cause, Signs & Symptoms, Diagnosis, Treatment: Dacryocystitis	Cause: infection of the nasolacrimal gland (tear duct), most common organism = S. aureus SSx: swelling, purulent discharge, possible adjacent cellulitis Dx: clinical exam Tx: warm compresses, antibiotics, ophtho f/u
Signs & Symptoms, Diagnosis, Treatment: HSV keratitis	SSx: pain, photophobia Dx: dendritic pattern on blue light Tx: ophtho consult, topical trifluridine +/- oral acyclovir
In bacterial conjunctivitis, contact lens use is a risk factor for which organism? What is the treatment?	Risk for <i>Pseudomonas</i> infection Tx: fluoroquinolone drops (increasing resistance) or Tobramycin
Diagnosis, Treatment: Metal worker + eye pain	Dx: Intraocular foreign body Tx: look for teardrop pupil and/or Seidel's sign to r/o globe rupture, remove foreign body, rust rings removed in 24-48 hours
Signs & Symptoms, Diagnosis, Treatment: Skier or welder + eye pain	SSx: bilateral decreased visual acuity, pain, and redness Dx: UV keratitis ; exam shows multiple punctate lesions Tx: analgesia, cycloplegics, eye rest
Diagnosis, Treatment: Hyphema	Dx: bleeding in the anterior chamber of the eye (often traumatic) Tx: consult ophtho, bedrest, HOB elevation, pain meds, cycloplegics, discontinue any anticoagulation *More urgent ophtho consult needed for patients with sickle cell disease *
What are two potential complications of hyphema ?	Rebleed (most common complication), risk for glaucoma

<p>Cause, Signs & Symptoms, Diagnosis, Treatment: Endophthalmitis</p>	<p>Cause: infection of anterior, posterior and vitreous chambers of the eye, often d/t iatrogenic etiology (cataract surgery = MCC in US) or trauma SSx: severe pain & visual impairment Dx: exam with decreased visual acuity, injected conjunctiva, chemosis, possible hypopyon (pus in the anterior chamber) Tx: ophtho consult, intraocular + systemic antibiotics</p>
<p>What is the risk of laceration close to the medial canthus? How should it be evaluated and repaired?</p>	<p>High risk for lacrimal duct injury. Evaluate with fluorescein staining. Should be repaired by oculoplastics.</p>
<p>Treatment: Acute angle closure glaucoma</p>	<p>1. Decrease intraocular pressure (consider mannitol or hypertonic saline). 2. Constrict the pupil to promote drainage of aqueous fluid. ↓ aqueous production: α-agonist (apraclonidine), β-blocker (timolol), carbonic anhydrase inhibitor (acetazolamide) ↑ outflow: pilocarpine (miotic)</p>
<p>Diagnosis: Sudden painless unilateral vision loss, retina with "box-cars" or "cherry-red macula"</p>	<p>Central retinal artery occlusion (stroke equivalent)</p>
<p>Diagnosis: Sudden painless unilateral vision loss, retina with "blood and thunder" appearance (dilated retinal veins, diffuse hemorrhage, cotton wool spots)</p>	<p>Central retinal vein occlusion (increased risk with chronic glaucoma)</p>
<p>Diagnosis, Treatment: Painless unilateral loss of vision with floaters and visual field cuts</p>	<p>Dx: retinal detachment - clinical diagnosis, though often visible on ocular ultrasound SSx: classically described as a "curtain coming down" over the visual field or as "floaters and flashes of light" Tx: ophthalmology consult</p>
<p>Diagnosis, Treatment: Painful red eye, decreased visual acuity, ciliary flush, "cell and flare" on slit lamp</p>	<p>Dx: Iritis or anterior uveitis Tx: Cycloplegics to dilate (e.g. cyclopentolate, tropicamide), pain control, ophtho consult or follow up w/n 24 hrs</p>
<p>Diagnosis: Dizzy + vertical, multidirectional, or non-fatigable nystagmus</p>	<p>Central vertigo</p>
<p>Differential diagnosis: Gingival hyperplasia</p>	<p>Acute necrotizing ulcerative gingivitis, HIV, phenytoin toxicity, acute leukemia</p>
<p>Diagnosis: White plaques on oral mucosa, can be scraped off</p>	<p>Candida, associated with immunocompromise, abx use, diabetes, oral steroid inhalers</p>
<p>What are the Centor criteria for acute bacterial pharyngitis? What score indicates the need for empiric treatment?</p>	<p>1. Fever 2. Tender anterior lymphadenopathy 3. Lack of cough 4. Tonsillar exudates 4/4 → empiric abx 3/4 → culture/rapid strep</p>
<p>What medication is most likely to improve symptoms of viral pharyngitis?</p>	<p>Dexamethasone</p>

<p>Associations, Cause, Signs & Symptoms, Diagnosis, Treatment: Peritonsillar abscess</p>	<p>Association: complication of acute tonsillitis, most common deep facial infxn in adults Cause: Strep (GAS) = most common SSx: sore throat, odynophagia, muffled voice, referred otalgia. Dx: exam w/ trismus, deviation of uvula + soft palate Tx: needle aspiration, abx (augmentin vs clindamycin), ENT follow up</p>
<p>Diagnosis, Cause, Treatment: Fever, sore throat, brawny neck edema, tongue elevation, dysphagia, drooling</p>	<p>Dx: Ludwig's Angina Cause: dental infection = MCC, immunodeficiency Tx: airway management, ENT consult, broad spectrum abx</p>
<p>What is the most common cause of death in a patient with Ludwig's Angina?</p>	<p>Sudden asphyxiation (laryngospasm)</p>
<p>What are the three Ellis classifications of dental fracture? What is the exam and appropriate management for each?</p>	<p>Ellis I: fracture of enamel; tooth appears white and painless; treatment = smooth rough edges, dental follow-up Ellis II: fracture through dentin & enamel; exposed tooth appears yellow and painful; treatment = smooth rough edges, apply calcium hydroxy paste, dental follow-up Ellis III: fracture through pulp, dentin & enamel; exposed tooth appears pink/red and painful; treatment = cover with moist cotton & dental foil, give antibiotics (pencillin), and obtain emergent dental consult</p>
<p>Diagnosis, Signs & Symptoms, Treatment: Dry socket (alveolar osteitis)</p>	<p>Dx: localized osteomyelitis due to loss of protective clot; exam with exposed bone SSx: severe pain 3-5 days after dental extraction Tx: irrigate with saline, iodoform gauze, eugenol (oil of clove), abx if needed (signs of infection), PAIN CONTROL, oral surgery referral</p>
<p>Signs & Symptoms, Diagnosis, Treatment: CMV retinitis</p>	<p>SSx: ↓ visual acuity, floaters/visual field cuts, photophobia Dx: white fluffy retinal perivascular lesions with hemorrhage, CD4 <50 (AIDS defining illness) Tx: IV ganciclovir</p>
<p>Compare/contrast the appearance of vitreous versus retinal detachment on ultrasound.</p>	<p>BOTH show serpiginous structure within the globe Vitreous detachment: can cross over the optic nerve Retinal detachment: will NEVER cross the optic nerve</p>
<p>What is an afferent pupillary defect? What does it indicate?</p>	<p>Dx: "swinging flashlight test" performed by moving a light from one eye to the other; affected eye will dilate when light is shined directly into it (cannot sense light but can constrict as a consensual response) Indicates a lesion of the retina or optic nerve.</p>
<p>Name 4 possible causes of afferent pupillary defect.</p>	<p>CRAO, CRVO, optic neuritis, and retrobulbar neuritis</p>
<p>Diagnosis, Treatment: Monocular vision loss, worse centrally, afferent pupillary defect, pain with extraocular movements, diminished color vision</p>	<p>Dx: Optic neuritis Causes: Idiopathic > multiple sclerosis (MS) > toxicological (methanol, ethambutol) or infectious (herpes zoster virus) Tx: Consult neurology & ophtho, IV steroids, MRI (for evidence of MS)</p>

Treatment: Central retinal artery occlusion	↓ IOP (acetazolamide, mannitol, timolol), vasodilators (nitro), may also see TPA as an option (per AHA guidelines)
Compare/contrast the etiology, exam findings, and treatment of pinguecula vs. pterygium .	Pinguecula: degenerative eye lesion 2/2 chronic inflammation from wind and UV light; exam shows yellow raised fleshy conjunctival mass (lateral) ; no treatment Pterygium: slow growing thickening of conjunctiva 2/2 wind/sand/dust; exam shows vascular triangular mass in "bat wing" shape (medial) ; surgical treatment if affecting vision
What should be the approach to stopping anterior epistaxis ?	Direct pressure , vasoconstrictors (oxymetazoline), silver nitrate cautery (unilateral), balloon tamponade device Send home with ENT f/u in 2-3 days. Note: Use of abx is controversial.
What are the most common sources of anterior and posterior epistaxis ?	Anterior: Kiesselbach plexus Posterior: sphenopalatine artery
What is the appropriate treatment and disposition for patients with posterior nasal packing?	Prophylactic antibiotics + admit to ICU/monitored bed d/t risk of vagally-mediated bradycardia and airway compromise
Most common site of sialadenitis? Treatment?	Most common site: submandibular gland (Wharton duct) Tx: milk stone, sialogogues , sour candy, abx if abscess
Diagnosis, Exam, Treatment: Sudden onset vertigo, worse with change in head position, no other neurologic symptoms	Dx: Benign Paroxysmal Positional Vertigo (BPPV) Exam: Dix Hallpike maneuver (look for unidirectional nystagmus) Tx: Epley maneuver, meclizine
Diagnosis, Cause, Treatment: Episodic peripheral vertigo + hearing loss + tinnitus	Dx: Meniere disease aka "idiopathic endolymphatic hydrops" Cause: increased endolymph in the inner ear Tx: treat vertigo symptomatically, diuretics, refer to ENT
Diagnosis, Treatment: Severe vertigo + URI	Dx: vestibular neuritis Tx: supportive (will resolve w/o intervention)
Diagnosis, Treatment: Severe vertigo + acute otitis media (plus or minus hearing loss)	Dx: Labrynthitis Tx: Suppurative labyrinthitis (caused by extension of bacterial infxn) should be treated with intravenous antibiotics and ENT referral.
How does vestibular neuritis differ from Ménière disease ?	Vestibular Neuritis: SSx: recent URI, vertigo (NOT recurrent) + hearing loss + unstable gait Tx: steroids, self limited Ménière: SSx: episodic vertigo + hearing loss + tinnitus Tx: avoid triggers, antihistamines, diuretics, benzodiazepines, surgery (refractory cases)
What potential causes are indicated by a positive head impulse test (i.e. presence of corrective saccade) in a patient with continuous vertigo?	Peripheral causes (vestibular neuritis or labyrinthitis) If NO corrective saccade >> central cause

<p>Etiology, Signs & Symptoms, Treatment: Perichondritis</p>	<p>Etiology: infection of the cartilage; presents after ear surgery, trauma, or upper ear piercing; <i>pseudomonas</i>, staph/strep spp. SSx: swollen, warm, tender, erythematous auricle; no TM or earlobe involvement Tx: fluoroquinolone (e.g. cipro); admit for IV coverage if severe.</p>
<p>Etiology, Signs & Symptoms, Diagnosis, Treatment, Complications: Acute otitis media</p>	<p>Etiology: viral (RSV) > bacterial (<i>Strep. Pneumo</i>) SSx: ear pain, fever, URI symptoms Dx: TM bulging/erythema & decreased mobility of TM (most sensitive) Tx: amoxicillin (simple); augmentin (recurrent/persistent) Complications: hearing loss, perforation (add abx in suspension), facial nerve paralysis (needs myringotomy)</p>
<p>Mechanism, Signs & Symptoms, Diagnosis, Treatment: Acute mastoiditis</p>	<p>Mechanism: Bacterial infection of mastoid air cells most often d/t direct extension from AOM MCC: <i>Strep. Pneumo</i> SSx: postauricular erythema & tenderness, protrusion of the auricle Dx: CT temporal bone Tx: abx, surgical drainage Complications: osteomyelitis, intracranial infxn, venous sinus thrombosis</p>
<p>Etiology: Signs & Symptoms, Diagnosis, Treatment: Necrotizing (malignant) otitis externa</p>	<p>Etiology: necrotizing infection of auditory canal + skull base (a form of osteomyelitis); risk factors = elderly, diabetes; MCC = <i>Pseudomonas</i> SSx: otorrhea, otalgia, severe/persistent pain, CN VII palsy Dx: CT temporal bone Tx: ciprofloxacin (outpatient), antipseudomonal β-lactam + aminoglycoside (inpatient) Complication: CN VII palsy, intracranial infxns</p>
<p>Diagnosis, Treatment: Sudden pain + decreased hearing after ear irrigation</p>	<p>Dx: TM perforation Tx: pain control, keep ear dry, <u>antibiotics only if concurrent infection</u>, ENT follow-up in 1-2 wks Overall, infection is the MCC of TM perforation.</p>
<p>Diagnosis, Etiology, Treatment: Pain/swelling/tenderness over parotid gland + fever, trismus, dysphagia</p>	<p>Dx: Suppurative (bacterial) parotitis <i>Staph aureus</i> = MCC Tx: abx (ampicillin-sulbactam)</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Preseptal cellulitis</p>	<p>SSx: eyelid swelling WITHOUT painful EOM or proptosis Dx: infection of the anterior portion of the eye (NOT orbital structures); clinical diagnosis but obtain CT orbits if concerned for orbital cellulitis MCC = staph/strep spp. Tx: amoxicillin-clavulanate, outpatient ophtho follow-up</p>

<p>Presentation, Signs & Symptoms, Diagnosis, Treatment: Orbital cellulitis</p>	<p>Presentation: infection of the contents of the orbit (posterior to orbital septum); children > adults MCC: bacterial rhinosinusitis SSx: eyelid swelling, deep eye pain, pain + limitation with eye movements, proptosis Dx: CT orbits Tx: broad spectrum abx (vancomycin + piperacillin-tazobactam), ophtho consult, <u>admit</u> Complications: vision loss, cavernous sinus thrombosis, meningitis</p>
<p>What structures travel through the cavernous sinus? What is the most common cranial nerve palsy associated with cavernous sinus thrombosis?</p>	<p>CN III, IV, V1, V2, VI Internal carotid artery Most common CN palsy = isolated CN VI (abducens) palsy</p>
<p>Association, Signs & Symptoms, Diagnosis, Treatment: Cavernous sinus thrombosis</p>	<p>Association: classically follows acute bacterial sinusitis; staph = MCC SSx: headache (MC sx) + fever + CN palsies (MC is CN VI) + periorbital edema Dx: magnetic resonance venography Tx: IV abx (nafcillin, ceftriaxone, and metronidazole); ENT consult; ICU admit Anticoagulation is controversial.</p>
<p>Diagnosis: Patient with recent tracheostomy placement + brisk bleeding from the tracheostomy site</p>	<p>Tracheoinnominate artery fistula (TIF)</p>
<p>Management: Tracheoinnominate fistula</p>	<p>Needs ENT consult for emergent OR management. In the interim: overinflate trach cuff, visualize the source of bleeding, apply direct pressure (digital pressure in the trach stoma against the sternum), intubate from above (unless s/p laryngectomy).</p>
<p>Etiology, Signs & Symptoms, Diagnosis, Treatment: Vitreous hemorrhage</p>	<p>Etiology: trauma, diabetic retinopathy, retinal detachment, posterior vitreous detachment SSx: blurry or tinted (reddish) vision, floaters/fashers Dx: ocular US shows hyperechoic opacities in vitreous chamber Tx: ophtho consult w/n 24-48 hrs, avoid NSAIDs/anticoagulants, elevate head of bed</p>
<p>Is alkali or acidic injury more dangerous to the eye? How is each treated?</p>	<p>Alkali injury is more dangerous (leads to liquefaction necrosis). Treatment is the same: copious saline irrigation to a normal pH (7.4), ophtho consult</p>
<p>Diagnosis, Treatment: Globe rupture</p>	<p>Dx: vision loss, teardrop pupil, flattened anterior chamber, Seidel's sign is present DO NOT CHECK PRESSURES! Tx: antibiotics, CT orbit, cover with eye patch, emergent ophtho consult</p>

What medication is contraindicated in sickle cell patients presenting with hyphema ?	Acetazolamide (induces sickling of RBCs)
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Obstetrics & Gynecology

Bizz	Buzz
What size ovarian cyst is high risk for torsion?	5-10 cm
What is the cause of injury in ovarian torsion ?	Twisting leading to obstruction of VENOUS and lymphatic flow → congestion → edema and ischemia Arterial obstruction is rare 2/2 dual blood supply.
What is the most common finding on US with ovarian torsion ?	MC: Ovarian enlargement Other findings: loss of echogenicity, pelvic free fluid, diminished blood flow
What is the most common type of ovarian cyst ?	Simple follicular cysts that are thin walled and fluid filled. Present in first 2 weeks of the menstrual cycle.
What is the most likely type of ovarian cyst to bleed ?	Corpus luteal cyst Present in the last 2 weeks of the menstrual cycle. It 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)
Diagnosis: Vaginal bleeding in postmenopausal woman	Gynecologic cancer until proven otherwise Will need outpatient TVUS and endometrial biopsy .
What type of cancer is CA-125 a marker for?	Ovarian
Classic presentations for ovarian, endometrial, and cervical cancers	Ovarian: age 50s-60s, gradual subacute abd pain, abdominal distention (ascites), CA-125+ Endometrial: vaginal bleeding in postmenopausal women Cervical: h/o HPV or HIV, postcoital bleeding, abnormal cervix on pelvic exam
What is the age range for administering the HPV vaccine ?	9-26 yrs
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 age 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 are potential medications for non-pregnant vaginal bleeding ?	Mild/Moderate: combined OCP's, Progesterone, NSAIDs Severe: IV premarin (estrogen), TXA

Diagnosis, Treatment: Middle aged female with "ball coming out of vagina"	Uterine prolapse or cystocele Worse with valsalva Tx: 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: excessive flow (heavy periods) Metrorrhagia: irregular cycles
Diagnosis, Treatment: Abdominal pain and tenderness, vaginal discharge, cervical motion tenderness	Pelvic inflammatory disease (PID) Cause: C. trachomatis (MCC) , N. gonorrhoeae, polymicrobial Dx: consider pelvic US to r/o TOA Tx: Ceftriaxone IM 500mg, 14 days of doxycycline (azithro 1g not recommended for PID) and metronidazole **Note: They won't always have CMT, but may just have focal uterine or adnexal pain.
Diagnosis, Treatment: PID with RUQ and shoulder pain	Fitz-Hugh-Curtis syndrome (perihepatitis): Infection to perihepatic space causing liver capsule inflammation and adhesions MCC: Chlamydia Dx: TVUS (if concerned for TOA). CT: ±"violin-string" adhesions (usually normal LFTs) Tx: 500mg ceftriaxone IM and 14 days of doxycycline and metronidazole (just like PID)
Most common GYN problem in children	Vulvovaginitis
Diagnosis, Treatment: Vaginal discharge and clue cells	Bacterial vaginosis Cause: <i>Gardnerella/anaerobes</i> Dx: <u>Amsel criteria (3 out of 4 means positive test): thin, white d/c, "clue cells", vaginal pH >4.5, fishy odor (+whiff test)</u> Tx: metronidazole (including if pregnant)
Diagnosis, Treatment: Vaginal discharge and pseudohyphae on wet mount	Candidal vaginitis: overgrowth of normal flora, causing pruritus Dx: "cottage cheese" discharge, +wet prep Tx: fluconazole x1 week . If pregnant, intravaginal Clotrimazole or Miconazole.
What other conditions are associated with frequent yeast infections ?	Diabetes, HIV, pregnancy, antibiotics, steroids, SGLT2 inhibitors (-flozins)
Signs & Symptoms, Diagnosis, Treatment: Vaginal discharge and "strawberry cervix"	Trichomoniasis: protozoal infection SSx: "frothy yellow-green" discharge Dx: pH >5, WBCs, motile trichomonads Tx: PO metronidazole for patient and partner
What is the definitive treatment for Bartholin's cyst/abscess ?	Marsupialization In ED: I&D of abscess, place Word catheter
Diagnosis, Treatment: Early pregnancy with big uterus and high hCG	Hydatidiform mole: painless vaginal bleeding, uterus bigger than dates, hyperemesis, preeclampsia Dx: hCG >100,000 ; US: "grape-like vesicles", "snowstorm" Tx: D&C Complication: high risk of malignancy (choriocarcinoma)

What is the ultrasonographic and prognostic difference between partial and complete hydatidiform moles ?	Partial: nonviable fetus, <5% become malignant, triploid karyotype Complete: "snowstorm" appearance on US, 20% become malignant, diploid karyotype
Treatment: Asymptomatic bacteriuria in pregnancy	Treat with ABX (otherwise high risk of pyelonephritis) ABX: Keflex, macrobid
What is the most common side for pyelonephritis in pregnancy ?	Right sided (70-80%) Pregnant women are much more likely to have pyelonephritis than non-pregnant women.
Treatment: pregnant woman with pyelonephritis	IV ABX and admission
Define components of labor progression: dilation, effacement, station	Dilation: opening of cervical os, up to 10 cm Effacement: thinning of the cervix, up to 100% Station: fetal presenting part location, ranges neg (above) to positive (below) cm relative to ischial spines (0)
Define: 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
What is the duration of stage 1, 2, and 3 of labor ?	First stage- primips: 6-20 hrs; multiparous: 2-14 hrs Second stage- primips: 30 mins-3 hrs; multiparous: 5-60 mins Third stage- everyone: 0-30 mins.
What is the concern with late decelerations on tocodynamometer monitoring during labor?	Uteroplacental insufficiency → move to c-section
What is the concern with variable decelerations on tocodynamometer monitoring during labor?	Cord compression
What do early decelerations on tocodynamometer monitoring during labor indicate?	Fetal head compression (not concerning)
Review the initial management of low FHR on tocodynamometer monitoring	Change mom's position (left lateral is best to move uterus off of IVC), 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
Apgar scoring	Max 10pts measured, 0-2 pts for each Appearance (pink, acrocyanosis, central cyanosis) Pulse (>100 bpm, <100 bpm, 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. Call for OB/neonatology/anesthesia. 1st line (and most tested): McRobert's Maneuver (hyperflex hips), suprapubic pressure. Next steps: Rubin/Woods rotational maneuvers, deliver posterior arm, episiotomy, empty mother's bladder with foley. If all of these have failed, manually break newborn's clavicle, C-section.
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
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). Keep the presenting part elevated while the patient is moved to the OR for c-section.
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 500 cc for vaginal birth or 1 L for c-section
What are the most common causes of postpartum hemorrhage based on timing of presentation (< 24 h or > 24 h)?	Early (< 24 hr): uterine atony (most common) , retained POC, lacerations Late (> 24 hr): retained POC, lacerations
What is the appropriate management of postpartum bleeding 2/2 uterine atony, lacerations, and retained products , respectively?	Atony: MCC < 24hrs; Tx: bimanual massage, oxytocin, IVF, NO MAG Lacerations: birth trauma = 2nd MCC. Tx: surgical repair Retained products: early or late bleeding; Dx: US; Tx: surgical removal All: transfuse as needed
What is the incidence of postpartum depression ?	Up to 50% , overall underdiagnosed
Signs & Symptoms, Diagnosis, Treatment: Uterine rupture	Higher risk: trauma, previous c-section or trauma SSx: fetal distress, palpation of fetal parts, loss of uterine tone, shock Dx: US, nonreassuring FHR = most reliable sign Tx: emergency C-section (and likely hysterectomy)

Signs & Symptoms, Treatment: Fever and abdominal pain 2-3 d postpartum	Endometritis (infxn of decidua) Polymicrobial MC postpartum infxn Risks: 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
What is the most common cause of third trimester vaginal bleeding ?	Abruptio placentae: premature separation of placenta
What can increase the risk of placental abruption ?	HTN (MCC) , preeclampsia (most significant risk factor), sympathomimetics (cocaine , meth), trauma, high parity, smoking, heavy EtOH, advanced maternal age
Signs & Symptoms, Diagnosis, Treatment: Placental abruption	SSx: (TRIAD): vaginal bleeding (**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) Tx: fetal monitoring, Rho(D) immunoglobulin, stabilize mother (IVF, blood), non-reassuring mother/fetus = c-section, term+stable = delivery (vaginal or OR), preterm+stable = inpt conservative mgmt
What is the most sensitive test for predicting placental abruption ? What is the most specific test for predicting placental abruption ?	Most sensitive: Tocodynamometer monitoring Most specific: ultrasound but can miss placental abruption especially if there is a retroplacental clot. If you think they're abructing, call OB and start toco
What's the best way to determine fetal distress after trauma ?	Tocography. Monitor all viable (> 20 wk) women for 4 hrs. Okay to go home: If there are less than 3 contractions per hour for four hours, no late decelerations, or baby bradycardia
Most common cause of painless vaginal bleeding during third trimester?	Placenta previa: placenta partially or completely covering cervical os, which causes bleeding when the os starts to dilate. **No digital or speculum exams until previa ruled out by US
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 20 wks will resolve spontaneously ?	50%
What distinguishes PROM and PPROM ?	Premature rupture of membranes (PROM): rupture of membranes before onset of labor > 37 wks Preterm PROM (PPROM): PROM < 37 wks
What are potential complications of PROM ?	Infection (chorioamnionitis), cord prolapse

What are methods to confirm rupture of membranes ?	Clinical: "gush" of fluid, pooling of amniotic fluid in vaginal fornix **least specific** Nitrazine paper: pH > 7 turns paper blue , has high false positive rate from using lubricant on speculum, sperm, trichomonas infection) Ferning test: dried secretions will show branching pattern of crystallization (ferning = amniotic fluid) **most specific**
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 ?	Premature/Preterm labor: contractions + cervical changes < 37 wks Tx: Tocolytics (Mag [IV 4-6g then infusion], indomethacin, nifedipine, 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 if 24-34 weeks.
What medications are typically used (and safe) in pregnancy for HTN ?	Alpha-methyldopa, labetalol, hydralazine, nifedipine
What distinguishes chronic HTN vs. pregnancy-induced HTN vs. preeclampsia/eclampsia ?	Chronic HTN: onset prior to pregnancy or before 20 wks Gestational HTN: onset > 20 wks but no sx Preeclampsia/eclampsia (vascular endothelial dysfunction): HTN > 20 wks (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 ?	20 wks gestation until 6 wks postpartum
What are risk factors for preeclampsia/eclampsia ?	First pregnancy, < 20y/o or > 35y/o, multiple gestation (e.g. twins), HTN, DM
What defines mild preeclampsia vs. severe preeclampsia vs. eclampsia ?	Mild: BP 140-160/90-110 , proteinuria > 300 mg/24hr but < 5g/24hr. Severe: BP >160-180 or >110 diastolic on 2 occasions 6 hrs apart, proteinuria >5g/24hr (or Udip 4+ protein), Cr > 1.1, LFTs 2x normal, pulmonary edema cerebral/visual sx. Eclampsia: preeclampsia + seizures
What are clinical symptoms for severe preeclampsia ?	Headache, blurred vision, RUQ pain, clonus Can progress to HELLP syndrome.
What is the appropriate treatment for severe preeclampsia or eclampsia ?	Emergent delivery , hydralazine/labetalol/nifedipine for BP control , steroids if < 36 wks (fetal lung development), IV Mg sulfate (4-6 g) to treat/prevent seizures
Signs & Symptoms, Treatment: Magnesium toxicity	SSx: neurotoxic (loss of DTRs , respiratory failure, asystole) Tx: IVF, calcium gluconate/chloride

What defines HELLP syndrome and how is it treated?	Hemolysis, Elevated Liver enzymes, Low Platelets (<100) Smear will have schistocytes. Tx: similar to severe preeclampsia/eclampsia with HTN control, Mg, steroids if < 36 wks, emergent delivery
Diagnosis: 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 → 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 28-29 wks and delivery 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 and when ?	Gestational age less than 12 wk: 50 mcg Gestational age greater than 12wks or UNKNOWN gestational age: 300 mcg Must be given within 72 hrs of the bleeding event.
How much blood does 300 mcg of Rhogam neutralize?	300 ml If patient undergoes significant trauma, they may need a second dose.
What is the Kleihauer-Betke Test ? 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 5 ml 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 Rhogam is needed.
Review the definitions of threatened, inevitable, incomplete, complete, septic, and missed abortions	Threatened: vaginal bleeding + IUP + closed os. Inevitable: vaginal bleeding + IUP + open os. Incomplete: vaginal bleeding + open os + some POC expelled/some still in uterus. Complete: vaginal bleeding + closed os + complete passage of POC. Missed: nonviable fetus (no heart tones) aged <20 wks in the uterus for at least 8 wks w/o passage. Septic: 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 with TVUS Refer for serial hCG if no IUP and below discriminatory zone (important if early ectopic possible) Pelvic rest and outpatient OB f/u RhoGam if Rh- mom
Diagnosis: 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 is MC (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 1,500 mU/mL Transabdominal: hCG 2,400-4,000 mU/mL If no IUP and hCG below these cutoffs, patient needs OB f/u in 48 hours for repeat hCG and US to rule out ectopic
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 < 3.5 cm, no fetal cardiac activity, no evidence of rupture, reliable for follow up
Diagnosis: Patient presents 1 week after being started on methotrexate for an ectopic. Workup?	“Separation pain” - thought to be from tubal abortion or hematoma formation Needs labs and TVUS 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
What are the ED options for emergency contraception ? How long after intercourse can each be used?	Copper IUD is most effective and can be used up to 5 days out . Ulipristal can be used up to 5 days but is not as effective as Copper IUD . Combined OCPs can be used up to 72 hours out . Progestin is recommended within 48 hours .
What suggests hyperemesis gravidarum ?	No strict clinical definition, however nausea and vomiting causing ketonuria and loss of > 5% of body weight are commonly used. Peak incidence is 8-12 weeks GA .
High risk time for fetal radiation exposure ?	Between 2-7 weeks , during organogenesis
Signs & Symptoms, Treatment: Mastitis/breast abscess	Due to blocked duct and secondary infection (<i>Staph</i> > <i>Strep</i>). SSx: breast pain, fever, erythema, induration Tx: warm compresses, I&D if abscess is present, antibiotics (dicloxacillin , cephalexin). **Patient should continue breastfeeding**
What is first line pharmacological management of nausea and vomiting in pregnancy ?	Pyridoxine (B6) and doxylamine (antihistamine)

Renal & Genitourinary

Bizz	Buzz
What defines acute renal failure ?	50% increase from baseline Cr OR 50% decrease in GFR
Causes, Labs: prerenal cause of acute renal failure	Most common cause of acute AKI in the community. Cause: most common is ↓ Renal hypoperfusion (ACEI, NSAIDs); ↓ Intravascular volume (hypovolemia, sepsis, blood loss, etc). Labs: BUN:Cr ratio >20 and FENa < 1% (use FEUrea if on diuretics), urine Na <20, relatively normal UA.
Causes, Labs: intrinsic renal cause of acute renal failure	Causes: 2/2 pathology within the kidney, with acute tubular necrosis being the most common cause (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.
Causes, Labs, Diagnostic Test: postrenal cause of acute renal failure	Cause: 2/2 obstruction of urine outflow, with BPH being the most common cause. Others - bladder CA, ureteral stone, urethral stricture. Labs: relatively normal UA. Diagnostic Test: US looking for post-void residual (>150 cc is abnormal).
What is the most likely cause of cardiac arrest 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 (hyperkalemia refractory to medical management) I ntoxication (toxins like ethylene glycol, methanol, Li, etc.) O verload (volume, any pulmonary edema, hypoxia) U remia with symptoms (e.g. pericarditis, AMS, BUN 100 or Cr 10)
Treatment: bleeding AV fistula	Apply tourniquet proximal to the fistula (for arterial supply) and a blood pressure cuff distally (for venous flow). Purse string or figure of eight sutures with a non-cutting needle. Can use topical/IV DDAVP for uremic bleeding syndrome. Alt: press plastic soda bottle cap with hollow side toward wound and secure in place with tight coban
What are symptoms of uremia?	Pericardial effusion/tamponade, altered mental status, n/v, anemia/bleeding (2/2 platelet dysfunction)
Treatment: uremic bleeding syndrome	DDAVP - topical or IV
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 oxalate. Patients with hypercalcemia (2/2 sarcoid, multiple myeloma, hyperthyroid and hyperparathyroid, cancer), Crohn's disease (2/2 increased oxalate absorption).
What is the composition of Struvite kidney stones ? What are the risk factors for these stones?	Magnesium-ammonium-phosphate stones. Most common cause of staghorn calculi. Increased risk with chronic UTIs , caused by urease-splitting bacteria (e.g. Proteus).
What are the major risk factors for uric acid kidney stones ? What is the recommended treatment ?	Increased risk with gout, leukemia , myeloproliferative disorders, tumor lysis syndrome. Tx: IVF, bicarb to alkalize urine, surgical removal PRN. Note: Uric acid stones are radiolucent (don't show up on Xray)
How often is there hematuria on UA when the patient has 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
Most common cause: glomerulonephritis	Post-streptococcal GN
Signs and symptoms, Treatment: glomerulonephritis and nephritic syndrome	Ssx: 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
Signs and symptoms, Treatment: nephrotic syndrome	"NEPHROTIC" N a decrease (hypoNa) A lbumin decrease (hypoalbuminemia) P roteinuria (>3.5g/day) H yperlipidemia R enal vein thrombosis O rbital edema T hromboembolism I nfection (lose Ig's in urine) C oagulability (lose ATIII in urine) Tx: IVF, Na restriction, steroids, ACE-I (dilates efferent arterioles, reduces glomerular pressure, and decreases protein loss), VTE prevention
Most common causes: nephrotic syndrome in kids and adults	Kids: Minimal change disease Adults: Focal segmental glomerulosclerosis
Most common cause: painless hematuria in older men, children, and young adults/older women	Older Men: Bladder cancer followed by renal cancer. Children: glomerulonephritis. Young Adults/Older Women: UTI.
Diagnosis: UTI + fever + nausea, vomiting	Pyelonephritis (cystitis rarely presents with fever)
Diagnosis: UA with WBC but no bacteria	Sterile Pyuria , 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>Indirect</u> : through inguinal canal into scrotum (lateral to inferior epigastric arteries) <u>Direct</u> : through muscle of abdominal wall .
What are potential complications of hernias?	Bowel obstruction, incarceration (hernia gets stuck out), strangulation (no blood flow, dead tissue)
Cause : balanitis/balanoposthitis	Inflammation of glans 2/2 fungal infection , less commonly bacterial; seen in uncircumcised men, diabetics , obese.
Cause : bilateral orchitis	Mumps virus, often associated with parotitis. Think about college age patients
Most common cause, Treatment : epididymitis/orchitis in young vs. old men	<u>Young (<35 yo)</u> : Cause - STIs Tx - CTX + doxycycline <u>Old (>35 yo)</u> : Cause - <i>E. coli</i> Tx - fluoroquinolone
What is Prehn's sign ?	Relief of pain with scrotal elevation in patients with epididymitis/orchitis
Signs and Symptoms, and Treatment : Prostatitis	SSx : dysuria, urinary frequency, pain with defecation, tender prostate. Tx : If <35 yo cover for STDs, otherwise give cipro to cover gram negatives and enteric flora **Avoid Foley as this will increase inflammation.**
What are the key differences between low-flow and high-flow priapism ?	<u>Low-flow</u> : most common form; due to venous obstruction, ischemic and painful . Causes: sickle cell (most common), meds (antipsychotics, penile injections). <u>High-flow</u> : usually painless . Cause: trauma and AV fistula (most common).
ABG analysis : ischemic priapism	acidemic (pH < 7.25), hypoxic (pO2<30), hypercapnic (pCO2 >60)
Treatment : priapism	Pain control (opiates, dorsal penile or ring nerve block); Intracavernosal aspiration (first line Tx); Intracavernosal phenylephrine (Tx after irrigation has failed), consider terbutaline (IM), and consult urology. In sickle cell patients consider exchange transfusion (but low threshold to drain).
Signs and Symptoms, Diagnostic Test, Treatment : 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). Tx : emergent urologic consultation for orchiopexy, can try manual detorsion via external rotation. **Consider this diagnosis 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
Characteristic clinical finding, Diagnostic Test, Treatment: torsion of the appendix testis	"Blue dot sign" (tender bluish nodule on the upper pole of the testis on physical exam - present in 25%). Diagnostic Test: US. Tx: scrotal support, NSAIDs
What is the most common misdiagnosis in patients with testicular cancer?	Epididymitis. Testicular cancer is the most common cancer in men aged 15-35. Exam will show a painless, firm, fixed nodule or mass.
CXR findings: metastatic testicular cancer	"Cannonball" lesions in lungs
What are extrarenal problems commonly associated with polycystic kidney disease ?	Liver cysts, cerebral berry aneurysms
What is the most common sign of bladder injury ?	Gross hematuria
What medication can cause epididymitis ?	Amiodarone
Diagnostic Test, Treatment: Peritonitis in a patient on peritoneal dialysis	Diagnostic Test: cloudy effluent, UA with 100 WBC, > 50% neutrophils or + Gram stain. Tx: <u>Stable:</u> intraperitoneal antibiotics and continued use of catheter. <u>Unstable:</u> admission + IV antibiotics. All antibiotics should cover skin flora (Strep and Staph).
Definition, Treatment: Phimosi	Definition: Condition of uncircumcised penis where foreskin is constricted and unable to be retracted. Tx: topical steroid cream, improved hygiene and gentle retraction. <u>If able to urinate:</u> no signs of infection or ischemia can be discharged w/ follow up with urology for elective circumcision. <u>If unable to urinate:</u> needs foley.

Hematology & Oncology

Bizz	Buzz
Diagnosis, Treatment: Transfusion + Fever + Otherwise well	Dx: Febrile non-hemolytic transfusion reaction. Most common transfusion reaction. Tx: tylenol, pause transfusion for 30 minutes, likely restart if hemolytic reaction ruled out
Diagnosis, Treatment: Transfusion + Urticaria + Otherwise well	Dx: Simple Allergic (Urticarial) Reaction Tx: IV benadryl (premedicate in future) but don't need to stop transfusion unless they have other signs of anaphylaxis
Signs and Symptoms, Diganosis, Treatment: Transfusion + Shock + AKI	Acute Hemolytic Transfusion Reaction. Often due to ABO incompatibility. SSx: fever, flank pain, shock Dx: +Coombs test, drop in Hgb, low haptoglobin, elevated LDH Tx: stop transfusion, IVF, diuretics, treat hyperK; Alternate Dx SEPSIS
Diagnosis, Treatment: Transfusion + Shock + Angioedema + Normal CXR	Dx: Severe Allergic Reaction (Anaphylactic). Associated with hereditary IgA deficiency. Tx: stop transfusion, epinephrine, IV benadryl, IV fluids, supportive care
Signs and Symptoms, Diagnosis, Treatment: Transfusion + Pulmonary Edema without other signs of heart failure	Transfusion Related Acute Lung Injury (TRALI) SSx: HIGH fever, hypoxemia, hypotension. Dx: ARDS after transfusion. CXR with pulmonary infiltrates but no other signs/symptoms of overload Tx: stop transfusion, supportive, don't benefit from diuresis. Most common cause of death following blood transfusion.
Signs and Symptoms, Treatment: Transfusion + Pulmonary Edema WITH other signs of heart failure	Transfusion-Associated Circulatory Overload (TACO) SSx: Presentation similar to TRALI but differentiated by hypertension, signs of volume overload (e.g JVD, peripheral edema, high bnp), NO Fever Tx: stop transfusion, supportive care, diuresis
Which 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 Hemophilia B ? Which coagulation studies will be abnormal?	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, abnormal PTT
What are the common clinical features of Hemophilia A and Hemophilia B ?	Minor trauma causing large amounts of bleeding or hemarthrosis (hallmark sign). Children: ankle (most common joint) Adults: knee (most common) > elbow & ankle CNS bleeding is the leading cause of death in hemophilia. In CNS bleeding, factor replacement should precede diagnostic imaging.

<p>What are the appropriate dosages of factor replacement for a patient with Hemophilia A with Minor, Moderate or Severe Bleeding?</p>	<p>Number of Factor VIII units = weight (kg) x (desired % increase in factor activity) x 0.5. (each unit increases by 2%) Minor (hemarthrosis): 20-30% factor desired (10-15 U/kg of Factor VIII) Moderate (epistaxis, GI bleed): 50% factor desired (25 U/kg of Factor VIII) Severe (CNS, RP bleed): 100% factor required (50 U/kg of Factor VIII)</p>
<p>What are the appropriate dosages of factor replacement for a patient with Hemophilia B and Minor, Moderate or Severe Bleeding?</p>	<p>Number of Factor IX units = weight (kg) x (desired % increase in factor activity); (each unit increases by 1%) Minor (hemarthrosis): 20-30% factor required (25 U/kg of Factor IX) Moderate (epistaxis, GI bleed): 50% factor required (50 U/kg of Factor IX) Severe (CNS, RP bleed): 100% factor required (100 U/kg of Factor IX)</p>
<p>What are alternative treatments if Factor is not available for bleeding hemophilia patient?</p>	<p>FFP (1 mL FFP = 1 U F8) Cryoprecipitate (1 bag = 100 U F8) DDAVP: 0.3 mcg/kg IV/SQ, 150 vs 300 mcg nasally, increases F8 activity & vWF (carries F8) PCC</p>
<p>What is the function of Von Willebrand Factor (vWF) during hemostasis?</p>	<p>1° hemostasis: attaches subendothelium to platelets (platelet aggregation) 2° hemostasis: protects factor VIII from degradation + delivers Factor VIII to site of injury (Factor VIII carrier protein)</p>
<p>Signs and Symptoms, Diagnosis, Treatment: Von Willebrand's Disease</p>	<p>Most common inherited bleeding disorder. SSx: easy bruising, skin bleeding, prolonged bleeding from mucosal surfaces (mouth, GI/GU) Dx: platelet count normal, normal PT/INR, possibly prolonged PTT (affects F8), prolonged bleeding time Tx: DDAVP (first-line treatment, increases release of vWF), non-recombinant Factor VIII, Cryo NOT recommended (risk of viral transmission), no FFP (very little F8); ± Antifibrinolytics (Amicar, Tranexamic acid) which inhibit clot breakdown</p>
<p>Signs and Symptoms, Diagnosis, Treatment: Polycythemia Vera</p>	<p>Clonal proliferation of RBCs/increased RBC mass. SSx: pruritus (aquagenic, plethora (facial), hypertension, engorged retinal veins, <u>thrombosis</u>, erythromelalgia (burning of hands/feet), splenomegaly Dx: all cell lines inc (especially RBC) Tx: serial phlebotomy, hydroxyurea, aspirin</p>
<p>How does heparin work? How is it monitored? How can it be reversed?</p>	<p>Mechanism: activates antithrombin III (inactivates Factor X and thrombin) Monitoring: PTT Reversal: Protamine Sulfate 1 mg per 100 U heparin, give slowly to avoid anaphylactoid reaction</p>

<p>How does LMWH work? How is it monitored? How can it be reversed?</p>	<p>Mechanism: activates antithrombin III (inactivates ONLY Factor X) Monitoring: Xa level Reversal: Protamine Sulfate (dose based on timing since last LMWH injection). Doesn't work nearly as well as it does for unfractionated heparin</p>
<p>How does warfarin work? How is it monitored? How can it be reversed?</p>	<p>Inhibits vitamin K clotting factors (2, 7, 9, 10, proteins C & S) Monitoring: PT and INR Reversal: FFP/Vitamin K (alternate PCC), dosage based on type of bleeding and INR</p>
<p>Review appropriate treatment to reverse coumadin based on severity of bleeding and INR.</p>	<p>INR < 5 & NO bleeding: lower or skip 1 dose INR ≥ 5 but ≤ 10 & NO bleeding: skip next 1-2 doses, alternative: skip 1 dose + Vitamin K 2.5-5 mg PO INR ≥ 10 & NO serious bleeding: hold med until INR is therapeutic + Vit K 5 mg PO ANY serious bleeding regardless of INR: hold med + Vitamin K 10 mg IV + FFP or PCC</p>
<p>How does tPA work? How can it be reversed?</p>	<p>Mechanism: converts plasminogen to plasmin to breakdown clots Reversal: no specific reversal agent. Can give large amount of everything (pRBCs, cryo, FFP, platelets, PCC, amicar, tranexamic acid)</p>
<p>How does clopidogrel work? How can it be reversed?</p>	<p>Mechanism: blocks glycoprotein 2b/3a & prevents platelet activation (crosslinking with fibrin) Reversal: nothing specifically reverses, can give platelets</p>
<p>How does Dabigatran (Pradaxa) work? How can it be reversed?</p>	<p>Mechanism: Direct thrombin inhibitor, associated with GI bleed Reversal: <u>Idarucizumab</u>, PCC/pRBC/platelets, can also do hemodialysis</p>
<p>How does Rivaroxaban (Xarelto) work and how can it be reversed?</p>	<p>Mechanism: Factor 10a inhibitor Reversal: no specific reversal (exam may want andexanet alfa, even though evidence is murky), NOT dialyzable, can try thrombin activation with PCC, FFP, cryo</p>
<p>Signs and Symptoms, Diagnosis: Elderly with chronic back pain, lytic lesions on x-ray</p>	<p>Multiple myeloma SSx: ("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 syndrom</p>
<p>What symptoms suggest aggressive Lymphoma?</p>	<p>"B symptoms": fever, night sweats, lymphadenopathy, weight loss</p>
<p>What distinguishes Non-Hodgkins from Hodgkin's Lymphoma?</p>	<p>NHL: more common, more widespread, less curable, leading cause of non-solid organ cancer-related death HL: less common, related to viral infection; often presents with B symptoms and local spread, high cure rates</p>

<p>What are the two most common types of Non-Hodgkin's Lymphoma and what distinguishes them?</p>	<p>Follicular Lymphoma: indolent, slow growing, widespread at diagnosis, no cure Diffuse Large B cell Lymphoma: aggressive and symptomatic, rapid spread, 50% cured</p>
<p>What are the two types of Burkitt's (non-Hodgkin's) lymphoma?</p>	<p>Associated with EBV Endemic (African) Burkitt lymphoma (eBL): most common; jaw and facial bone including the orbit (> 50%) Sporadic Burkitt lymphoma (sBL): less common; abdominal tumors with bone marrow involvement</p>
<p>Signs and Symptoms, Diagnosis, Treatment: Hodgkin's lymphoma</p>	<p>Bimodal age (teens/young adults, older adults) SSx: non-tender cervical lymphadenopathy, mediastinal mass on CXR, B symptoms Dx: Reed-Stenberg cell ("owls eye") Tx: chemotherapy, radiation</p>
<p>What is the difference between Acute and Chronic Leukemia?</p>	<p>Acute: rapid increase in blasts, most common in children Chronic: mature abnormal WBCs, slow growing, most common in elderly</p>
<p>What is the difference between cell types seen in Lymphocytic and Myelogenous Leukemias?</p>	<p>Lymphocytic: B & T cells Myelogenous: RBCs, platelets & other WBCs</p>
<p>What are the similarities and differences between the presentations of ALL vs AML?</p>	<p>BOTH: bony pain, big liver/spleen, anemia, bleeding, thrombocytopenia, infection and blasts in blood ALL: most common childhood leukemia, +LAD AML: more common in adults, no LAD, + gingival infiltration, Auer rods on blood smear</p>
<p>What are the similarities and differences between the presentations of CLL vs CML?</p>	<p>BOTH: slow onset, elevated WBCs CLL: most common adult leukemia, smudge cell, worst prognosis CML: mostly adults, Philadelphia chromosome, high platelets, good prognosis</p>
<p>Diagnosis, Treatment: neutropenic fever</p>	<p>Dx: one oral temp $\geq 38.3^{\circ}\text{C}$ or $\geq 38^{\circ}\text{C}$ for ≥ 1 hour + ANC < 500; obtain cultures (gram positive most common) Tx: admission, empiric antibiotics (solo coverage with zosyn or cefepime; add vancomycin if you suspected skin or soft tissue infection)</p>
<p>Signs and Symptoms, Diagnosis, Treatment: Hyperviscosity syndrome</p>	<p>Increased serum viscosity that causes sludging & vascular stasis Causes: Leukemias (AML or CML in blast crisis, WBC > 100k), multiple myeloma, Waldenstrom macroglobulinemia (most common cause), polycythemia vera. SSx: mucosal bleeding (epistaxis), CNS symptoms (blurred vision, headache, AMS, stroke), end-organ ischemia Dx: severe out of proportion elevations of the affected cell line, Rouleaux formation Tx: phlebotomy (polycythemia) + IVF, plasmapheresis (high proteins), leukapheresis for blast transformations (induction chemotherapy = definitive treatment)</p>

<p>Diagnosis, Treatment: Tumor Lysis Syndrome</p>	<p>Massive cytolysis + release of the intracellular contents, can occur with aggressive heme malignancies, large solid tumors/steroids after start of chemotherapy. Dx: HIGH uric acid, phosphate, potassium & LOW calcium. Tx: aggressive IVF, correct electrolytes (hyperUA: Allopurinol, Rasburicase; hyperphosphatemia: aluminum hydroxide, Renagel, iHD; hyperkalemia: calcium, insulin/glucose, bicarb, kayexalate, HD; hypocalcemia: secondary to high phosphate, treat hyperphosphatemia first, only treat if symptomatic). Complications: cardiac arrhythmias, renal failure</p>
<p>Name the criteria for emergent HD in tumor lysis syndrome</p>	<p>K ≥ 6, uric acid ≥ 10, Cr ≥ 10, phosphorus ≥ 10, volume overload, symptomatic hypocalcemia</p>
<p>Signs and Symptoms, Diagnosis, Treatment: Thrombocytopenia, otherwise normal labs, well patient</p>	<p>Idiopathic Thrombocytopenic Purpura (ITP), results from rapid destruction of plts (fxn is normal). Types: children (2-6 years): acute, post-infectious; adults (20-50 years) SSx: petechiae (most common), purpura, gingival bleeding, epistaxis, menorrhagia Dx: thrombocytopenia Tx: observation (only if asymptomatic + platelets > 50K), supportive (kids), steroids (platelets < 50K) & IVIG, platelets (only for severe bleeding, VERY low platelets), others: splenectomy (refractory cases)</p>
<p>Signs and Symptoms, Diagnosis, Treatment: Thrombotic Thrombocytopenic Purpura</p>	<p>Thrombotic Thrombocytopenic Purpura (TTP): enzyme defect leads to unstable platelet plugs & hemolytic anemia SSx (PENTAD): <u>F</u>ever <u>A</u>nemia (MAHA, schistocytes; HIGH indirect bili, LDH, retic count; LOW haptoglobin) <u>T</u>hrombocytopenia (10-50k) <u>N</u>euro symptoms Dx: decreased ADAMTS-13 activity, schistocytes, normal: PT/INR, fibrinogen, dimer Tx: supportive, plasmapheresis (treatment of choice); others: plasma exchange transfusion, steroids, DMARDs, IVIG, splenectomy; DO NOT GIVE PLATELETS</p>
<p>What types of patients are at higher risk for developing TTP?</p>	<p>African-American females, Lupus, HIV, medications (Clopidogrel, Quinine)</p>
<p>Diagnosis, Treatment: child with thrombocytopenia, hemolytic anemia, renal failure</p>	<p>Hemolytic Uremic Syndrome (HUS); often after diarrheal illness (O157:H7- shiga-like toxin) Dx: with evidence of hemolysis (schistocytes, high unconjugated bilirubin, high LDH) Tx: supportive care, transfuse pRBC's for Hgb < 6, DO NOT GIVE PLATELETS OR ANTIBIOTICS</p>

<p>What defines Heparin Induced Thrombocytopenia (HIT) and what is the treatment?</p>	<p>Antibodies that inactivate platelets usually at 5 days after initiating heparin if naive and only minutes to hours after initiation if prior exposure. Diagnosis: (4 T's): Thrombocytopenia (platelets < 150 K or > 50% drop after starting heparin [less often LMWH]), Time of onset (5-10 days), THROMBOSIS (thrombosis, skin reactions, PE, CVA, MI) no oTher cause. Labs: +HIT antibody. Tx: STOP heparin or LMWH, can change to direct thrombin inhibitor (Argatroban, Dabigatran), NO platelets</p>
<p>Diagnosis, Treatment: Disseminated Intravascular Coagulation (DIC)</p>	<p>Microvascular <u>thrombosis</u> AND <u>consumptive coagulopathy</u> causing multi organ failure. Related to underlying severe illness (sepsis is most common cause) & massive inflammation (trauma, pregnancy complications, cancers). Labs: LOW: platelets (most common) & fibrinogen; HIGH: PT/INR, fibrinogen degradation, dimer Tx: underlying cause Tx: (primarily bleeding): FFP, platelets, RBCs; Tx: (primarily thrombosis): heparin, LMWH</p>
<p>In what thrombocytopenic disorders are platelets contraindicated?</p>	<p>TTP, HIT, HUS</p>
<p>What are the 3 main causes of microangiopathic hemolytic anemia?</p>	<p>TTP, HUS, DIC</p>
<p>What are diseases commonly associated with thrombocytopenia?</p>	<p>TTP, HUS, DIC, SLE, HIV</p>
<p>What are classic causes of microcytic and macrocytic anemias?</p>	<p>Microcytic (MCV < 80): iron deficiency, thalassemia, anemia of chronic disease Macrocytic (MCV > 100): B12 or Folate deficiency</p>
<p>Anemia + Low reticulocytes Low ferritin Low iron High TIBC</p>	<p>Iron Deficiency Anemia</p>
<p>Anemia + High retic Normal to high ferritin Normal to high iron Target cells (smear)</p>	<p>Thalassemia - defective hemoglobin chains (A - Africa, B - India)</p>
<p>Anemia + Headache Abdominal pain Basophilic stippling (smear)</p>	<p>Chronic Lead Poisoning, may also see Burton's line (blue line on gums)</p>

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 peripheral smear), macrocytic
What patients are at higher risk for B12 deficiency ?	Crohns Disease (B12 is absorbed in the ileum), on PPI (decreased absorption), vegan diet (decreased intake)
Anemia + Hypersegmented neutrophils + NO neurologic changes	Folate Deficiency (also consider in alcoholics with anemia)
Which patients are at higher risk for Folate deficiency ?	Alcohol use disorder, tea and toast diet (elderly) - both from malnutrition
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 disease in infants ?	Acute Dactylitis: pain and swelling of hands and feet secondary vaso occlusive crisis, due to infarction NOT infection Tx: supportive
Treatment of Sickle Cell patient with Priapism ?	Low-flow (venous/ischemic) causes erect penis with soft glans Tx: aspirate corpus, intracavernosal phenylephrine, IM terbutaline, surgical drainage as needed
Treatment of Sickle Cell patient with Stroke ?	Emergent exchange transfusion
Diagnosis, Treatment: Acute Chest Syndrome	Sickle cell patient with fever, dyspnea and pneumonia, HIGH mortality (most common cause of death in sickle cell patients) Causes: infection, vaso-occlusive crisis, fat embolism Dx: infiltrate on chest x-ray Tx: ICU admit, supportive (incentive spirometer, IVF, supplemental oxygen, pain control) antibiotics for CAP, pRBCs vs. exchange transfusion (severe crises marked by PaO2 < 60 mm Hg, not first line)
Signs and Symptoms, Diganosis, Treatment: child with sickle cell + non-traumatic rapid drop in hemoglobin	Aplastic Crisis SSx: pallor, weakness/lethargy, shock; arthralgias, arthritis (adults) Dx: Hgb drop by at least 2 points from their baseline, LOW reticulocytes <2%; associated with Parvovirus B19. Tx: pRBCs, IVIG
Signs and Symptoms, Diagnosis, Treatment: child 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 Tx: IVF, transfuse pm, splenectomy

Which infections are more common in sickle cell patients ?	Encapsulated organisms: <i>S. pneumoniae</i> , <i>H. influenzae</i> , <i>N. meningitidis</i> because these patients have a non-functional spleen.
Diagnosis: 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: negative Coombs, Heinz bodies on smear.
What are potential G6PD triggers ?	Fava beans, Infections, Meds: dapsone, TMP-SMX , phenazopyridine, nitrofurantoin, antimalarials, rasburicase, and methylene blue
Diagnosis: Elderly patient, gradual face swelling, periorbital edema, cough, and cyanosis. History of smoking.	Superior vena cava syndrome Dx: CT chest with contrast
Thresholds for platelet transfusions in adults	Trauma or active bleeding ITP: only for platelets < 10k and severe bleeding Coagulation disorder if < 20k Everyone else: <5-10k
Diagnosis: Paresthesias and arm/face spasms after massive transfusion protocol	Hypocalcemia related to citrate in blood transfusion products

Dermatology

Bizz	Buzz
Diagnosis, Treatment: Erythema Multiforme	Often viral trigger (HSV most common); Hallmark = TARGET lesions , SYMMETRIC on palms & soles (± trunk, face), minimal to no mucosal involvement, -Nikolsky. Tx: remove trigger, supportive
What is the most common cause of Erythema Multiforme ?	Infections: HSV (most common viral cause) > Mycoplasma (most common bacterial cause)
Which drugs are most commonly associated with Erythema Multiforme ?	SOAPS: S ulfa O ral hypoglycemics A nticonvulsants P enicillin N SAIDS
What are the similarities and differences between Stevens Johnson Syndrome and Toxic Epidermal Necrolysis ?	BOTH: mucosal involvement, +Nikolsky , drugs = most common cause, flu-like prodrome, painful target lesions SJS: <10% TBSA, most common in children TEN: >30% TBSA, more common in elderly, fluid / lyte problems common; Treatment (both): supportive, remove trigger, transfer to burn center
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
Signs and Symptoms, Diagnosis, Treatment: Necrotizing Fasciitis?	Type 1: Polymicrobial (most common), abdomen/perineum, Type 2 diabetes = 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 (patient unconcerned) Dx: Clinical diagnosis! CT/radiograph with subcutaneous emphysema. Tx: Broad spectrum IV antibiotics (vancomycin/zosyn + clindaycin) AND surgical debridement (definitive treatment).
Diagnosis, Treatment: Urticaria	Dx: Transient/migratory pruritic edematous plaques , red border with central clearing, NOT symmetric Tx: remove trigger, benadryl/steroids/epinephrine as needed

<p>Signs and Symptoms, Diagnosis, Treatment: Rocky Mountain Spotted Fever?</p>	<p><i>Rickettsia rickettsii</i>. Transmission: Dermacentor tick, must be attached for 6 hours to transmit, eastern US (Carolinas, Oklahoma) SSx: fever (most common symptoms), centripetal (wrists/ankles → trunk) maculopapular rash (palms + soles), calf tenderness. Dx: low platelets, hyponatremia Tx: Doxycycline</p>
<p>Diagnosis, Treatment: College kid with petechiae → purpura presents in shock</p>	<p>Meningococemia; seen in college kids, military barracks (close quarters), caused by <i>Neisseria meningitidis</i> (requires airborne precautions). Dx: LP with CSF studies Tx: ceftriaxone, supportive</p>
<p>Who needs N. Meningitidis prophylaxis? Which medications are used?</p>	<p>Ppx: household contacts, intimate partners, flights >8 hours next to infected person, daycare exposure, intubation without proper PPE (mask, faceshield) Antibiotics: ceftriaxone, rifampin, or ciprofloxacin</p>
<p>What is the difference between Pemphigus Vulgaris and Bullous Pemphigoid?</p>	<p>Pemphigus: S: Superficial, flaccid bullae → break easily & crust, +mucosal involvement, +Nikolsky; Associations: Myasthenia, thymoma Treatment: steroids Pemphigoid: D: Deeper, elderly, pruritic papules → tense bullae, NO mucosal involvement, -Nikolsky Treatment: steroids, tetracycline or dapsone</p>
<p>Signs and symptoms, Treatment: Shock + Erythroderma and possible foreign body</p>	<p>Toxic Shock Syndrome. Bacteria that produce toxins. Staph (TSS): more common; erythematous rash with desquamation + hypotension + high fever ≥3 organ systems, <u>associated with foreign body</u> Strep (STSS): fever, but less rash often with existing wound, not associated with foreign bodies Tx: remove foreign bodies FIRST, supportive care, and antibiotics (clindamycin first to reduce protein production, then empiric broad-spectrum for sepsis coverage), IVIg for refractory cases</p>
<p>Signs and symptoms, Diagnosis, Treatment: Gunmetal gray pustules on palms</p>	<p>Disseminated Gonococemia (arthritis-dermatitis syndrome) SSx: fever + migratory arthritis + rash (papules → pustules with gray necrotic or hemorrhagic center) Dx: genital + throat culture Tx: ceftriaxone. Complications: tenosynovitis, septic arthritis</p>
<p>Signs and Symptoms, Treatment: Impetigo</p>	<p>SSx: most often in kids, facial vesicles rupture and become "honey-crusted", + contagious, Staph more common cause than strep Tx: topical mupirocin if small or localized area; systemic keflex if more extensive or bullous</p>
<p>What is the characteristic rash and cause of Erysipelas?</p>	<p>Well demarcated, slightly raised, beefy red plaque. Group A Strep = most common cause</p>

<p>Diagnosis and Treatment: Obese woman with red macular rash under breasts, noted satellite lesions</p>	<p>Dx: Candida; also associated with immunocompromised state Tx: Topical azoles for rashes, dry skin care</p>
<p>What is the difference in presentation and treatment between Candida and Tinea rashes?</p>	<p>Candida: seen in babies, immunocompromised, diabetes mellitus, obese adults (intertriginous), rash: red + macular with characteristic satellite lesions Tx: PO nystatin for thrush, Topical azoles for rashes, dry skin care Tinea: sharply marginated, annular lesion with raised or vesicular margins with central clearing and scaling Tx: topical azoles for everything except scalp and nails (griseofulvin or terbinafine)</p>
<p>What are the names for Tinea infections in the following areas: groin, foot, scalp, nail?</p>	<p>Groin: Crura (jock itch) Foot: Pedis Scalp: Capitis Nail: Unguium</p>
<p>Compare the rashes of HSV and HPV.</p>	<p>HSV: vesicular clusters with painful erosions (T1- mouth, T2- genitals); HPV: cauliflower-like and painless (anogenital warts) = most common sexually transmitted infection in US (> Chlamydia)</p>
<p>What is the significance of a vesicle or ulcer noted on tip of nose or ear?</p>	<p>Herpes Zoster (shingles) infection Tip of nose (Hutchinson sign) for herpes ophthalmicus (V1) Ear (Ramsay-Hunt) indicates involvement of CN 7/8</p>
<p>What is the characteristic rash Molluscum Contagiosum? What are common risk factors? Treatment?</p>	<p>Dome-shaped fleshy papule with central umbilication on face/torso/ext; most common in kids in daycare or adults with HIV Caused by a pox virus Tx: self-limited, cryotherapy</p>
<p>Compare the rashes of Scabies and Pediculosis. What are the treatments?</p>	<p>Scabies: linear burrows in interdigital web space and intertriginous areas with extreme pruritus Pediculosis (lice): erythematous macules/wheals, extreme pruritus, nits visible Tx: (BOTH): decontamination, Permethrin cream (often repeat at one week, especially with lice)</p>
<p>Compare atopic dermatitis and psoriasis. What are the treatments?</p>	<p>Atopic dermatitis (eczema): usually kids <5, allergy/asthma history, winter months, dry pruritus skin with lichenification (hyperpigmentation/thickening) in flexural areas Psoriasis: 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) Tx (BOTH): emollients, topical steroids, biologics (psoriasis)</p>
<p>Diagnosis, Treatment: Seborrheic Dermatitis</p>	<p>Cradle cap Dx: Occurs in infants. Yellowish, greasy scales on scalp, ± diaper area & axillae Tx: salicylate shampoo, mineral oil, avoid steroids</p>

Which disease is associated with seborrheic dermatitis in adults ?	HIV
Diagnosis, Treatment: Contact Dermatitis	Dx: Discrete, well-defined or demarcated rash (papules/vesicles/bullae) secondary to direct irritant vs allergic reaction Tx: remove trigger, protect skin, steroids
What is the indication for and duration of oral steroid treatment for poison oak/ivy ?	Indication: large areas of affected skin, usually with involvement of the face and genitals. Duration: Requires 3 weeks of oral steroids with a taper. Normal steroid bursts can cause rebound dermatitis.
What are the distinguishing features of Basal Cell vs Squamous Cell Carcinoma ? Treatment?	BCC: pink, pearly papules with telangiectasia in sun-exposed areas, more common SCC: UV exposure, ulcerated center with firm-raised border Tx: BOTH referred for biopsy
What characteristics are concerning for melanoma ? Treatment?	ABCDE: A symmetry B order (irregular) C olor (different shades, not uniform) D iameter (>6 mm) E volution Tx: excisional biopsy; depth = most important prognostic factor
Diagnosis, Treatment: Purple papules on gums and skin	Dx: Kaposi Sarcoma; lesions most commonly oral, also gastrointestinal and pulmonary, they are painless and non pruritic, seen in HIV/AIDS patients Tx: treat HIV
What is the most common diagnosis for a blanching strawberry lesion on an infant's head ?	Hemangioma; 50% resolve by 5 years of age Head > trunk > extremity
What distinguishes a Lipoma from a Sebaceous Cyst ?	Lipoma: well-circumscribed, mobile and painless, "Slippage sign" with normal overlying skin Sebaceous Cyst: central punctum, cottage cheese discharge, no slippage, may have secondary infection Tx (both): referral for excision
What defines the stages of decubitus ulcers ?	I: non blanching erythema, intact skin II: partial thickness, exposed dermis III: full thickness skin loss, exposed subcutaneous fat IV: full thickness tissue loss, exposed bone/tendon/muscle
Diagnosis, Treatment: Painful red nodules on shins	Dx: Erythema Nodosum; Associated with IBD, malignancy, infection (strep most common), or medications (OCPs). Patients often have a prodrome of fever, malaise and arthralgias. Tx: supportive, high dose aspirin 650 mg every 4 hours or NSAIDs
Characteristic rash of Pityriasis ? Treatment?	Herald patch → "Christmas tree" distribution rash to trunk, ± pruritus Tx: 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 a patient is immunocompromised or possibly has disseminated infection , then airborne + contact precautions are required. If a patient is immunocompetent with localized zoster , then standard precautions can be followed.
What rashes (5) are associated with palmar lesions ?	Syphilis (secondary) RMSF Scabies Erythema Multiforme Hand/foot/mouth
Which rashes are associated with + Nikolsky sign ?	SJS TEN SSSS Pemphigus Vulgaris
Which rashes (4) are associated with vesicles/bullae ?	Bullous pemphigoid Pemphigus Vulgaris Necrotizing fasciitis Disseminated Gonorrhea
Which rashes (4) are associated with petechiae/purpura ?	RMSF Meningococemia DIC Endocarditis
Which rashes (3) are associated with target lesions ?	Lyme disease Erythema Multiforme SJS
Signs and Symptoms, Treatment, Complications: Henoch-Schonlein Purpura (HSP)	SSx: typically age 4-12 years, recent URI, abdominal pain, arthralgia, and a rash (palpable purpura to buttocks and lower extremities, non-pruritic); Most commonly caused by IgA mediated vasculitis; Tx: supportive care. Complications: nephropathy, intussusception
Diagnosis, Treatment: Epidermoid cyst	Also known as sebaceous cyst. Dx: skin-colored lesion, often with central punctate area with white or yellowish waxy material. Tx: steroids, incision and drainage; excision. Follow up with dermatology

Musculoskeletal / Rheumatology

Bizz	Buzz
<p>Review the Salter-Harris classification for pediatric fractures. Which category is most common?</p>	<p>"SALTR" describes the fracture in relationship to the epiphyseal plate. I: <u>S</u>lip; separation straight across the physis II: <u>A</u>bove; fx through the physis and metaphysis III: <u>L</u>ower; fx through the physis and growth plate IV: <u>T</u>hrough; fx through the metaphysis, physis, and epiphysis V: <u>E</u>Rasure; crush injury to the physis More advanced fracture types are more likely to cause growth disturbance. Salter Harris II is the most common.</p>
<p>Cause, Diagnosis, Treatment: Torus or buckle fracture (pediatric)</p>	<p>Cause: incomplete fracture 2/2 impaction/axial load Dx: xray shows buckling or bulging of the cortex on one side of the bone without a clear fracture line; periosteum is intact; angulation may be present but is not required for diagnosis Tx: splint in functional position, outpatient pediatrician follow up</p>
<p>Cause, Diagnosis, Treatment: Greenstick fracture</p>	<p>Cause: incomplete fracture 2/2 impaction/axial load Dx: xray shows fracture line on only one side of the bony cortex with the opposite side bent but otherwise intact Tx: splint, pediatrician follow up (some sources say no follow up needed) Only manipulate if severely malaligned (<15° acceptable if <10 years old and <10° acceptable if >10 years old).</p>
<p>Definition, Diagnosis, Treatment: Toddler fracture</p>	<p>Definition: spiral fx of distal tibia in kids aged 9 months - 3 years who walk and fall (NOT a fracture pattern of abuse) Dx: xray of the tib/fib, may need oblique view if AP/Lat is negative but high suspicion (can also immobilize and f/u in 1 week for repeat imaging) Tx: long leg splint vs walking boot, next day ortho f/u for casting</p>
<p>What fracture patterns suggest child abuse?</p>	<p>Metaphyseal corner ("Bucket handle") fx Rib fractures (posterior rib fx = pathognomonic) Fracture of sternum/scapula/spinous process Long bone fx in <u>nonambulatory</u> infant <u>Multiple fx in various stages of healing</u> Bilateral acute long bone fx Vertebral body fx without h/o high force trauma Digital fx in children <36 mo Severe skull fx in children <18 mo</p>
<p>What are the general sensory and motor functions of the radial nerve?</p>	<p>Sensory: dorsal/radial aspect of hand (1st dorsal web space) Motor: wrist and finger extension</p>

What are the general sensory and motor functions of the median nerve ?	Sensory: palm & palmar aspect of distal dorsal digits 1-3 Motor: "tea drinking" (pincer grasp, flexion at wrist & elbow, pronation)
What are the general sensory and motor functions for the recurrent branch of the median nerve ?	Sensory: NONE (pure motor nerve) Motor: "thumb OAF" - opposition, adduction, flexion
What are the general sensory and motor functions of the ulnar nerve ?	Sensory: ulnar aspect of palmar & dorsal digits 4-5 Motor: hand intrinsic muscles
What is adhesive capsulitis ? How is it caused? How is it treated?	Adhesions between joint capsule and humeral head → stiffness and decreased range of motion Causes: injury or spontaneous Tx: Codman's exercises (pendulum swing with light hand weights), NSAIDs, intra-articular steroids, NO SLING
Cause, Signs & Symptoms, Diagnosis, Treatment: Rotator cuff injury	Cause: repetitive (overuse) movements, trauma SSx: shoulder pain, cannot ABduct or externally rotate Dx: xray (to rule out other injuries), outpatient MRI (ligamentous tears) Tx: NSAIDs , ortho referral for further care, do not place in a sling (increases the risk of developing adhesive capsulitis) *Note: rotator cuff includes SITS muscles (S upraspinatus, I nfraspinatus, T eres minor, S ubscapularis)
Diagnosis, Treatment: Clavicular fracture	Clavicular fracture is the MC fracture in kids . Dx: xray (middle 1/3 = MC location), CT only if additional injury is suspected Treatment depends on the location and severity. Nondisplaced/minimally displaced: supportive (sling, pain control, outpatient ortho f/u) Skin tenting: reduction to prevent open fx Open fx: admission, IV abx, and surgery
Cause, Management: Acromioclavicular joint separation	Occurs with direct blow (contact sports). Dx: XR: grades I-IV. Tx: Mild to moderate: sling, ortho f/u; moderate to severe: sling, ortho referral, surgery
What are the potential complications of anterior vs. posterior shoulder dislocations?	Anterior: injury to axillary nerve (check for sensation over deltoid); Hill-Sachs lesion (compression fx of posterolateral humeral head); Bankart lesion (tear in glenoid labrum) Hill-Sachs is the most common. Inferior: injury to axillary artery or brachial plexus Others: adhesive capsulitis (particularly with recurrent dislocations)
What circumstances increase the risk for posterior shoulder dislocation ?	Seizure or lightning strike 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.

<p>What are the potential complications of humeral head and humeral shaft fractures?</p>	<p>Head: adhesive capsulitis (most common), avascular necrosis Shaft: spiral fracture → radial nerve injury → wrist drop, brachial artery injury, difficulty with wrist supination</p>
<p>Diagnosis, Treatment, Complications: Pediatric supracondylar fracture</p>	<p>Dx: most common pediatric elbow fx; xray shows abnormal anterior humeral line (should pass through middle of the capitellum), posterior fat pad (always abnormal), elevated anterior fat pad ("sail sign") Tx: admit any type 3 supracondylar fx (displaced anterior and posterior periosteum); posterior long arm splint for other supracondylar fx Complications: high risk for brachial artery injury, compartment syndrome/Volkmann's contracture</p>
<p>What is a Volkmann ischemic contracture? What fracture pattern is it commonly associated with?</p>	<p>Compartment syndrome of the arm → ischemic necrosis of the wrist and finger flexors in the forearm → muscles scar and contract → wrist flexion contracture and claw-hand deformity Commonly associated with supracondylar fracture.</p>
<p>Patient presents with elbow injury and no obvious findings on initial imaging. What xray findings might indicate an occult supracondylar or radial head fracture?</p>	<p>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</p>
<p>What fracture is most commonly associated with a posterior hip dislocation?</p>	<p>Acetabular fracture</p>
<p>What are the potential complications of elbow dislocation?</p>	<p>2nd most common dislocated joint (shoulder = first) Posterior (ulna is posterior to humerus) > anterior Complications: ulnar nerve injury (most common), compartment syndrome, brachial artery injury (rare), median nerve injury Low threshold for CT angiography</p>
<p>Cause, Diagnosis, Treatment: Nursemaid's elbow</p>	<p>Found in children 1-3 years old. Caused by axial traction (e.g. parent pulls kid up by his arm) → radial head displaced from the annular ligament Dx: clinical; no XR needed Tx: immediate reduction with hyperpronation and/or simultaneous supination and flexion; monitor for normal use 30 min later</p>

<p>Define: Monteggia, Galeazzi, and Essex-Lopresti fracture</p>	<p>Monteggia: proximal ulnar fracture + radial head dislocation (look for radial nerve injury at radial head dislocation w/ wrist drop) Galeazzi: distal radial fracture + distal radioulnar joint disruption Essex-Lopresti: comminuted radial head crush fracture + distal radioulnar joint disruption ALL three require open reduction and internal fixation (ORIF). Remember "MUGgER" (Monteggia with Ulnar fracture, Galeazzi & Essex with Radial fractures).</p>
<p>Diagnosis, Treatment: Nightstick fracture</p>	<p>Dx: midshaft ulna fracture 2/2 direct blow (e.g. while trying to protect oneself from being struck with a policeman's nightstick) Tx: r/o Monteggia fx, posterior long arm splint</p>
<p>What is the difference between a Colles' fracture and a Smith's fracture? Describe the most common complications and indicated treatment for both.</p>	<p>Colles: distal radius fracture with dorsal angulation usually 2/2 fall onto outstretched hand Smith's: 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") Tx (both): closed or open reduction, sugar tong splint</p>
<p>Mechanism, Diagnosis, Treatment: Triquetral fracture</p>	<p>Mechanism: fall onto outstretched hand (FOOSH) → dorsal chip fracture of the triquetrum Dx: lateral hand xray Tx: volar splint, outpatient hand surgery f/u *Note: Triquetral fracture is the 2nd most common carpal bone fracture.</p>
<p>Mechanism, Signs & Symptoms, Diagnosis, Treatment, Complication: Scaphoid fracture</p>	<p>Mechanism: fall onto outstretched hand (FOOSH) SSx: snuffbox tenderness or pain with axial loading of thumb Dx: physical exam; xray may appear normal initially Tx: thumb spica and outpatient hand surgery f/u If there is no overt fx on xray, but suspicion remains high → splint + repeat xray in 10-14 days Complication: avascular necrosis of the proximal segment; nonunion *Note: Most common carpal bone fracture</p>
<p>Diagnosis, Treatment, Complication: Lunate fracture</p>	<p>Dx: focal tenderness of dorsal proximal hand and with axial load of 3rd digit; may have normal xray Tx: high clinical suspicion → place sugar tong splint + outpatient hand surgery f/u Complication: high risk of avascular necrosis</p>
<p>Diagnosis, Treatment: Scapholunate dislocation</p>	<p>Dx: >3 mm widening between scaphoid and lunate, "Terry Thomas sign," localized tenderness Tx: splint + hand surgery consultation (usually requires surgical repair)</p>

<p>Diagnosis, Treatment, Complications: Lunate dislocation</p>	<p>Dx: xray with "piece of pie sign" on AP view and "spilled teacup" (volar displacement of lunate) on lateral view Tx: splint, ortho consult for open reduction and internal fixation (ORIF) Complications: median nerve injury, avascular necrosis</p>
<p>Diagnosis, Treatment: Perilunate dislocation</p>	<p>Dx: capitate is displaced dorsally with normal lunate alignment over radius Tx: splint, immediate ortho consult for reduction</p>
<p>Diagnosis, Treatment: Boxer's fracture</p>	<p>Dx: xray with fx of 5th metacarpal neck or shaft Tx: 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.</p>
<p>Identify the site of injury and resulting hand position in (1) mallet finger, (2) boutonniere deformity, (3) swan neck deformity, and (4) jersey finger.</p>	<p>Mallet finger: digital extensor tendon disruption ± avulsion fx, unable to extend DIP joint Boutonniere deformity: slip of central extensor tendon at PIP joint → PIP in flexion + DIP in hyperextension Swan neck deformity: PIP in hyperextension + DIP in flexion Jersey finger: (usually ring finger) flexor digitorum profundus avulsion 2/2 hyperextension during active flexion, unable to flex DIP</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Gamekeeper's thumb</p>	<p>Dx: tear/sprain of ulnar collateral ligament SSx: weak pincer grasp, laxity with valgus stress Tx: thumb spica splint, hand surgery referral</p>
<p>What is a Bennett fracture-dislocation?</p>	<p>Two-part intra-articular fracture at the base of the 1st metacarpal *Note: Requires surgical repair.</p>
<p>What is a Rolando fracture?</p>	<p>Comminuted intra-articular fracture at the base of the 1st metacarpal *Note: Requires surgical repair.</p>
<p>Risk Factors, Diagnosis, Treatment: Carpal Tunnel Syndrome</p>	<p>Risk factors: obesity, female gender, hypothyroidism (NOT prolonged computer work) 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 Tx: wrist splint at night, NSAIDs, hand surgery referral PRN *Note: Carpal tunnel is the most common entrapment neuropathy of the wrist, and occurs 2/2 compression of the median nerve in carpal tunnel.</p>

<p>What are Kanavel's signs for flexor tenosynovitis? What is the treatment for flexor tenosynovitis?</p>	<p>1. Tenderness along course of the flexor tendon 2. Fusiform ("sausage digit") or symmetrical swelling of the finger 3. <u>Pain with passive extension</u> 4. <u>Finger held in flexion.</u> Treatment is IV broad-spectrum antibiotics, hospital admission, and immediate hand surgery consultation for I&D in the OR.</p>
<p>Diagnosis, Treatment: Compartment syndrome</p>	<p>Dx: classic "6 Ps" are typically later findings; <u>pain out of proportion</u> (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 \geq30-40. Delta pressure (more reliable than direct pressure alone) = Diastolic BP - direct pressure; \leq30 consistent with acute compartment syndrome. Tx: fasciotomy</p>
<p>Treatment: High-pressure injection injury</p>	<p>Hand surgery consultation for OR (even benign-appearing wounds have high likelihood of deep penetration)</p>
<p>What are the contraindications for finger reimplantation?</p>	<p>Mangled tissue, >6 hr elapsed since injury, fingertip amputation only</p>
<p>How should an amputated digit be transported?</p>	<p>Wrap in saline-soaked gauze, place in plastic bag, put bag in ice water. DO NOT put digit directly on ice - causes tissue injury.</p>
<p>List possible red flag symptoms for low back pain.</p>	<p>Trauma, fever, spinal surgery, focal neuro deficits, HIV/immunosuppression, weight loss, TB, cancer, age >55, symptoms >4 wks, IVDU, pain at rest or mostly in the evening, saddle anesthesia, constipation/urinary retention, urinary incontinence</p>
<p>Management: Low back pain (1) without and (2) with red flag symptoms</p>	<p><u>NO RED FLAGS:</u> short course of pain control (NSAIDs); early return to work; no imaging or additional workup needed <u>+RED FLAGS:</u> MRI</p>
<p>Demographic, Signs & Symptoms, Diagnosis, Treatment, Associations: Ankylosing spondylitis</p>	<p>Demographic: age 30s-40s; male > female SSx: morning back pain/stiffness; improved by motion Dx: xray shows "bamboo spine" (fusion of vertebrae) Tx: physical therapy; NSAIDs Associations: anterior uveitis and irritable bowel; genetic link to <u>HLA-B27</u></p>
<p>What finding is most sensitive for diagnosis of cauda equina syndrome?</p>	<p><u>Urinary retention:</u> post-void residual (PVR) >50-100cc Other SSx: saddle anesthesia, sexual dysfunction, neuro deficits, bowel/bladder dysfunction, BILATERAL symptoms</p>

<p>Identify the spinal cord level and nerve associated with each reflex: Biceps, Brachioradialis, Triceps, Patellar, Achilles, Babinski</p>	<p>Biceps: C5, musculocutaneous nerve Brachioradialis: C6, radial nerve Triceps: C7, radial nerve Patellar: L3-4, femoral nerve Achilles: S1, tibial nerve Babinski: L4-5 and S1-S2, tibial nerve</p>
<p>Diagnosis, Complication, Treatment: Unstable pelvic fracture</p>	<p>Dx: xray 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) Complication: may cause extensive blood loss, mostly posterior/retroperitoneal and venous Tx: pelvic binder, angioembolization or surgery, blood transfusion to temporize</p>
<p>What is the key landmark for application of a pelvic binder?</p>	<p>Wraps around the greater trochanters (often mistakenly placed higher)</p>
<p>Cause, Signs & Symptoms, Diagnosis, Treatment: Hip fractures</p>	<p>MCC = ground level fall SSx: high risk in elderly (esp. femoral neck), intertrochanteric fx = most common Exam: ABducted + externally rotated + shortened (classic) Dx: xray shows most; MRI if neg xray + high suspicion + unable to ambulate Tx: Ortho consult, open reduction and internal fixation (ORIF)</p>
<p>What is the most common type of hip dislocation? What associated injuries and sequelae should be anticipated?</p>	<p>MC = posterior (80-90%); usually due to high-energy force (e.g. MVC). Posterior dislocation on exam: hip flexed, ADducted, internally rotated & shortened Tx: neurovascular compromise + obvious dislocation → reduction without delay or xray; neurovascularly intact → xray Complications: delay in reduction >6 hrs → avascular necrosis</p>
<p>What is the timeline for reduction of a dislocated hip? What risks are associated with delayed reduction?</p>	<p>Needs to be reduced within 6 hours (delayed reduction → avascular necrosis)</p>
<p>What structures are often injured in posterior and anterior hip dislocations?</p>	<p>Posterior: sciatic nerve injury or acetabular fx Anterior: femoral artery/vein/nerve injury</p>
<p>What neurological findings are most likely to present with a posterior hip dislocation?</p>	<p>Sciatic nerve palsy Peroneal branch is more likely to be injured than the tibial branch On exam: foot drop (weakness w/ ankle dorsiflexion and foot eversion) + decreased sensation along the entire posterior leg below the knee</p>

<p>What are the Ottawa Knee Rules?</p>	<p>Age >55 Isolated patellar tenderness Tenderness over fibular head Inability to flex knee 90° Inability to bear weight (4 steps) immediately after injury AND in the ED If patient meets any of these criteria → get xray</p>
<p>Presentation, Diagnosis, Treatment: Osgood-Schlatter syndrome</p>	<p>Presentation: tibial tuberosity apophysitis 2/2 trauma or overuse, occurs mainly in preteens (M>F) Dx: localized tenderness, no xray needed (but may show avulsion fx of the tibial tuberosity) Tx: rest, ice, compression, elevation (RICE)</p>
<p>Cause, Diagnosis, Treatment: Meniscal injury</p>	<p>Occurs 2/2 rotational force. Dx: joint line tender to palpation; feeling of "clicking" and "locking" with knee giving out ± knee effusion; +Apley Grind Test and +McMurray's; confirmed by outpatient MRI Tx: rest/ice/compression/elevation (RICE), NSAIDs, surgery (refractory sx)</p>
<p>Cause, Signs & Symptoms, Diagnosis, Treatment, Complication: Cruciate ligament injuries</p>	<p>Occurs when pivoting while running/cutting. SSx: audible "pop," followed by knee instability, hemarthrosis; ACL tear = most common Dx: +Lachman's test (most sensitive test), xray ± outpatient MRI Tx: non-weight bearing, ortho referral, leg immobilizer <u>only</u> if the joint is very unstable ACL tears are associated with Segond fx (avulsion at lateral tibial plateau; treated with immobilization).</p>
<p>Cause, Signs & Symptoms, Diagnosis, Treatment, Complications: Tibial plateau fx</p>	<p>Occurs with high force blow to tibia (e.g. MVC or pedestrian vs. auto). SSx: localized tenderness to palpation and swelling Dx: xray often negative, <u>get CT if high clinical suspicion</u> Tx: knee immobilizer, crutches (pt should be totally non-weight bearing), and ortho consult for open reduction and internal fixation (ORIF) Complications: compartment syndrome, popliteal artery injury</p>
<p>Signs & Symptoms, Diagnosis, Treatment, Complications: Knee dislocation</p>	<p>SSx: 50% spontaneously relocate prior to arrival; maintain high index of suspicion; check for bicruciate instability on exam; most common = anterior dislocation (tibia anterior to femur) Dx: CTA = study of choice Tx: knee immobilizer, ortho consult Complications: popliteal artery injury, common peroneal nerve injury (foot drop) *Note: Don't confuse with patella dislocation!</p>
<p>What nerve injury is often associated with proximal fibula and tibial plateau fractures?</p>	<p>Deep peroneal nerve Patient will be unable to dorsiflex and have loss of sensation to 1st web space.</p>

<p>What are the Ottawa Ankle Rules?</p>	<p>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 If patient meets any of these criteria → get xray</p>
<p>Which ligament is most commonly injured in an ankle sprain?</p>	<p>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.</p>
<p>Diagnosis, Treatment: Maisonneuve fracture</p>	<p>Mechanism: ankle eversion Dx: xray with medial malleolus fx (or deltoid ligament injury) + proximal fibular fracture Tx: requires splint, non-weight bearing, and ortho referral for open reduction and internal fixation (ORIF)</p>
<p>Diagnosis, Treatment: Jones fracture (vs. pseudo-Jones fracture)</p>	<p>Jones fracture: fx of metaphysis of the 5th metatarsal (1.2-3 cm distal to proximal tuberosity) Tx: splint and non-weight bearing, usually requires surgery Pseudo-Jones/dancer's fracture: due to avulsion at the base of the 5th metatarsal Tx: splint and ortho follow-up, nonsurgical</p>
<p>Definition, Mechanism, Diagnosis, Treatment: Lisfranc fracture/dislocation</p>	<p>Definition: unstable midfoot due to disruption of the Lisfranc joint (arch of the foot) Mechanism: high-energy shearing force (e.g. falling from a horse with your foot in a stirrup) or with axial loading onto a hyper-plantar flexed foot Dx: xray (obtain AP/lateral/oblique films), Fleck sign (avulsion fx of 2nd metatarsal = pathognomonic), consider stress view if high suspicion Tx: splint, non-weight bearing, and most require surgery</p>
<p>Association, Risk Factors, Signs & Symptoms, Diagnosis, Treatment: Achilles tendon rupture</p>	<p>Association: landing from jumping (e.g. in basketball) Risk factors: chronic steroids, fluoroquinolones SSx: audible pop over Achilles tendon, unable to plantarflex Dx: consider US for dx of partial tears, +Thompson squeeze test (most sensitive sign) Tx: Splint in equinus (plantar flexion), complete tears require surgery</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Septic arthritis</p>	<p>SSx: acute joint pain (on passive ROM), erythema, warmth, swelling Dx: arthrocentesis; use Kocher criteria in kids (see Peds section); typically >50k WBC's Tx: IV abx, ortho consult with surgical washout</p>

<p>In a patient with septic arthritis, what are the expected findings on synovial fluid analysis?</p>	<p>Purulent/yellow/green synovial fluid WBC >50k PMNs >75% Glucose <25 Culture positive</p>
<p>What is the most common location of septic arthritis? What is the most common cause?</p>	<p>Knee = most common joint (50%) Hematogenous spread = most common Bacterial = most common cause Staph aureus = most common overall Neisseria gonorrhoea = most common in young, sexually active adults <35</p>
<p>Cause, Signs & Symptoms, Diagnosis, Treatment: Osteomyelitis</p>	<p>Children: hematogenous, monomicrobial Adults: contiguous, mono-/polymicrobial S. Aureus = most common overall; increased risk of Salmonella in sickle cell SSx: local tenderness, warmth, erythema, swelling, systemic sx (fevers) Dx: elevated ESR/CRP (not specific), bone biopsy with bacterial culture (definitive dx), xray may show periosteal elevation or bony erosions, negative xray → can obtain MRI (more sensitive) Tx: IV abx, surgery consult (debridement)</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Necrotizing fasciitis</p>	<p>Definition: rapidly progressing infxn of the fascia with necrosis of the subcutaneous 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 (MC finding), crepitus, dusky blue skin with bullae/vesicles Dx: CLINICAL dx (CT or xray may show subcutaneous emphysema) Tx: broad spectrum IV abx (including clindamycin for antitoxin effects) AND surgical debridement (definitive tx)</p>
<p>What is the most common primary bone cancer? How is it diagnosed?</p>	<p>Multiple Myeloma ("CRAB"): hyperCalcemia, Renal failure, Anemia, Bone lesions/Back pain Dx: abnormal SPEP (M-spike) & UPEP (Bence-Jones protein), peripheral smear: rouleaux formation, cranial xray with "punched out lesions" Complications: hypogammaglobulinemia (leads to sepsis), hyperviscosity syndrome *Note: Most bone tumors are metastatic and located in the spine (prostate, breast, kidney, thyroid, skin).</p>

<p>Location, Signs & Symptoms, Diagnosis, Treatment: Osteosarcoma</p>	<p>Location: metaphysis of long bones (distal femur = most common site, tibia, humerus) Bimodal age distribution: teens & >65 years Associated with radiation for childhood cancer. SSx: persistent bone pain (worse at night) Dx: xray showing lytic lesion at metaphysis with "sunburst" pattern and/or Codman's triangle (elevation of periosteum at periphery of tumor); labs may show elevated Alk Phos, LDH, and ESR *Osteosarcoma is the second most common primary bone cancer.</p>
<p>What features suggest Ewing sarcoma?</p>	<p>Painless mass in shaft of femur Occurs in adolescence (M>F) Xray showing "onion peel" pattern</p>
<p>Describe the crystals associated with gout and pseudogout.</p>	<p>Gout: negatively birefringent, needle-like crystals (urate) Pseudogout: positively birefringent, rhomboid crystals (calcium pyrophosphate)</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Polymyositis</p>	<p>Inflammation of striated muscle SSx: proximal limb and neck muscle weakness; similar to dermatomyositis but NO rash; strong association with malignancy Dx: elevated ESR/CRP, CPK, and aldolase Tx: high dose steroids, methotrexate</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Dermatomyositis</p>	<p>Inflammation of striated muscle SSx: proximal limb and neck muscle weakness, +heliotrope rash around the eyes; strong association with malignancy Dx: elevated ESR/CRP and CPK Tx: methotrexate</p>
<p>Signs & Symptoms, Association, Diagnosis, Treatment: Polymyalgia rheumatica</p>	<p>SSx: bilateral, symmetric proximal ("cape-like" distribution) muscle weakness, stiffness worse in AM Association: temporal arteritis/giant cell arteritis (GCA) Dx: elevated ESR, rheum consultation Tx: steroids</p>
<p>Definition, Association, Signs & Symptoms, Diagnosis, Treatment: Reactive arthritis (formerly Reiter's Syndrome)</p>	<p>Definition: seronegative arthritis occurring after an infection (classically chlamydia) Associated with HLA-B27. SSx: conjunctivitis, urethritis, asymmetric arthritis ("can't see, can't pee, can't climb a tree") Tx: NSAIDs, physical therapy, treat underlying infection</p>

<p>Signs & Symptoms, Diagnosis, Treatment: Rheumatoid arthritis</p>	<p>SSx: polyarticular, symmetric, deforming arthritis; classically affecting the hands (ulnar deviation of fingers, Boutonniere & swan neck deformities) but sparing the DIP joint Dx: elevated rheumatoid factor (70-80%, but also present in 5-10% of healthy population) or anti-CCP, elevated ESR/CRP, xray showing bony destruction Tx: NSAIDs, disease-modifying anti-rheumatic drugs (DMARDs), steroids **Note: associated with atlantoaxial joint instability: DO NOT hyperextend neck with intubation**</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Juvenile idiopathic arthritis (JIA)</p>	<p>Systemic JIA (Still's disease): SSx: daily fever, myalgias, polyarthritis, "salmon-pink rash", anemia, thrombocytosis Dx: ANA neg, RF neg Tx: NSAIDs, steroids, methotrexate Polyarticular JIA: SSx: symmetric chronic arthritis, ≥5 joints, mild systemic sx, uveitis (rare) Tx: NSAIDs, steroids, methotrexate Pauciarticular JIA: SSx: chronic arthritis in 1-4 joints, +uveitis Dx: ANA pos, RF neg, ESR normal Tx: NSAIDs, steroids, methotrexate</p>
<p>Signs & Symptoms, Diagnosis, Treatment: Psoriatic arthritis</p>	<p>SSx: symmetric polyarthritis, "sausage digits" (dactylitis), nail pitting (skin lesions usually precede joint disease) Dx: anemia, RF usually neg, ANA pos, xray with erosion AND new bone formation, "pencil in cup" deformities Tx: NSAIDs, DMARDs, NO STEROIDS (causes pustular psoriasis)</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Wegener's granulomatosis (granulomatosis with polyangiitis)</p>	<p>Definition: medium vessel vasculitis with upper & lower respiratory tract involvement + renal failure SSx: sinusitis, epistaxis, hemoptysis (pulmonary infiltrates), nephritis (hematuria), cutaneous nodules, palpable purpura Dx: +c-ANCA Tx: steroids, DMARDs</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Goodpasture's syndrome</p>	<p>Definition: small vessel vasculitis SSx: cough/dyspnea, hemoptysis from alveolar hemorrhage, glomerulonephritis, hematuria Dx: anti-basement membrane antibodies Tx: steroids, DMARDs, plasmapheresis</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Churg-Strauss syndrome</p>	<p>Definition: medium vessel vasculitis = vasculitis + eosinophilia + asthma SSx: bronchospasm, sinusitis, possible cardiac + GI symptoms Dx: peripheral eosinophilia Tx: steroids, DMARDs</p>

<p>Definition, Associations, Signs & Symptoms, Diagnosis, Treatment: Polyarteritis nodosa</p>	<p>Definition: medium-vessel vasculitis with multiorgan involvement but <u>sparing the lungs</u> 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+ Tx: high-dose steroids, DMARD</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Systemic lupus erythematosus</p>	<p>Definition: autoimmune chronic inflammatory disease with multiorgan involvement, high risk of thrombosis (ACS, PE) On test, patient will be an African-American female. Can be drug-induced ('HIPPS'): <u>H</u>ydralazine, <u>I</u>NH, <u>P</u>rocaïnamide, <u>P</u>henytoin, <u>S</u>ulfonamides SSx (≥4): classic "butterfly" malar rash, or discoid rash, photosensitivity, oral ulcers, arthritis, renal disease (nephritis), encephalopathy (seizures or psychosis), serositis (pericarditis), cytopenia (any cell line) Dx: ANA+ (sensitive), anti-dsDNA+ (specific), anti-Smith, antiphospholipid antibody Tx: NSAIDs, steroids, DMARDs</p>
<p>Signs & Symptoms, Cause, Treatment: Scleroderma (systemic sclerosis)</p>	<p>SSx: fatigue, stiff joints, loss of strength, pain, sleep disturbances, CREST syndrome (Calcinosis, Raynaud's syndrome, Esophageal dysmotility, Sclerodactyly, Telangiectasias), renal failure (most common cause of death) presenting with HTN Cause: collagen deposition in skin + other organs Tx: supportive, rewarm digits, calcium channel blockers</p>
<p>Definition, Signs & Symptoms, Diagnosis, Treatment: Sjögren syndrome</p>	<p>Definition: autoimmune chronic inflammation of salivary & lacrimal glands SSx: dry eyes and xerostomia (dry mouth) Dx: Anti-Ro & Anti-La antibodies, RF+, ANA+, Schirmer's test (tests tear production) Tx: lubricants, pilocarpine, DMARDs</p>
<p>Definition, Signs & Symptoms, Diagnosis: Rhabdomyolysis</p>	<p>Definition: 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: elevated CK (>5x upper limit normal), UA (+blood, but NO RBCs), electrolyte abnormalities (hypocalcemia = most common, hyperK, hyperPhos) Tx: aggressive rehydration targeted to UOP goals, +/- bicarb infusion, +/- hemodialysis *Note: get an ECG on all patients!</p>

Treatment: Rhabdomyolysis	IVF with UOP 3cc/kg/hr , ± bicarb gtt to alkalinize urine (controversial & may not be helpful) Goal is to prevent renal failure (d/t ATN). Relative endpoint is CK <1000 (although the initial CK level does not correlate with the likelihood of renal failure).
What feature of rheumatoid arthritis can complicate potential intubations?	Atlantoaxial joint instability (don't hyperextend with intubation)
Diagnosis: Non-caseating granulomas in multiple organs	Sarcoidosis (commonly eyes and chest, skin lesions)
What characteristic laboratory abnormalities may be found in a patient with sarcoidosis?	1. Elevated ACE levels 2. Decreased PTH 3. Hypercalcemia/hypercalciuria
To perform arthrocentesis , why is the extensor surface most commonly used?	Extensor surfaces avoid neurovascular structures that typically overlie the flexor aspect of joints
On fluid analysis, what white blood cell count indicates (1) septic joint and (2) septic bursitis?	1) Septic joint: >50,000 2) Septic bursitis: >10,000
What is the common rotator cuff injury ?	Supraspinatus (responsible for first 30 degrees of abduction)
What is the most common adult wrist fracture ?	Colles Fracture
Which additional injury is often associated with Colles fracture?	Ulnar styloid fracture (present in 60-70% of cases)
Which nerve is most commonly injured in Colles fracture?	Median nerve
Overuse of which muscle group is responsible for lateral epicondylitis (tennis elbow)?	Extensor muscles most commonly (extensor carpi radialis brevis)
Which lower extremity fracture is most commonly associated with compartment syndrome?	Proximal tibial fracture

Immunology

Bizz	Buzz
What is the difference between anaphylaxis and anaphylactoid reactions?	Anaphylaxis: IgE mediated Anaphylactoid: histamine release independent of IgE Both receive the same treatment .
Presentation, Treatment: Anaphylaxis	Presentation: 2+ organ system involvement (e.g., bronchospasm, hypotension, urticaria, GI sx) Usually occurs within 60 min of exposure. Tx: ABCs (intubate PRN), IV, supplemental O ₂ , cardiac monitor, Epinephrine (1:1000) 0.3 mg IM (not subQ), steroids, H1/H2 blockers Refractory Tx: epi drip, glucagon (pt on beta blockers), vasopressors
What medication should be given for a patient on beta blockers who develops anaphylaxis ?	Glucagon (patients on beta blockers may not respond to epi)
Mechanism: Angioedema	Hereditary: deficiency or dysfunction of C1-esterase inhibitor Drug-induced: ACE-I/ARB, ↑ bradykinin
Signs & Symptoms, Treatment: Angioedema	SSx: painless, non-pruritic, non-pitting edema of skin (NO rash) May affect abdominal organs & upper airway. Tx: supportive , intubate pm, give standard anaphylaxis Tx (unlikely to work), FFP (contains C1 esterase)
Mechanism, Example: Type I-IV hypersensitivity reactions	I: IgE-mediated , requires 2 separate exposures; e.g. anaphylaxis/urticaria II: cytotoxic antibody (IgG & IgM-mediated); e.g. hemolytic transfusion reaction III: IgG-immune complex deposition; e.g. serum sickness & vasculitis IV: T-cell mediated (no antibodies), delayed ; e.g. Stevens-Johnson syndrome, TB skin test
Diagnosis: Serum sickness	Dx: clinical, ↓ C3/C4 Type III rxn ; classically occurs after meds (PCN, sulfas)
Signs & Symptoms, Treatment: Serum sickness	SSx: fever, rash (fingers/toes → morbilliform), arthralgias Tx: supportive care
Management: s/p transplant + sick	Assume infection AND rejection (they look the same); immune response to infection may be blunted by anti-rejection meds. Cultures (blood, urine, sputum, +/- CSF), broad spectrum abx
Signs & Symptoms, Treatment: 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 Also important: renal US with doppler (remember most transplanted kidneys are in the pelvis)
What are the most likely sources of infection in transplant patients in the following periods post-transplant: <1mo, 1-6mos, 6+mos?	<1 mo: infection related to procedure and hospitalization such as wound infections (Strep, Staph/MRSA, Pseudomonas) 1-6 mos: viruses (CMV, EBV) >6 mos: 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 ?	Hyperacute: minutes to hours after transplant, 2/2 preformed antibodies causing irreversible graft destruction (esp. ABO mismatch) Acute: 1-2 wks, humoral/T-cell mediated Chronic: months to years
Diagnosis, Treatment: Pruritus and erythematous rash while receiving Vancomycin	Vancomycin Flushing Syndrome (previously Red Man Syndrome), anaphylactoid reaction Tx: stop infusion; diphenhydramine; may restart if sx resolve (to r/o anaphylaxis) at a slower rate
Signs & Symptoms, Diagnosis, Treatment: Systemic Lupus Erythematosus (SLE)	SSx: arthritis, malar rash ("butterfly rash"), fever, lymphadenopathy, weight loss, general malaise Dx: +ANA (sensitive), +anti-dsDNA Ab or anti-smith Ab (specific), anti-histone Ab, thrombocytopenia On boards, patient is likely to be an African-American female. Tx: NSAIDs, steroids, immunosuppressants, hydroxychloroquine
What drugs/drug classes are commonly implicated in Drug-induced Systemic Lupus Erythematosus ?	Hydralazine, INH, Procainamide, Phenytoin, Sulfonamides (HIPPS)
Diagnosis, Treatment: Elderly woman with monocular vision loss, unilateral HA, jaw claudication, tender temple	Temporal Arteritis (Giant Cell Arteritis); associated with polymyalgia rheumatica (PMR) Dx: ESR > 50, temporal artery biopsy Tx: high-dose IV steroids ASAP (don't wait for biopsy results).
Common lab findings of sarcoidosis	↑↑ ACE levels, ↑ Calcitriol production (1, 25-dihydroxyvitamin D, active metabolite of Vitamin D), Hypercalcemia (MCC of acute renal failure in sarcoidosis), Hypercalciuria, ↓ PTH levels
Common presentation of renal transplant rejection	↑ Cr, tenderness around graft (likely in pelvis), ↓ urine output
Common presentation of lung transplant rejection	Cough, chest tightness
Common presentation of heart transplant rejection	Fatigue, decompensated heart failure, unlikely to have angina/chest pain
Common presentation of liver transplant rejection	Fever, abnormal LFTs, RUQ pain, jaundice
Treatment: Transplant rejection	Steroids

Common presentation of Takayasu's arteritis	Asian predominance, decreased/ asymmetric pulses , aortic involvement
Infection associated with Polyarteritis Nodosa	HBV
Common presentation of Buerger's disease	Claudication of hands/feet, arterial ulcers Population: Smokers
Common presentation of Granulomatosis with polyangiitis (GPA)	Upper (otitis media, sinusitis, mucosal ulceration, epistaxis) & lower respiratory sx (cough, dyspnea, hemoptysis) + renal (renal failure, GN), +c-anca
Common presentation of Microscopic polyangiitis	Lower respiratory sx and renal dysfunction - similar to GPA but without nasopharyngeal involvement, +p-ANCA
Common presentation of Churg-Strauss syndrome	Vasculitis + eosinophilia + asthma AKA eosinophilic granulomatosis with polyangiitis (EGPA), +IgE
Common associations with Cryoglobulinemia	HCV , malaise, skin lesions, arthralgias
Common presentation of Behçet's disease	Oral and genital ulcers , pathergy (hyperreactivity to needle sticks)
What mediastinal mass is associated with Myasthenia Gravis ?	Thymoma
In a myasthenic crisis , what test is important to measure to determine severity?	Forced vital capacity and/or negative inspiratory force

Environmental

Bizz	Buzz
<p>Signs and Symptoms, Treatment: <u>chilblains</u> (pernio) vs <u>trench foot</u> (immersion foot)</p>	<p>Both are <u>NON-freezing</u> cold injuries. Chilblains: inflammatory lesions resulting from exposure to DRY and cold SSx: red/blue edematous plaques with itchy, burning pain Tx: warming, drying, topical steroids Trench foot: nerve and tissue injury resulting from repetitive exposure to WET and cold temperatures → vasoconstriction → ischemia/gangrene Tx: warming, drying, prevention with dry footwear</p>
<p>Diagnosis, Treatment: <u>frostnip</u> vs. <u>frostbite</u></p>	<p>Both are <u>FREEZING</u> cold injuries. Frostnip: no ice formation or tissue loss Tx: rewarming Frostbite: most common freezing injury. Intracellular ice forms and causes tissue loss (can't initially distinguish the from frostnip). Tx: rewarming in warm water immersion in a circulating bath (37°C - 39°C), NO DRY HEAT EVER, pain control, Tdap as needed, delayed debridement Blister treatment: debride clear blisters, leave hemorrhagic blisters (simple wound care).</p>
<p>What are the phases of frostbite? How is severity graded?</p>	<p>Phase I: vasoconstriction/ice formation Phase II: reperfusion of warmed tissues → edema/blisters & dry gangrene Severity: graded by tissue death Grade I: epidermis; erythema & edema; minimal pain with rewarming Grade II: epidermis/dermis; hard edema + clear blisters; mild to moderate pain with rewarming Grade III: hypodermis; hemorrhagic blisters, pale grey extremities; severe pain with rewarming Grade IV: skin/muscle/tendon/bone; insensate, black/grey painless during rewarming</p>
<p>Distinguish the stages of hypothermia by the temperature ranges and symptoms observed.</p>	<p>Mild (32-35° C): amnesia, dysarthria, shivering Moderate (28-32° C): stupor, dysrhythmias, AMS, <u>NO shivering</u> Severe (22-28° C): dysrhythmias (susceptible to VF), loss of reflexes, pulmonary edema, cold diuresis, obtunded Profound (9-20° C): flat EEG, asystole</p>
<p>Review common physiologic changes in <u>hypothermia</u>.</p>	<p>Hyperglycemia (don't treat initially), functional coagulopathy (clotting factors stop working), irritable myocardium, OxyHb curve shifted to left (↑ O₂ affinity + ↓ O₂ delivery)</p>

<p>Review appropriate treatment of severe hypothermia.</p>	<p>Temp > 30° C and hemodynamically stable: rewarm passively (warm room, warm/dry clothes, insulating blanket) Temp < 30° C or hemodynamically unstable: active rewarming (Bair Hugger, hot packs, warm O2/IVF, bladder/stomach/peritoneal/chest tube lavage; ECMO is most effective) Very unstable: ABCs: CPR/intubation, one round of epinephrine/defibrillation while cold, rewarm to at least 32° C during arrest - "Not dead until they're warm and dead"</p>
<p>Pathophysiology and prevention: altitude sickness</p>	<p>Unclear but likely related to hypoxia causing pulmonary vasoconstriction & pulmonary hypertension → pulmonary edema; cerebral vasodilation → headaches/acute mountain sickness & ultimately altered mental status/cerebral edema. Acclimatization causes hyperventilation → respiratory alkalosis + bicarbonate diuresis. Prevention: acetazolamide causes bicarbonate 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).</p>
<p>Signs and Symptoms, Treatment: acute mountain sickness</p>	<p>Most common high altitude illness. Occurs when: > 2000 meters (6500 feet). SSx: headache, nausea/vomiting, anorexia, insomnia Tx: stop ascent, supportive care (fluids, analgesia, antiemetics), acetazolamide/steroids, descent if possible</p>
<p>Signs and Symptoms, Diagnosis, Treatment: high altitude pulmonary edema (HAPE)</p>	<p>Most common lethal altitude illness. Occurs when: > 3000 meters (9500 feet). SSx: initial (2-4 days at new altitude): cough, dyspnea on exertion. Progresses to dyspnea at rest (classic), pink-frothy sputum, ↑HR, ↑RR, fever, hypoxia Dx: CXR with patchy alveolar infiltrates (ARDS-like) Tx: Immediate descent, supplemental oxygen, nifedipine or tadalafil (for pulmonary hypertension), portable hyperbaric chamber **Acetazolamide is NOT helpful in acute illness**</p>
<p>Diagnosis, Treatment: high altitude cerebral edema (HACE)</p>	<p>Most severe altitude sickness but uncommon. Occurs when: > 4500 meters (14000 feet). SSx: ataxia (early and most sensitive finding), encephalopathy, severe lassitude, altered mental status, seizure Tx : requires immediate treatment, descent = definitive (best when patient is still ambulatory), supplemental O2, steroids (rescue treatment), portable hyperbaric chamber (e.g. Gamow bag)</p>

<p>What causes barotrauma (diving) related illness?</p>	<p>Illness secondary to descent/ascent. Explained by <u>Boyle's Law</u> (gas volume = 1/pressure). Volume change is greater closer to surface (rapid change of 30 feet near surface worse than deeper down).</p>
<p>Review injuries related to descent (localized "squeeze") and appropriate treatment.</p>	<p>Barotitis Externa: edema and hemorrhage to external auditory canal secondary to blockage Tx: cortisporin, dry ear precautions</p> <p>Barotitis Media: most common diving related disorder SSx: pain, vertigo secondary to TM rupture Tx: decongestants, antibiotics, dry ear precautions</p> <p>Barotitis Interna: rupture/bleeding of round window SSx: decreased hearing, vertigo, nystagmus Tx: ENT consult/evaluation</p> <p>Sinus squeeze: frontal sinus is most commonly affected. Sinuses = second most common cause "squeeze" injuries. SSx: edema, pain, epistaxis Tx: decongestants</p> <p>Mask squeeze: petechiae and subcutaneous hemorrhages</p>
<p>Review injuries related to ascent (localized "reverse squeeze") and appropriate treatment.</p>	<p>Barodontalgia: air in dental cavity/filling expands with ascent and causes pain, tooth may fall out.</p> <p>GI barotrauma: excess intraluminal gas causes burping/flatus.</p> <p>Pulmonary Overpressurization Syndrome ("POPS"): pulmonary alveolar rupture → pneumomediastinum, ± pneumothorax SSx: crepitus, SOB, chest pain Tx: supplemental O2, supportive (needle decompression as needed)</p> <p>Arterial Gas Embolism ("AGE"): rupture of air or nitrogen into pulmonary vein + left heart → enter circulation → systemic emboli SSx: LOC on ascent OR within 10 minutes of surfacing; can cause ACS, CVA, etc Tx: 100% O2, supine positioning, hyperbaric O2 therapy *Note: POPS & AGE are caused by ascending without exhaling</p>
<p>What causes dissolved gas (diving) related illness?</p>	<p>Illnesses related to gas in tissue. Explained by <u>Henry's law</u> (↑ pressure → ↑ gas pushed into solution).</p>
<p>Review illnesses related to dissolved gas while diving and appropriate treatment.</p>	<p>Nitrogen narcosis: "rapture of the deep" (> 30 meters/100 feet). Breathing nitrogen at high partial pressures leads to ↑ nitrogen in CNS with anesthetic. SSx: acts drunk & dumb, may drown due to confusion/behavior</p> <p>O2 toxicity: ↑ pO2 with depth causes toxicity, usually with deep diving or using Nitrox SSx: muscle spasm, nausea, vision changes, seizure Tx: ascent or can prevent by decreasing %O2 in tank</p>

<p>Describe the two types of decompression sickness and their treatment.</p>	<p>Decompression sickness (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.</p> <p>Type I: musculoskeletal symptoms (most common: arthralgia/myalgias; most common affected shoulders and elbows ("The Bends"), cutis marmorata rash due to lymphatic obstruction</p> <p>Type II: pulmonary: dyspnea, chest pain, cough ("The Chokes"); neuro: vertigo, tinnitus, ataxia ("The Stagers"); spinal cord: paralysis, paresthesias; derm: pruritus, burning ("Skin Bends")</p> <p>Tx: supportive (IVF, O2, ASA), hyperbaric O2 (must do this quickly)</p> <p>Prevention: slow ascent with frequent stops (Navy dive tables)</p>
<p>What is the key to diagnosis of arterial gas embolism vs decompression sickness?</p>	<p>Timing - arterial gas embolism symptoms occur within minutes of surfacing, decompression sickness within hours.</p>
<p>Diagnosis, Treatment: heat syncope</p>	<p>Dx: standing in heat with peripheral pooling due to vasodilation and decreased preload causes syncope.</p> <p>Tx: passive cooling, fluids</p>
<p>Diagnosis, Treatment: heat cramps</p>	<p>Dx: muscle spasms due to dehydration and electrolyte depletion</p> <p>Tx: rest, passive cooling, fluid replacement, salt replacement</p>
<p>Diagnosis, Treatment: heat exhaustion</p>	<p>Dx: flu-like symptoms. No CNS changes. Core temp usually < 104° F.</p> <p>Tx: passive cooling, rest, IVF, replete electrolytes.</p>

<p>Signs and Symptoms, Diagnosis, Treatment: heat stroke. Discuss classic vs exertional types.</p>	<p>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, rhabdomyolysis, ATN, pulm edema <u>Types:</u> "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 Tx: rapid evaporative cooling is BEST (spray lukewarm water on body and use fans to help evaporate - cold water can cause shivering - prevent with low-dose benzodiazepines or thiorazine), ice packs, cold water GI lavage if intubated, IVF (small if "classic," more if "exertional"), AVOID pressors, <u>STOP COOLING at 39° C to avoid hypothermia overshoot</u> *Note: aspirin & tylenol do NOT help - the problem is hyperthermia, not fever.</p>
<p>When treating heat stroke, at what temperature do you stop active cooling measures?</p>	<p>39° C to avoid hypothermia overshoot</p>
<p>What are the key differences between heat exhaustion and heat stroke?</p>	<p>Heat exhaustion: temperature < 104° F, flu-like symptoms, NO neurologic symptoms Heat stroke: temperature > 104° F with neurologic symptoms</p>
<p>Describe how to differentiate and treat the different degrees of thermal burn injury.</p>	<p>1st degree: superficial, epidermis SSx: sunburn, redness, blanching, pain, no blisters (does NOT count towards TBSA) Tx: NSAIDs 2nd degree: partial thickness, upper dermis SSx: blistering with pain, intact sensation Tx: NSAIDs, topical antibiotics 3rd degree: full thickness, hypodermis; charred insensate, eschar formation Tx: skin grafting 4th degree: deep tissue, muscle/tendon/bone; painless Tx: skin grafting</p>
<p>Review how to use Rule of 9's (in adults) to calculate TBSA for burns.</p>	<p>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**</p>

<p>Review appropriate fluid resuscitation in thermal burns using the <u>Parkland Formula</u>.</p>	<p>Resuscitation volume = 4 mL/kg x TBSA% x weight (kg) Tx: LR, 50% in first 8 hours since time of burn, 50% over next 16 hours UOP goal 0.5-1 mL/kg/hr Children: resuscitation volume above + maintenance IVF; dextrose containing fluids for age < 5 Note: the Parkland Formula is HIGHLY tested, but Burn Surgeons now favor Brooke Formula for adults, and in particular, using UOP to guide fluid resuscitation.</p>
<p>Review appropriate fluid resuscitation in thermal burns using the <u>Brooke Formula</u>.</p>	<p>Resuscitation volume = 2 mL/kg x TBSA% x weight (kg) Tx: LR, 50% in first 8 hours since time of burn, 50% over next 16 hours UOP goal 0.5-1 mL/kg/hr</p>
<p>Signs and Symptoms, Treatment: Inhalation injury</p>	<p>Higher risk if enclosed space; inhaled toxins cause edema & loss of surfactant. SSx: cough, stridor, hypoxia, carbonaceous sputum but symptoms are often delayed Tx: intubate early due to anticipated airway edema. Also consider cyanide and carbon monoxide exposure, traumatic Injuries, rhabdomyolysis (esp. with electrical burns or 4th degree thermal burns, DIC)</p>
<p>Review key components of <u>treatment</u> of thermal burns.</p>	<p>Intubate early if needed (e.g., inhalational injury), IVF per Parkland formula, check for associated traumatic injuries, update tetanus, keep warm, and provide wound care; no need for prophylactic antibiotics.</p>
<p>Appropriate management of restrictive full thickness burns (respiratory compromise or decreased peripheral perfusion)?</p>	<p>Immediate escharotomy.</p>
<p>What are indications for referral to a burn center?</p>	<p>2nd degree/partial thickness burns > 10% TBSA; ANY 3rd/4th degree/full thickness burn; Burns involving hands, feet, genitals/perineum, face, major joints; Electrical/chemical/inhalational burns; all circumferential burns; patients with significant comorbidities.</p>
<p>What is the appropriate treatment for tar burns?</p>	<p>Cool tar with cold water to harden it and limit tissue damage. Remove with emulsifier (oils, butter, mayonnaise, neosporin, mineral oil). Needs to return to rinse in 24 hours, do not peel off - this worsens damage.</p>
<p>Review the key differences between high and low voltage <u>electrical injuries</u>.</p>	<p>Voltage = Current x Resistance. Household circuits are low voltage (110 V) but more accessible; high voltage/industrial (1000 V) 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 secondary to heat produced by resistance (bone, tendon).</p>

<p>Review the key differences between alternating current (AC) and direct current (DC) electrical injuries.</p>	<p>AC: more lethal; more extensive - 1,000-10,000 volts; back-and-forth current; electrical outlets - Symptoms include: sustained contractions (tetany): flexor > extensor → draws person to source, posterior shoulder dislocation; V Fib; burns - Myoglobinuria/fasciotomy: common DC: - 10 million - 2 billion volts; single direction; batteries/lightning - Single powerful muscle spasm with blow back → traumatic fracture as thrown from source; asystole (acts like defibrillator) - Myoglobinuria/fasciotomy: rare</p>
<p>Review primary clinical concerns with electrical injuries.</p>	<p>Arrhythmia/asystole, deep burns, rhabdomyolysis, associated trauma, vascular spasm and thrombosis, AMS/seizure, and delayed peripheral neuropathies.</p>
<p>Review appropriate management of electrical injuries.</p>	<p>Low voltage: Asymptomatic → home without testing Mild symptoms → cards monitor, ECG and UA (rule out rhabdomyolysis) → d/c if normal. High voltage: ALL patients: labs, UA, CPK, CT Head if AMS, tetanus for burns, admit.</p>
<p>Review clinical concerns and appropriate treatment for pediatric commissure burns of the mouth.</p>	<p>Association: kid chews on cord and gets burns at corner(s) of mouth. Concern for delayed bleeding of labial artery (day 5). Safe for discharge if no LOC, no other injury, normal ECG, tolerating PO and reliable parents - instruct parents to hold pressure and return if delayed bleed occurs. Patient will need outpatient follow up with plastics/OMFS for wound check and further care.</p>
<p>Review the general pathophysiology and signs/symptoms of lightning injuries.</p>	<p>Large DC voltage → asystole + apnea. Other signs and symptoms: steam burns, TM rupture (classic), superficial fern-shaped/branching burns (Lichtenberg figures), associated trauma, delayed cataracts.</p>
<p>How does disaster triage differ in lightning strikes?</p>	<p>Patients that are apneic and pulseless after a lightning strike are treated FIRST because they can survive with rescue breathing (otherwise black tagged in MCI triage).</p>
<p>Diagnosis, Treatment: leg numbness/weakness and cyanosis after lightning strike</p>	<p>Dx: Keraunoparalysis: current goes up one leg and down the other causing vasospasm and neuroparalysis Tx: spontaneously resolves after 6hr</p>
<p>What skin finding is pathognomonic for lightning injury?</p>	<p>Lichtenberg figure: superficial burn or feather pattern from "electrical shower"; usually resolves within 6 hours.</p>

<p>What is the usual cause of death in submersion injury?</p>	<p>Breath holding → involuntary gasp → aspiration / laryngospasm / loss of consciousness → active aspiration of fluid → loss of surfactant / hypoxia / ARDS → death. Also causes airway obstruction and metabolic acidosis (most common abnormality), delayed pneumonia. Consider associated trauma.</p>
<p>What is the mammalian diving reflex?</p>	<p>More common in children. Sudden submersion in cold water causes bradycardia, blood shunting to CNS, and slowed metabolism. ***This is why infants with SVT respond to ice bags to the face***</p>
<p>What is the appropriate treatment for submersion/near drowning?</p>	<p>Asymptomatic: monitor for 4-6 hours then likely discharge home Mild symptoms: normal SpO2 & CXR → home after 4-6 hour observation Moderate symptoms: hypoxia or abnormal CXR → admit for observation Apneic, unconscious, severe respiratory distress: apply BiPAP or intubate, consider ECMO, warm → admit to ICU</p>
<p>What is the difference in affect on the body between alpha, beta and gamma rays?</p>	<p>α- & β-particles = subatomic particles emitted during radioactive decay α-particles: larger, do not penetrate clothing or skin, dangerous if inhaled or ingested β-particles: smaller, can penetrate superficial skin layers Gamma rays: high energy electromagnetic radiation, dangerous in any form of exposure because they can penetrate tissues very deeply</p>
<p>What body systems are affected by whole-body radiation exposure at different levels?</p>	<p>Hematopoietic syndrome: > 2 Gy, first to show injury, most sensitive organ system SSx: pancytopenia (lymphopenia first) & infection; onset < 2 days GI syndrome: 2nd most sensitive system, > 6 Gy SSx: nausea, vomiting, diarrhea, GI bleed; onset hours CV/CNS syndrome: >10Gy SSx: dizziness, LOC, ataxia, coma, cerebral edema, CV failure</p>
<p>Which lab test is most prognostic in patient's with acute radiation syndrome?</p>	<p>Absolute Lymphocyte Count at 48 hrs is key to prognosis > 1500 indicates good prognosis < 1500 indicates significant exposure < 300 is expected to be lethal</p>
<p>Which medication can be taken before inhaled/ingested radioactive iodine to prevent thyroid cancer?</p>	<p>Potassium iodide.</p>
<p>What is the correct approach to decontamination of patient exposed to radiation?</p>	<p>Remove clothing (90%), wash with soap/water, don't abrade skin.</p>

<p>Which bacteria cause infection in human, dog, and cat bites and what is the correct antibiotic therapy?</p>	<p>Human: <i>Eikenella corrodens</i> (Think HACEK bacteria); Dog & cat: <i>Pasteurella multocida</i>. Tx: amoxicillin-clavulanate or ampicillin-sulbactam; 2nd line: doxycycline. Dog bites cause crush injuries; cat bites cause puncture wounds (caution near joints), but have equal rates of infection.</p>
<p>Which infection is most concerning after a primate bite? What are the symptoms and treatment?</p>	<p>Primates carry herpes simiae ("B virus"). Fatal in humans if not treated early. SSx: paresthesias (early) followed by vesicular rash and encephalitis Tx: IV acyclovir</p>
<p>Appropriate management of dog bites?</p>	<p>Thorough irrigation and decontamination. Rule out retained FB and underlying fracture. Consider closure for bites to the face within 6 hours of injury. No closure if crush wound or wound on the hands/feet. May consider closing large gaping wounds outside the face with loose sutures. Antibiotics: augmentin or doxycycline. Tetanus update.</p>
<p>Which type of arthropod bites/stings are most concerning?</p>	<p>Hymenoptera (bees, wasps, hornets, ants): venom contains histamine and proteins potentially leading to anaphylaxis.</p>
<p>Review the following sting reactions: local, toxic, anaphylactic, delayed.</p>	<p>Local: redness, swelling, pain (e.g. typical bee sting) Toxic: > 10 stings (killer bees, fire ants), syncope, headache, nausea/vomiting; can resemble anaphylaxis but WITHOUT generalized hives/edema or bronchospasm Anaphylaxis: onset within minutes, includes bronchospasm, hypotension, urticarial rash Delayed: like serum sickness with arthralgias, fever, malaise, occurring 1-2 weeks later</p>
<p>What is the appropriate treatment for Hymenoptera stings?</p>	<p>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 antibiotics are not necessary (though initially stings may look infected).</p>
<p>Signs and Symptoms, Treatment: brown recluse bites</p>	<p>Brown recluse (<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 hemeatologic abnormalities (hemolysis, DIC) Tx: supportive (wound care), tetanus, NO antivenin</p>

<p>Signs and Symptoms, Treatment: black widow bites</p>	<p>Black widow (<i>Latrodectus</i>): yellow/red "hourglass" on belly, warm/dry places across the US; they tend to be more aggressive SSx: painful bite, venom is <u>neurotoxic</u> → ACh + NE release → painful muscle cramping (can mimic acute abdomen), CNS excitation, sweating Tx: supportive (opioids, benzos), antivenin only for severe pain (risk of anaphylaxis)</p>
<p>Signs and Symptoms, Treatment: 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 <u>neurotoxic</u> (ACh + NE release). SSx: local sx (heightened sensitivity to touch = pathognomonic), sympathomimetic symptoms (hypertension, tachy, <u>hypersalivation</u>, bronchoconstriction), CNS symptoms (roving eye movements, <u>bulbar neuropathies</u>), somatic effects (fasciculations, muscle spasm) Tx: supportive (opioids, benzodiazepines), intubate as needed, atropine for secretions, antivenom as needed. Most likely fatal in children.</p>
<p>Distinguish signs and symptoms of Crotalid vs. Elapid snake bites.</p>	<p>Crotalidae ("pit vipers" = rattlesnakes, copperheads, cottonmouths): most US envenomations, ~25% are dry bites. Venom types: cytotoxic & hemolytic. SSx: <u>local</u>: painful, edema/erythema/bullae, compartment syndrome; <u>systemic</u>: paresthesias, metallic taste; <u>hemeatologic</u> - coagulopathy, DIC, thrombocytopenia Elapidae (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 (inhibits ACh receptors). SSx: broadly think neuro symptoms: bulbar palsies, respiratory paralysis.</p>
<p>Distinguish treatment of Crotalid vs Elapid snake bites.</p>	<p>Crotalid Rx: local wound care, supportive care, frequent coag checks, update tetanus, consider antivenom for moderate to severe envenomations (CroFab- from sheep, allergic reactions not common). Elapidae Rx: admit ALL for monitoring of delayed neuro symptoms, antivenin for eastern coral snakes, intubate and give supportive care as needed.</p>
<p>Diagnosis, Treatment: Crotalid compartment syndrome</p>	<p>Rare. Only occurs with bite into deep compartment, causing classic "5 Ps" Tx: 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, compression (air splint or elastic bandage). NO tourniquet unless it is possibly a coral snake bite with neurotoxic venom. Do not try to suck the venom out of the wound.</p>

<p>When should CroFab be given and what is the appropriate administration?</p>	<p>Severe symptoms after suspected Crotalid bite. CroFab causes less anaphylaxis than previous versions. Dosing is based on amount of venom, not patient weight. Typically 4-6 vials (slow over 10 minutes 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>
<p>Signs and Symptoms, Treatment: 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 saltwater SSx: "erysipeloid" cellulitis Tx: ciprofloxacin or penicillin/cephalosporin <i>M. marinum</i>: acid-fast bacillus in salt water SSx: "fish tank granuloma" nodules/papules in areas exposed to water weeks after cleaning a fish tank Tx: clarithromycin + ethambutol (TB meds) <i>V. vulnificus</i>: Gram negative rod in saltwater, ingestion of oysters or exposure to salt water SSx: hemorrhagic bullae, necrotizing fasciitis, primary septicemia (e.g. cirrhotic patient eats raw shellfish) Tx: ceftriaxone + doxycycline</p>
<p>Treatment: jellyfish envenomations (stings)</p>	<p>Cnidaria: sea anemones, fire coral, jellyfish, box jellyfish, portuguese man-of-war. Contain nematocysts: venom causes localized pain (most common symptoms), erythema, pruritus, arrhythmia (systemic). Tx: deactivating with HOT SALTWATER (not freshwater), topical lidocaine, scrape off nematocysts, give antivenom if box jellyfish.</p>
<p>What is the appropriate treatment of marine vertebrate wounds (e.g. stingrays, lionfish, stonefish)?</p>	<p>Provide local wound care, give antibiotics (cephalexin/doxycycline), rule out retained foreign body, HOT water immersion (>113° F (45° C) deactivates heat labile toxin), no acetic acid; do NOT close wounds.</p>
<p>Signs and Symptoms, Treatment: ciguatera vs. scombroid</p>	<p>Ciguatera: eating eel, barracuda, amberjack, snapper; SSx: vomiting, diarrhea, paresthesias, ataxia, hot/cold temperature reversal; Tx: supportive, anti-emetics, atropine (for bradycardia), mannitol if severe neuro sympoms Scombroid: improperly stored fish (Tuna, mahi-mahi, mackerel), peppery taste, histamine-mediated reaction; SSx: within one hour of ingestion, flushing, urticaria, palpitations, dizziness, headache; Tx: antihistamines, consider epinephrine/albuterol if respiratory difficulty</p>
<p>What are medication options for treating neurologic symptoms of ciguatera poisoning?</p>	<p>Amitriptyline and gabapentin</p>
<p>How do Tarantula bites present?</p>	<p>Mostly pain with very little swelling at the bite site. They have "urticating hairs" which can cause localized allergic reactions.</p>

Psychiatry & Behavioral

Bizz	Buzz
Define Axis 1-5 (these are not present in DSM-5, but may still be tested)	Axis 1: psychiatric disorders. Axis 2: 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 addiction and dependency ?	Addiction: compulsion to use substances despite adverse consequences (e.g. car crash, arrested, fired). Dependency: difficulty functioning without the substance, may have tolerance, withdrawal, and social retreat.
What are the key differences between anorexia nervosa and bulimia nervosa ?	Anorexia nervosa: restriction of calorie intake relative to needs, intense fear of weight gain, and calorie restriction rather than purging. Associated with successful suicide, bradycardia (dysrhythmia = MCC death), 50% good outcome. Bulimia nervosa: binge eating with purging cycle, 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 secondary to 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 ?	Anorexia: IVF, electrolyte repletion, admit for refeeding, avoid TPN Bulimia: CBT + SSRI. (Bupropion is contraindicated as it lowers the seizure threshold)
What SSx typically distinguish psychiatric and medical (organic) causes of psychosis ?	Psychiatric: auditory hallucinations, flat affect, intact orientation, symptoms are continuous, younger patient, gradual onset, negative symptoms Medical: visual hallucinations, labile affect, ± disoriented, symptoms wax and wane, older patient, abrupt onset
What are the classic positive and negative symptoms of schizophrenia ?	Positive: hallucinations, delusions, disorganized speech, catatonia Negative: blunted/flat affect, poverty of speech, anhedonia, social withdrawal
Signs & Symptoms, Treatment: 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. Tx: mood stabilizers (e.g. lithium, valproate) and antipsychotics (if psychotic features are present)
What distinguishes mania from hypomania ?	Mania: at least 1 week Hypomania: 4 consecutive days

What are the criteria for diagnosis of depression ?	Depressed mood for 2 weeks + 4 of "SAD CAGES" (changes in S leep, changes in A ppetite, D epressed mood, poor C oncentration, decreased A ctivity, feelings of G uilt/worthlessness, decreased E nergy, S uicidal ideation)
What are significant risk factors for completed suicide ?	"SAD PERSONS" : S ex (male), A ge (<19 or >45), D epression (or hopelessness), P revious attempt (most concerning risk factor), E xcessive alcohol or drug use, R ational thinking loss (e.g. 2/2 psych dx, dementia, etc.), S eparated (divorced or widowed), O rganized (or serious) attempt, N o social support, S tated future attempt Protective: marriage, pregnancy
Do no self harm contracts work?	No
What is the most common method of attempted and completed suicides ?	Attempted : girls/women, drug ingestions (MC: antidepressants) Completed : boys/men, firearms. Note : check acetaminophen level on all overdose/SI patients
What distinguishes malinger ing, 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.
Diagnosis : Kid with unusual presentation of disease, biological mom happy with abnormal results	Factitious disorder imposed on another (Formerly Munchausen by proxy)
How is Generalized Anxiety Disorder characterized?	Frequent and prolonged periods of worry and anxiousness (> 6 months)
How is Panic Disorder characterized?	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 (e.g. hyperthyroidism, PE, MI).
What is agoraphobia ?	Specific fear of open/public place .
What is a specific phobia ?	Clinically significant anxiety or worry that develops in response to a specific situation or object leading to avoidance behavior

<p>What distinguishes the anxiety-related symptoms of PTSD, OCD, GAD, panic disorder, agoraphobia, social phobia, and specific phobia? What is the treatment for all?</p>	<p>PTSD: long-lasting anxiety response following a traumatic/catastrophic event; SSx: sx > 1 month, flashbacks, hypervigilance, insomnia, poor concentration, irritable with angry outbursts. OCD: recurrent thoughts, images or urges (obsessions) and repetitive acts (compulsions). GAD: extreme multifaceted and uncontrollable worrying most days for > 6 months. Panic disorder: frequent panic attacks, at least some of which are not triggered. Agoraphobia: panic attacks triggered by being in (or the expectation of being in) situations that are difficult to escape (e.g. crowds). Social phobia: panic attacks or excessive fear triggered by anticipating or being in situations of social scrutiny. Specific phobia: panic attack + specific fear (e.g. snakes, spiders, enclosed spaces). Tx: cognitive behavioral therapy (CBT) and SSRI; Benzos can help abort panic attacks.</p>
<p>Patient with a panic attack hyperventilates and syncopezizes. What lab value and resultant physiologic response is associated with this phenomenon?</p>	<p>Hypocarbica → respiratory alkalosis → cerebral vasoconstriction. The alkalosis also causes increased serum calcium binding → decreased serum calcium → tetany</p>
<p>What is the difference between delirium and dementia?</p>	<p>Delirium: secondary to medical problem/drugs/tox; rapid onset, symptoms fluctuate, last hours to weeks, impaired attention/alertness/orientation, sleep-wake cycle disrupted, agitated, incoherent speech, +delusions/hallucinations. Dementia: gradual onset, slow decline, lasts months to years, attention intact (early in disease), sleep-wake normal, normal alertness, orientation intact (early in disease), behavior normal, word-finding problems, perceptions intact (early in disease)</p>
<p>Diagnosis: Patient suddenly unable to recall where he lives, but has no other neurological signs and symptoms?</p>	<p>Transient global amnesia. Temporary disruption of short-term memory loss. NOT an infarct (no sensory/motor deficits). SSx: sudden onset, patient often repeats questions. Workup negative Tx: self-resolves without intervention</p>
<p>Signs & Symptoms, Treatment: Delirium tremens</p>	<p>SSx: Severe ETOH withdrawal sx + autonomic instability + hallucinations + delirium + seizures. Peaks 2-5 days (~72 hours) after EtOH cessation Tx: Benzos, phenobarbital</p>

<p>What prophylaxis should be offered to patients after sexual assault?</p>	<p>Emergency contraception (e.g. Plan B) STD prophylaxis (don't test due to legal implications for patient), treat empirically with antibiotics for Gonorrhea/Chlamydia/Trichomonas HBV vaccine HIV post-exposure prophylaxis (do not give HIV PEP or Plan B after 3 days because ineffective)</p>
<p>Five clinical features of psychosis</p>	<ol style="list-style-type: none"> 1. Delusions 2. Disorganized thinking 3. Hallucinations 4. Negative symptoms 5. Grossly disorganized or abnormal motor behavior
<p>Distinguish flight of ideas and disorganized thinking</p>	<p>Flight of ideas: seen in mania. They frequently shift from one topic to another with continuous, accelerated speech pattern Disorganized thinking: seen in psychosis. Inferred from speech, answers are loosely related or unrelated, switches from one topic to the other, word salad (disorganized and incomprehensible speech)</p>
<p>What is the difference between paranoid, schizoid, and schizotypal personality disorders? (Cluster A: "weird", odd, and eccentric)</p>	<p>Paranoid: suspicious of others Schizoid: social detachment with restricted emotions (isolated, think hermit) Schizotypal: social detachment with eccentric behavior (e.g. magical thinking)</p>
<p>What is the difference between histrionic, narcissistic, borderline, and antisocial personality disorders? (Cluster B: "wild", dramatic, emotional, erratic)</p>	<p>Histrionic: excessive emotional lability & attention-seeking behaviors. Often sexually provocative. Narcissistic: grandiose, constant need for admiration, lacks empathy Borderline: unstable relationships, labile affect/mood, poor self-image, impulsive, demonstrates splitting (quickly regards others as the "worst" or "best" person ever) Antisocial: disregard for rights of others, frequent lying/cheating/stealing (associated with malingering)</p>
<p>What is the difference between avoidant, dependent, and obsessive-compulsive personality disorders? (Cluster C: "worried", anxious or fearful)</p>	<p>Avoidant: social withdrawal (awkward/uncomfortable in social situations), constantly feels inadequate, hypersensitive to criticism (Key: avoidant wants social interaction but is afraid due to fear of embarrassment) Dependent: indecisive (and needs others to make decisions), lacks self confidence, submissive Obsessive-compulsive: perfectionism/order valued over flexibility/efficiency Passive-aggressive: forceful, stubborn, dependent on others</p>

What is the difference between somatization, hypochondriasis, conversion disorder? (psychosomatic disorders, all unintentional)	<p>Somatization: physical complaints unexplained by medical workup, multiple different symptoms of multiple different systems (GI, GU, neuro) with unexplained cause, often affects life</p> <p>Hypochondriasis (Illness Anxiety Disorder): preoccupation with and fear of disease, conviction one is sick, symptoms out of proportion to clinical findings, often displays "doctor shopping"</p> <p>Conversion disorder: SUDDEN unexplained neuro symptoms (e.g. blindness, paralysis), often but not always in response to an emotional stressor.</p> <p>MUST rule out organic disease in all before making these diagnoses.</p>
What is the most common personality disorder?	Borderline (Cluster B)
Diagnosis: Patient with wide variety of complaints, complicated medical history, no clear cause of symptoms	Somatization disorder
Diagnosis: 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
Diagnosis: Sudden paralysis after an emotionally charged event	Conversion disorder
Diagnosis, Signs & Symptoms, Treatment: Drug ingestion + violent behavior with superhuman strength	<p>Dx: PCP ingestion (dissociative agent)</p> <p>SSx: sympathomimetic effects, bizarre & violent behavior, perceptions of superhuman strength; ± horizontal, vertical or rotatory nystagmus</p> <p>Tx: supportive care, sedate/restrain to ensure safety (benzos, AVOID Haldol), monitor for rhabdo and seizures</p>
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.
What is the difference between a brief psychotic disorder, schizophreniform disorder, schizophrenia, and schizoaffective disorder?	<p>Brief psychotic disorder: psychotic features < 1 month</p> <p>Schizophreniform: psychotic features for 1-6 months</p> <p>Schizophrenia: psychotic features > 6 months</p> <p>Schizoaffective: psychotic features + mania or depression</p>
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, delirium tremens	<p>Tremor (6-12 hrs)</p> <p>Hallucinations & seizures (12-48 hrs)</p> <p>DTs (>48hrs)</p>
What are the five stages of grief? At what point does grief become pathological grief?	Denial → Anger → Bargaining → Depression → Acceptance Pathological grief reaction if lasting > 6 months + causes severe functional impairment, suicidal ideation, psychotic symptoms

<p>What are the features of frontotemporal neurocognitive disorder?</p>	<p>Characterized by disruptive behavioral changes: hyperorality, wondering, and generally disinhibited. Characterized by a rapid decline.</p>
<p>What are the features of lewy body dementia?</p>	<p>Age: mid-70s. Executive functions and attention are affected. Visual hallucination and REM sleep behavior disorder. Parkinsonian motor symptoms.</p>
<p>What are some features of prion diseases?</p>	<p>Any age. Neurocognitive deficits + motor abnormalities (Ataxia, myoclonus, dystonia, chorea). Examples: Mad cow disease, kuru, Creutzfeldt-Jakob Dx. No treatments available.</p>

Miscellaneous Topics

Bizz	Buzz
What is the difference between an Emergency Medical Responder, Emergency Medical Technician, Advanced EMT and Paramedic?	EMR: immediate life-saving care (e.g. hemorrhage control, CPR , AED use) EMT: emergency care (e.g. BLS , O2, pt's own meds, patient transport) Advanced EMT: add limited advanced life support (e.g. IV ACCESS , some meds, airway management, ECGs, IVF) Paramedic: licensed, advanced care (e.g. ACLS meds, advanced airway, defib/pacing, needle decompression)
What is the difference between Off-line and On-line medical control for EMS providers?	Off-line/Indirect: developed protocols and standing orders for specific situations, training and education, quality review On-line/Direct: direct orders while in field (on scene or over radio), direct observations
When can a patient refuse care/transport by EMS?	Any adult 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; 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?	Rotary: limited by weather, less safe, expensive but access to more locations Fixed: 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 local healthcare system to manage them under normal operating procedures
What defines a Level I, Level II and Level III medical disaster ?	I: local resources sufficient II: requires resources from adjacent communities III: 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 Operations: field work including search and rescue, treat and transportation, triage (this is where the physicians typically are) Planning: collects data, communicates and coordinates plans Logistics: facilities, supplies, equipment, food, people Finance: manages money, payment

<p>What are the categories of primary triage using the Simple Triage and Rapid Treatment (START) protocol?</p>	<p>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: BLACK: <u>deceased, hopelessly injured</u> (no pulse and no breathing despite repositioning airway), no resuscitation RED: immediate priority - <u>requires treatment/stabilization for survival</u> (abnormal respirations/perfusion/mental status) YELLOW: delayed priority - seriously injured but <u>delayed treatment ok without loss of life or limb</u> (stable respirations/perfusion/mental status) GREEN: minor priority, <u>walking wounded</u>.</p>
<p>What are the only two interventions performed under START triage?</p>	<p>Airway repositioning, hemorrhage control</p>
<p>What is the exception to the typical Primary Triage "BLACK" (hopelessly injured, no resuscitation) rule?</p>	<p>Lightning strikes/electrical injuries; pulseless or apneic patients may be more easily resuscitated with immediate ACLS; "Reverse Triage"</p>
<p>What is the most common problem in any disaster?</p>	<p>Poor Communication</p>
<p>What are the key components of the Emergency Medical Treatment and Active Labor Act (EMTALA, part of COBRA)?</p>	<p>Patients presenting to ED by EMS require a medical screening exam to identify and stabilize life-threatening conditions; once ambulance on hospital property the hospital is obligated to eval the patient; if the facility cannot 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 physician</p>
<p>What rules exist under EMTALA for transferring a patient from the ED to another facility?</p>	<p>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</p>
<p>How does EMTALA apply to pregnant women in labor?</p>	<p>They may be transferred to another facility after a medical screening exam as long as 1) you can demonstrate that they require a higher level of care at another facility (i.e. OB care that your hospital doesn't have) 2) The patient has been medically stabilized 3) Labor is not imminent (e.g. fully dilated, close contractions). Always transport with fetal monitoring. Consider transport times when making decisions (labor may not seem imminent now, but that may change if there's a 4 hour transport time)</p>
<p>What are federal guidelines defined by the Joint Commission regarding language translation in the ED?</p>	<p>ED must provide language assistance to non-English speaking patients; family members should not be used as interpreters</p>

What is the difference between Expressed, Implied and Informed Consent?	Expressed: verbal or written willingness to be treated, covers "usual" care Implied: action implies willingness Informed: patient 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 physician 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 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 ?	Physician 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 provide consent), but under EMTALA, physician 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
What variables define a " mentally incompetent " patient?	Intoxicated (alcohol or drugs), psychotic, confused, disoriented or unconscious
When can parents NOT refuse care for their child?	They cannot forbid life-saving treatment (this includes religious freedom arguments); if parents withhold consent under such circumstances the physician can take temporary protective custody of the child (common boards scenarios: blood transfusion despite religious objection, treatment of dangerous conditions despite parental disagreement, i.e. meningitis)
When can involuntarily hold be placed on patients with mental illness?	If they are deemed to be a threat to themselves or others (i.e. suicidality, homicidality, manic behavior, decompensated psychosis)
What 4 elements are required in a malpractice suit to prove negligence/liability ?	1) Duty to the Patient 2) Breach of Duty 3) Injury caused by the breach 4) Resulting Damaged: tangible injury occurred
For what patients are physicians mandatory reporters for abuse?	Children/Minors and Elders (>60) (true in most states)
True or False: Emergency physicians 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?	Pre: occur during specimen collection and prior to processing (Most common) Analytic: processing/machine error Post: after results complete, incorrect reporting or interpretation

What factors specific to emergency medicine hinder physician wellness ?	Shift work, 12 hour 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?	Sensitivity: a test's ability to designate an individual with disease as positive. Specificity: a test's ability to designate an individual without disease as negative.
What type of test (high sensitivity or high specificity) is best for Screening/Ruling Out disease versus 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). Mnemonic: SP-IN, SN-OUT
What is the difference between Positive Predictive Value (PPV) and Negative Predictive Value (NPV)?	PPV: proportion of people with +Test who also have +Disease (True Pos / TP + False Pos) NPV: proportion of people 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 . (NNT = 1/ARR)
What are exceptions to confidentiality rules?	Public safety threats, 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?	Primary: targets <u>at risk and prevents</u> problem, includes vaccines, education, water treatment Secondary: <u>detect disease early</u> to prevent progression, includes pap smear, colonoscopy, mammography, etc. Tertiary: <u>limits progress</u> of known disease, includes risk factor modification, strict glucose control for DM, post MI meds
What is necessary to document on a patient who 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 protected health information be shared without explicit consent?	Another medical provider with direct patient care responsibilities or patient's insurance for billing purposes
What patients are appropriate for disposition to an ED Observation Unit?	Unclear diagnosis in the ED and require a limited amount of further evaluation (workup typically protocolized and completed within 24 hours)
How do you calculate a Positive Likelihood Ratio?	positive LR = Sensitivity/(1 – specificity)

<p>What are the 4 main principles of medical ethics? How are they defined?</p>	<ol style="list-style-type: none">1) Autonomy - recognizing the patient's values and right to make their own decisions2) Beneficence - act in the patient's best interest, balance the benefits of treatment with the risks/costs3) Nonmaleficence - do no harm4) Justice - treat patients fairly, evenly distribute the benefits and burdens of care across society, strive for equality in access and outcomes
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